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Tracheal gas insufflation reduces the tidal volume while $PaCO_2$ is maintained constant

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Abstract. *Objective:* The aims of the present study were two-fold: first, to confirm the effect of tracheal gas insufflation (TGI) throughout the respiratory cycle on alveolar ventilation at various catheter flows and constant total inspired V_T as an adjunct to conventional volume cycled mechanical ventilation in patients with acute lung injury; second, to test the efficacy of TGI in the reduction of toal V_T, peak and mean airway pressure while maintaining $PaCO_2$ in its baseline value. The hemodynamic effect and the consequences on oxygenation as result of the reduction of V_T, were also estimated.
Design: Prospective study of patients with acute lung injury requiring mechanical ventilation.
Setting: 12 bedded, adult polyvalent intensive care unit in a teaching hospital.
Patients: 7 paralyzed and sedated patients with acute respiratory failure were studied. All patients were clinically and hemodynamically stable without fluctuation of the body temperature. All patients were orally intubated with cuffed endotracheal tubes, and mechanically ventilated with a standard circuit of known compliance.
Interventions: Continuous flows (4 and 6 l/min) were delivered through a catheter positioned 1 cm above carina while tidal volume or $PaCO_2$ were maintained constant at their baseline value.
Results: In this study a modest level of TGI significantly enhanced CO_2 elimination in patients with acute respiratory failure. Improved ventilatory efficiency resulted from the functional reduction of dead space during TGI allowing the same $PaCO_2$ to be maintained at the same frequency with lower tidal volume and lower airway pressure requirement. Tidal volume, peak and mean airway pressure decreased linearly with catheter flow, without significant changes in oxygenation, while $PaCO_2$ remained stable.
Conclusion: The results of this study suggest that TGI may be an useful adjunct mode of mechanical ventilation that limits alveolar pressure and minute ventilation requirements.

Key words: Mechanical ventilation – Acute lung injury – Dead space – Airway pressure – Gas insufflation – Barotrauma

Mechanical ventilation with high tidal volume and high inflation pressure is a well recognized risk factor predisposing to ventilator-associated barotrauma by producing alveolar distention. Clinical manifestations of barotrauma include pulmonary interstitial emphysema, pneumothorax, subcutaneous emphysema, pneumoperitoneum, tension lung cysts, hyperinflated left lower lobe, subpleural air cysts, and air embolization [1, 2]. In addition to these forms of barotrauma, animal experiments have shown that high cycling volume and pressure can cause high permeability pulmonary edema even in previously normal lungs [3–9]. The mechanism of lung injury is unknown, but it appears to be a consequence of lung overdistension. It has been demonstrated in animals that ventilation with high positive inspiratory pressure in the presence of thoraco-abdominal strapping to prevent lung overdistension does not result in lung injury. In contrast high volume ventilation using a negative pressure ventilator with ambient airway pressure does result in lung injury [5, 10, 11]. In all animal studies commencing with normal lungs, large tidal volumes were required to induce lung injury. It may appear unlikely that such volume would be used in clinical practice. However, Gattinoni et al. [12] have shown that in patients with the Adult Respiratory Distress Syndrome (ARDS) most ventilation is distributed to a small amount of remaining aerated lung resulting in very high regional ventilation, overdistention, and injury of these lung regions [12, 13].

Several ventilatory approaches have been used to reduce ventilator-induced lung injury: Extrapulmonary gas exchange (extracorporeal or intercaval CO_2 removal) reduced the need for alveolar ventilation [14, 15]. Anoth-

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er less invasive approach is the use the permissive hypercapnia. In most circumstances hypercapnia appears to be well tolerated. However, hypercapnia could be detrimental in some patients, for example those with ischemic heart disease or hypertension, and bicarbonate retention may retard machine withdrawal later in the clinical course [16]. An alternative to "permissive hypercapnia" would be the reduction the CO_2-laden anatomic dead space. Conceptually, the dead space could be reduced by insufflating fresh gas via a small catheter placed in the trachea. The flow of fresh gas during expiration would replace a variable portion of CO_2-laden gas of anatomic dead space proximal to the catheter tip. In addition, catheter flow during inspiration not only contributes to V_T but also by-passes the airway segment proximal to the catheter tip [17–20].

The aims of the present study were twofold; first, to confirm the effect of tracheal gas insufflation (TGI), throughout the respiratory cycle, on alveolar ventilation at various catheter flows and constant total inspired V_T as an adjunct to conventional volume cycled mechanical ventilation in patients with acute lung injury; second, to test the efficacy of TGI in the reduction of total V_T, peak and mean airway pressure while maintaining $PaCO_2$ in its baseline value. Apart from these, special interest was paid to evaluation of the hemodynamic effect and the consequences on oxygenation as result of the reduction of V_T, which have not yet been estimated.

Materials and methods

Subjects

Seven patients with acute hypoxemic respiratory failure due to acute lung injury (ALI) were studied. The catastrophic events which had led to the development of ALI as well as additional patient data are shown in Table 1. The diagnosis of ALI was based on clinical, radiological, respiratory system compliance, gas exchange and hemodynamic criteria. Severity scoring of ALI was performed as described by Marray et al. [21]. Patients were accepted in this study when the severity score of ALI was equal or more than 1.5, (moderate and severe injury), and were clinically and hemodynamically stable without fluctuation of the body temperature. Patients with high filling pressures (pulmonary capillary wedge pressure > 16 mmHg) at the initial evaluation by the attending physician, hemodynamic instability, barotrauma or pneumothorax from other cause, and patients with craniocerebral trauma with evidence of increased intracranial pressure were excluded. All patients were transorally intubated with cuffed endotracheal tubes, 8.0–9.0 mm ID and 31 cm long (Portex Ltd; Hythe, Kent, England) and mechanically ventilated with a volume control mode (Servo 900C, Siemens, Sweden) and a standard circuit of known compliance (0.002 l/cmH_2O). For the baseline measurements, patients were maintained on the ventilator setting previously chosen by the attending physicians. A radial arterial catheter was placed for blood pressure monitoring and blood gas sampling. Opticath flow directed pulmonary artery catheter (Critical care system, Oximetrix- 3 computer $S\bar{v}$ O_2-CO, Abbot laboratories, Chicago, USA) had been in place in 3 patients by their attending physicians. During the study period, all patients received continuous intravenous infusions of sedatives (midazolam 0.05–0.2 mg/kg/h) and paralytic agents (vecuronium bromide 0.1–0.2 mg/kg/h). An adapter, that contains a rubber diaphragm (Swivel elbow plus seal cap, Intersurgical, Twickenham, Middesex, UK) was attached to the endotracheal tube to facilitate passage of the intratracheal catheter, 1.1 mm ID (Vygon intravenous catheter, Ecouen, France). The catheter was connected to a oxygen blender (Sechrist Sarns, air-O_2 mixer, Michigan). The airway pressure was measured from a side tap placed proximal to endotracheal tube and connected by air filled tubing to the pressure transducer (Validyne MP 45±100 cmH_2O; Validyne Co, Northridge, CA). Flow was measured with a pneumotachograph (Fleish #2 Lausanne, Switzerland) inserted distal to the Y-piece of the ventilator circuit and connected to a differential pressure transducer (Validyne MP 45±2 cmH_2O, Validyne Co, Northridge, CA). Inspiratory and expiratory flow were used to verify the catheter flow as well as the selected ventilator tidal volume. Volume was obtained by electronic integration of the flow signal (Validyne FV 156-871). A mixing chamber (~15 l) was attached to the expiratory port of the ventilator to facilitate continuous monitoring of mixed expired CO_2 concentration (Datex capnograph 103-23-01, Multicap, Sweden). All signals were recorded on an 8-channel recorder (Gould ES 1000, Ohio, USA).

The protocol was approved by the institutional scientific committee and informed consent was obtained from the patients or family member closest in kinship.

Calculations [17]

Tidal volume. The effective V_T (V_{Teff}) inspired by the patients during each stage was calculated by integrating the inspiratory signal from pneumotachograph over the inspiratory time, correcting for compressible volume, and adding the volume delivered by the catheter during the inspiratory time (T_I). The complicance factor was used to correct for compressible volume [22]:

$$V_{Teff} = V_{Tpneumo} - [(P_{pk} - PEEP) \cdot 0.002] + (\dot{V}_{cath} \cdot T_I)$$

where P_{pk} is peak airway pressure (cmH_2O), and $\dot{V}_{cath}$ is catheter flow (l/s)

Table 1. Demographic and mechanical ventilation data under baseline conditions

Patients no.	Cause	Age (years)	Sex	Score[a]	MV (l/min)	FIO_2	F (B/min)	PEEP (cmH_2O)	Crs (ml/cmH_2O)
1	Gastrointestinal bleeding	64	M	2	9.5	0.5	15	5	45.24
2	Sepsis (cholangitis)	52	M	2.7	10.8	0.5	16	8	39.71
3	Trauma (lung contution)	57	F	3	12	0.5	18	6	35.09
4	Aspiration gastric content	61	M	1.5	11.2	0.5	20	5	43.08
5	Sepsis (pneumonia)	39	M	3.5	14.5	0.6	24	8	30.21
6	Pancreatitis	70	F	2.2	8.6	0.5	12	5	47.78
7	Sepsis (postoparation)	68	M	2	14	0.5	22	5	63.64
Mean		59.00		2.41	11.50		18.00	6.00	53.53
SD		11.00		0.69	2.20		4.00	1.41	10.71

Definition of abbreviation: M, Male; F, female; MV, minute ventilation; F, frequency; Crs, respiratory system compliance; FIO_2, inspired fraction of oxygen; B, breaths/min

[a] Score 1.5–2.5 are associated with moderate lung injury, score>2.5 denotes severe lung injury

Carbon dioxide production. The carbon dioxide production (VCO_2) was calculated by the following equation:

$$\dot{V}CO_2 = \dot{V}_I \cdot [\bar{P}_ECO_2/(P_B\text{-}47)] \cdot (\text{BTPS to STPS correction factor})$$

where $\dot{V}_I$ is the effective inspired minute ventilation ($V_{Teff} \cdot$ frequency), and $\bar{P}_ECO_2$ is the mixed expired CO_2 partial pressure corrected for dilutional effects of compressive volume and catheter flow ($\dot{V}_{cath}$) delivered during expiratory time (T_E). $\dot{V}_{cath}$ remained constant throughout the respiratory cycle, forming part of V_T during inspiratory phase. During exhalation, however, $\dot{V}_{cath}$ (and decompressed circuit volume) diluted the $\bar{P}_ECO_2$ measured at the mixing chamber. $\bar{P}_ECO_2$ measurement were adjusted to represent only the CO_2 concentration in V_{Teff}:

$V_I \cdot$ corrected $\bar{P}_ECO_2 = [V_I + \text{compressive volume} + \dot{V}_{cath} \cdot T_E] \cdot$ measured $\bar{P}_ECO_2$ ($\dot{V}_I = V_{Teff} \cdot$ frequency).

Dead space: The dead space to tidal volume ratio (V_D/V_T) was calculated using the Enghoff modification of Bohr equation [23]:

$$V_D/V_T = (PaCO_2 - \bar{P}_ECO_2)/PaCO_2$$

Mean airway pressure. Mean airway pressure (P_{aw}) was determined averaging the integrated area under the airway pressure tracing over the total respiratory cycle time (T_T):

$$\bar{P}_{aw} = \frac{1}{T_T} \int_0^{T_T} P_{aw} dt$$

The area of airway pressure over time was measured by computer assisted planimetry.

Compliance of respiratory system. The compliance of respiratory system (Crs) was calculated as the quotient of inspiratory V_{Teff}) and the static airway pressure minus the end-expiratory alveolar pressure.

Study protocol

The distance from the tip of the endotracheal tube to the carina was measured using a recent chest radiograph. This measurement was used to adjust the catheter length for each patient. The total length of the catheter was equal to the sum of the distance from the tip of the endotracheal tube to the carina plus the length of the endotracheal tube and the length of the adapter. The catheter position was verified by a chest radiograph and replaced if necessary. Gas delivered through the intratracheal catheter was blended to match the fractional inspired O_2 concentration of the ventilator. When the intratracheal catheter was placed, data to determine thoracic mechanics were recorded on an 8 channel recorder and on a video tape for later analysis. Recorded data included inspiratory and expiratory flow, inspired and expired volume (integrated flow signal) peak, static (baseline stages) and end-expiratory airway pressure.

Baseline. Baseline stages were conducted before and after completion of the study with catheter flow 0 l/min, the catheter was placed at the level of the carina.

First experiment. Throughout this experiment the inspired tidal volume, frequency, and inspiratorion:expiration (I:E) ratio were kept stable and equivalent to those used prior to the study. The ventilator-delivery V_T was adjusted to maintain a constant inspiratory V_T (the sum of ventilator-delivered and catheter-delivered volumes). The ventilator's inspiratory flow rate was also adjusted to keep I:E ratio constant. We studied in random order two catheter flow rates (4 and 6 l/min).

Second experiment. After the end of each stage of the first experiment, the ventilator-delivery V_T was reduced to achieve the baseline $PaCO_2$. We studied in random order two catheter flow rates (4 and 6 l/min), the frequency, and the I:E ratio were kept stable. To allow equilibration of gas exchange, patients remained at each stage for a minimum of 45 min before data collection. At the end of each stage inspiratory and expiratory flows, volumes, and tracing of the airway pressure were recorded. Mixed expired carbon dioxide ($\bar{P}_ECO_2$), blood gases (Radiometer ABL 3, Copenhagen, Denmark), cardiac output (CO) and $S\bar{v}O_2$ (when available) were measured.

Statistical analysis

Results are expressed as mean+SEM. Statistical analysis was done using two-way analysis of variance. Internal comparisons were made using Student's *t*-test with Bonferroni adjustment for multiple comparisons. We used a paired *t*-test to determine the reproducibility of baseline values recorded at the beginning and at the end of each experiment.

Results

Patient stability

Carbon dioxide production remained unchanged throughout the experiments. Mean arterial blood pressure and heart rate were unaffected by the catheter flow. Baseline measurements of pH, $PaCO_2$, PaO_2, V_D/V_T, peak airway pressure, mean airway pressure, and Crs obtained before and at the end of the study showed no significant changes. Nore of the patients developed any kind of adverse effects. Drying effects of the catheter flow were not observed during the sort time of this study.

First experiment

Tidal volume. During this experiment the total V_T remained essentially constant, and there was no statistically significant difference from the baseline V_T.

Gas exchange and hemodynamics. In all patients, $PaCO_2$ decreased from baseline at both catheter flow rates ($p < 0.0001$). TGI reduces $PaCO_2$ more effectively at high flow rate (6 l/min) than at the lower flow rate (4 l/min) ($p < 0.01$). The reduction of $PaCO_2$ is expressed both as the percentage from baseline values (Fig. 1) and as its actual value (Table 2). Physiologic dead space fraction (V_D/V_T decreased significantly with the increasing catheter flow rate ($p < 0.0001$) (Table 2). There was a significant increase in PaO_2 only with the high flow rate (6 l/min) ($p < 0.05$), but there was no significant change in alveolar-arterial oxygen difference and SaO_2 in both catheter flow rates. DO_2, CO and $S\bar{v}O_2$ were unaffected in the first experiment (Table 2).

Airway pressure. There were no significant changes in peak or mean airway pressure with both catheter flow rates (Table 2). The instantaneous airway pressure measured at the onset of inspiratory flow (a rough estimate of the end-expiratory alveolar pressure) was not affected by the catheter flow (Table 2).

Second experiment

Tidal volume. In all patients, the total V_T decreased significant with catheter flow while $PaCO_2$ was maintained stable at the baseline values. The higher catheter flow was more effective in reduction of V_T ($p < 0.0001$) (Table 2, Fig. 2).

Gas exchange and hemodynamics. During this experiment the $PaCO_2$ remained essentially constant, and there was no statistically significant difference from the base-

Table 2. Effects of catheter flow on gas exchange, hemodynamics and airway pressure

	Experiment 1 (V_T constant)			Experiment 2 ($PaCO_2$ constant)		
$\dot{V}_{cath}$ (l/min)	0	4	6	4	6	0
pH	7.36±0.02	7.38±0.02	7.4±0.02	7.35±0.02	7.36±0.02	7.36±0.01
$PaCO_2$ (mmHg)	46±4.8	37.5±4*	34.5±6.2*	43.7±3	44.1±5.6	48±5.3
PaO_2 (mmHg)	82±6	84±8	89±6.2*	80±8	78±9	82±6
SaO_2 (%)	95±1.8	93±2.1	96±1.8	94±2.3	92±2	94±1.9
$\dot{D}O_2$ (mm/kg/min)	15.6±1.2	15.8±1	16±1.1	17±1.2	17.1±2	16±1.3
$P(A\text{-}a)O_2$	211±35	219±41	220±45	206±38	202±45	210±30
V_D/V_T	0.59±0.07	0.50±0.04*	0.46±0.05*	0.46±0.04*	0.43±0.05*	0.58±0.04
V_T (ml)	643±62	637±60	640±62	533±48*	488±57*	647±66
$\dot{V}CO_2$ (ml/min)	258±37	254±31	244±27	244±31	249±30	250±30
$S\bar{v}O_2$ (%)	75±5.5	74±6.2	74±6	76±5	77±5	74±5
P_{peak} (cmH_2O)	31±4.3	29.4±4	29±4.7	27.2±3.9*	25.1±4*	31.2±4.4
$\bar{P}_{aw}$ (cmH_2O)	12.8±1.9	12.5±1.7	12.5±2	11±1.7*	9.8±1.8*	13.1±1.9
P_{IZ} (cmH_2O)	6.1±1.3	6.2±1.2	6.5±1.1	6.1±1.3	6.1±1.2	6±1.3

Asterisk denotes statistically significant difference from baseline values

line $PaCO_2$. Physiologic dead space fraction (V_D/V_T) decreased significantly with the increasing catheter flow rate ($p<0.0001$) (Table 2). There were small decreases, but no statistically significant change, in PaO_2 and in alveolar-arterial oxygen difference. SaO_2 remained unchanged, DO_2, CO and $S\bar{v}O_2$ were slightly increased, but not statistically significant with decreasing V_T in the second experiment (Table 2).

Airway pressure. Peak airway pressure and mean airway pressure declined as a function of catheter flow ($p<0.0001$) (Table 2, Fig. 3). The instantaneous airway pressure measured at the onset of inspiratory flow (Piz) was not affected by the catheter flow and as well as by the reduction of tidal volume (Table 2).

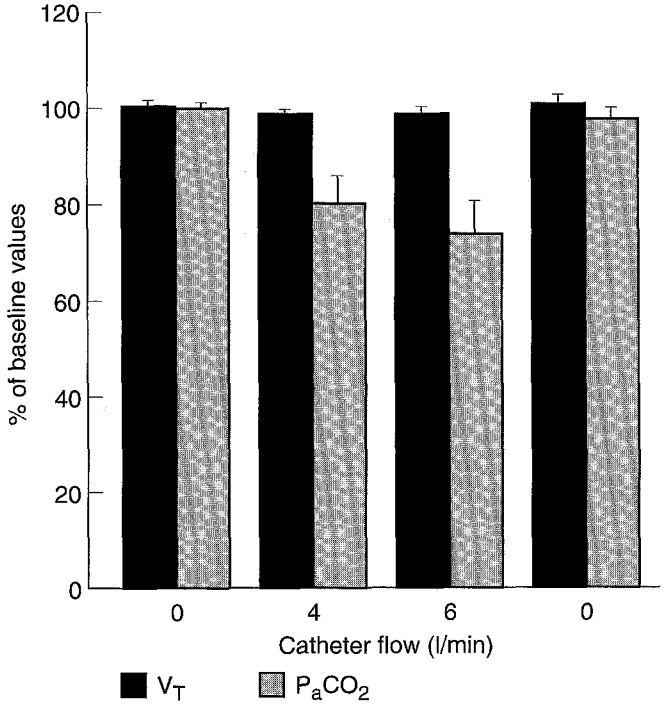

Fig. 1. The effect of catheter flow on $PaCO_2$ while V_T was maintained without significant changes. Data are expressed as mean±SD of the percentage change from the mean baseline value. $PaCO_2$ was linearly reduced with increasing catheter flow ($p<0.0001$). Zero (0) catheter flow represents the baseline values at the beginning and at the end of the study

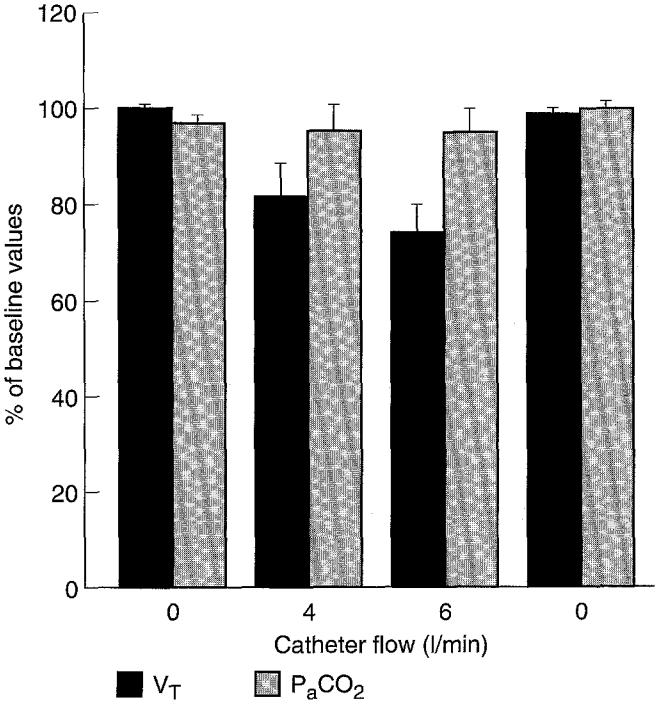

Fig. 2. Percent reduction in V_T from the baseline value as a function of catheter flow, while $PaCO_2$ was maintained without significant changes from baseline values. Data are expressed as mean±SD of the percentage change from the mean baseline value. Increasing catheter flow caused a reduction in V_T ($p<0.0001$). Zero (0) catheter flow represents the baseline values at the begining and at the end of the study

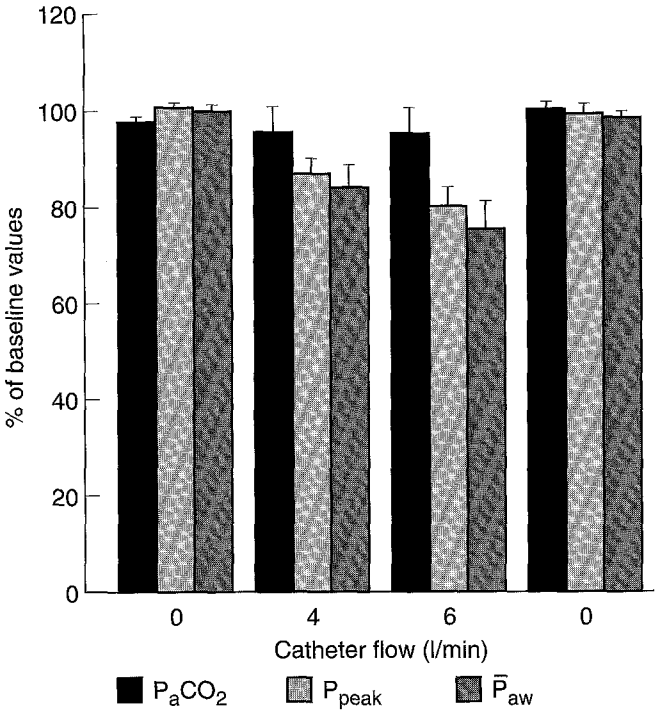

Fig. 3. Percent reduction in P_{peak} and $\bar{P}_{aw}$ from the baseline value as a function of catheter flow, while $PaCO_2$ was maintained without significant changes from mean baseline value. Data are expressed as mean ± SD of the percentage change from the baseline value. Increasing catheter flow caused a reduction in P_{peak} and $\bar{P}_{aw}$ ($p < 0.0001$). Zero (0) catheter flow represents the baseline values at the beginning and at the end of the study

Discussion

In this study, a modest level of TGI significantly enhanced CO_2 elimination in patients with acute lung injury. Improved ventilatory efficiency resulted from the functional reduction of dead space during TGI allow the same $PaCO_2$ to be maintained at the same frequency with lower tidal volume and lower airway pressure requirement. V_T, peak and mean airway pressure decreased linearly with catheter flow, while $PaCO_2$ remained stable.

The primary mechanism for enhancing the CO_2 elimination is likely to be a functional decrease in anatomic and apparatus dead space. Fresh gas flowing from the catheter during expiration flushes the proximal to catheter tip dead space, reducing the amount of CO_2 that returns to the alveolar with the next inspiratory cycle [24]. At high catheter flow, gas exits the catheter's orifice forming a jet stream in the trachea which generates turbulent eddies downstream, the resulting turbulence might also enhance gas mixing distally and improve alveolar ventilation [25–28]. Although, TGI applied in the constant flow mode resulted in similar improvements in $PaCO_2$ as catheter flow was delivered selectively during the expiratory period [27]. In the present study, we chose to use a constant rather than phasic catheter flow, in order to avoid specialized apparatus and to simplify the application of this technique.

Effects of TGL on gas exchange

In the first experiment, arterial oxygenation was improved during tracheal gas insufflation with relatively high flow (6 l/min). The change of PaO_2 may be explained by catheter flow causing increase in the alveolar pressure ($\bar{P}_A$) and alveolar recruitment. $\bar{P}_{aw}$ was unchanged during this experiment, but $\bar{P}_{aw}$ may not accurately reflect $\bar{P}_A$ [28, 29]. Theoretically, an increase in aveolar pressure improves PaO_2 and Alveolar-arterial oxygen tension difference as a consequence of alveolar recruitment. Our results showed an increase in PaO_2 but $P(A\text{-}a)O_2$ was unchanged. We speculate that the improvement of PaO_2 was not caused by the raise of $\bar{P}_A$, but only reflects the increase in P_AO_2 caused by the reduction of P_ACO_2.

In the second experiment, the reduction of V_T, $\bar{P}_{aw}$ and $\dot{V}_E$, all key determinants of $\bar{P}_A$ cause no statistically significant, decrease in PaO_2. Reduction in $\bar{P}_A$ is likely to be a cause of gas exchange deterioration. In our group of patients and under tested conditions, the small decrease in PaO_2 did not affect oxygen saturation and oxygen content. The reduction in V_T could decrease intrathoracic pressure and consequently a significant increase in cardiac output and intrapulmonary shunt [30]. Our results indicate that reduced V_T could improve $\dot{D}O_2$ (increase 7% of the baseline values, not statistically significant, probably because of the small number of patients tested despite an increase of venous admixture as indicated from the slight increase (4% of the baseline values, not statistically significant) in $P(A\text{-}a)O_2$. In the clinical setting, a lower V_T could used to increase $\dot{D}O_2$ [31, 32].

Effects of TGL on airway pressure

In the first experiment (V_T constant), P_{peak}, $\bar{P}_{aw}$, and P_{IZ} did not change significantly. In the second experiment ($PaCO_2$ held constant), both P_{peak} and $\bar{P}_{aw}$ were reduced linearly with catheter flow, and P_{IZ} did not change significantly.

When the patient is mechanically inflated from the airway opening the simplified equation of motion for the respiratory system states that:

$$P = R \cdot \dot{V} + V_T/C_{rs} + P_{ex}$$

The pressure applied on inspiration across the system (P_{aw}) has 3 major components: first, the flow-resistive component, $R \cdot \dot{V}$, where R and $\dot{V}$ are inspiratory resistance and flow, respectively; second, the tidal elastic component, V_T/C_{rs}, where V_T is the tidal volume distending the system above the end-expiratory position; third, the residual elastic pressure component, P_{ex}. P_{ex} represents the sum of PEEP and auto-PEEP.

The primary mechanism of airway pressure reduction was the reduction of V_T. The decreased V_T reduces the tidal elastic component, and secondly, reduces the inspiratory flow-resistive pressure by the product of the machine's flow reduction. The catheter flow bypasses the resistance of the segment between the site of airway pressure measurement and the catheter tip.

TGI could be ineffective or less effective in reduction of airway pressure if the reduction of V_T leads to a decrease in lung compliance and if the constant TGI increases the expiratory resistance (increased expiratory flow) and results in the development of autoPEEP. In this study the complicance of the respiratory system was unchanged in the second baseline measurements. AutoPEEP could not be measured directly during the study due to the experimental set-up and was estimated from Piz. It is not clear whether Piz accuracy reflects autoPEEP. The effect of TGI on hyperinflation due to autoPEEP is uncertain in this study. In a recent study using TCF with V_T constant [17] lung volume changes were assessed by plethysmography and it was demonstrated that FRC (end-expiration volume) increase 45 ± 7 and 62 ± 16 ml at 4 and 6 l/min catheter flow respectively. The long-term effect of low V_T are beyond the scope of this study.

Comparison with previous works

The benefits of by-passing the dead space were noted in the early 1960s when tracheostomy was employed as a treatment for patients with acute respiratory failure [33]. The technique of supplying oxygen through a catheter implanted into the trachea (transtracheal oxygenation) was introduced by Heimlich in 1982 [34] and appears to offer some advantages to hypoxic patients in terms of convenience, cost savings relevant to the use of oxygen [35] and reduction of the minute ventilation and work of breathing [36]. The technique of tracheal air or oxygen mixture insufflation has been used to maintain adequate oxygenation in apneic animals and humans [37–39]. Slutsky et al. [36] demonstrated that tracheal insufflation of oxygen at low flow rate can produce sufficient gas exchange to support life for prolonged periods in apneic dogs. Expiratory flushing of the proximal anatomic dead space is now being applied experimentally as an adjunct to mechanical ventilation. In previous work with normal dogs it has been demonstrated that a significant reduction in $PaCO_2$ and V_D/V_T occur when constant catheter flow was used in conjunction with volume-preset ventilation. The result of this study showed that TGI can effectively augment alveolar ventilation during mechanical ventilation and depends strongly on catheter flow [19]. Oxygen has been insufflated directly into the trachea of spontaneously breathing hypercapnic patients, decreasing the inspired V_T, V_D and minute ventilation without affecting $PaCO_2$ in acute state, and maintaining or reducing $PaCO_2$ in the chronic state [40]. TGI has been reported as an adjunct to conventional mechanical ventilation in a single patient with acute lung injury [41]. In that report, two catheters positioned alongside the endotracheal tube into the mainstem bronchi were used. Oxygen mixture was delivered selectively on expiration during pressure controlled ventilation. With this technique the inspired V_T requirement was reduced while maintaining $PaCO_2$ constant. These results were consistent with ours in the second experiment. In another study, Ravenscraft and colleagues [17] tested the effect of intratracheal gas insufflation in 8 sedated, paralyzed, mechanically ventilated patients with a variety of lung disorders. A catheter was positioned 1 and 10 cm above carina. During volume cycled ventilation, constant flow of 2, 4, and 6 l/min were delivered. $PaCO_2$ and V_D/V_T decreased significantly as a function of both catheter flow and position. The highest catheter flow and the distal catheter position were the most effective combination tested. The results of this study are quite consistent with ours in the first experiment.

Clinical applications

Before TGI can be applied routinely to patients, certain issues must be addressed. Airway obstruction proximal to the catheter tip during TGI can lead to dynamic hyperinflation with possible hemodynamic compromise and barotrauma. Moreover, TGI can increase the expiratory resistance by increasing expiratory flow and decreasing the effective cross-sectional area of the trachea, this effect could amplify dynamic hyperinflation. A pressure release mechanism needs to be incorporated into the ventilatory circuit designed for clinical application. With long term use it also be necessary to humidify catheter flow to minimize inspissation of mucus. Humidification of the insufflated gas is not a problem at all when low catheter flow (< 10 l/min) and a large internal diameter (> 1.3 mm) are used. Small catheters and high flows cause very high back pressure and special designed humidifiers are needed. The positioning of the catheter near the carina under chest X-ray guidance is a simple means of catheter placement.

Tracheal gas insufflation has clinical application in settings where it is desirable to minimize tidal volume and alveolar pressure while maintaining acceptable values for $PaCO_2$ and pH. The proportion of each breath ventilating the anatomic dead space (V_D/V_T) increases when small V_T are used. TGI mainly improves alveolar ventilation by flushing anatomic dead space it results in large decrease in $PaCO_2$ by decreasing V_D/V_T. From the preliminary results of this study, we believe that the reduction of V_T, following TGI, decreases airway pressure without significant changes in oxygenation and hemodynamic.

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