Aspiration of Dead Space in the Management of Chronic Obstructive Pulmonary Disease Patients With Respiratory Failure

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INTRODUCTION: Carbon dioxide clearance can be improved by reducing respiratory dead space or by increasing the clearance of carbon-dioxide-laden expiratory gas from the dead space. Aspiration of dead space (ASPIDS) improves carbon dioxide clearance by suctioning out (during expiration) the carbon-dioxide-rich expiratory gas while replacing the suctioned-out gas with oxgenated gas. We hypothesized that ASPIDS would allow lower tidal volume and thus reduce exposure to potentially injurious airway pressures. METHODS: With 8 hemodynamically stable, normothermic, ventilated patients suffering severe chronic obstructive pulmonary disease we tested the dead-space-clearance effects of ASPIDS. We compared ASPIDS to phasic tracheal gas insufflation (PTGI) during conventional mechanical ventilation and during permissive hypercapnia, which was induced by decreasing tidal volume by 30%. The mean P_{aCO_2} reductions with PTGI flows of 4.0 and 6.0 L/min and during ASPIDS (at 4.0 L/min) were 32.7%, 51.8%, and 53.5%, respectively. Peak, plateau, and mean airway pressure during permissive hypercapnia were significantly lower than during conventional mechanical ventilation but PTGI increased peak, plateau, and mean airway pressure. However, pressures were decreased during permissive hypercapnia while applying ASPIDS. Intrinsic positive end-expiratory pressure also increased with PTGI, but ASPIDS had no obvious influence on intrinsic positive end-expiratory pressure. ASPIDS had no effect on cardiovascular status. CONCLUSIONS: ASPIDS is a simple adjunct to mechanical ventilation that can decrease P_{aCO_2} during conventional mechanical ventilation and permissive hypercapnia. *Key words: mechanical ventilation, chronic obstructive pulmonary disease, tracheal gas insufflation.* [Respir Care 2004;49(3):257–262. © 2004 Daedalus Enterprises]

Introduction

Ventilator-induced lung injury is a potential complication of mechanical ventilation and has received increasing attention over the past 10 years. Determining an effective lung-protective ventilation strategy is an important and challenging task for critical care practitioners.[1–6] Permissive hypercapnia, tracheal gas insufflation (TGI), and partial liquid ventilation have been proposed to reduce the risk of ventilator-induced lung injury. Several studies have reported that TGI can decrease carbon dioxide retention and promote ventilation efficiency,[7–12] but TGI may also increase airway pressure during expiration. Furthermore, unconditioned TGI gas has the potential to dry airway secretions and damage airway mucosa.[13] Aspiration of dead space (ASPIDS), which is an alternative to TGI, employs controlled aspiration instead of insufflation.[14,15] During expiration, tracheal gas is aspirated through a catheter in the tracheal tube as a compensatory flow of fresh gas is injected into the inspiratory line. Therefore ASPIDS may be able to avoid certain potential hazards of TGI, such as higher airway pressure and intrinsic positive end-expiratory pressure (PEEPi) during expiration. We stud-

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Table 1. Characteristics of Patients

Patient No.	Age (y)	Sex	Weight (kg)	Disease
1	78	Male	72	COPD and cor pulmonale
2	71	Male	68	COPD
3	71	Female	80	COPD and cor pulmonale
4	73	Male	65	COPD
5	65	Female	70	COPD and cor pulmonale
6	58	Male	75	COPD and cor pulmonale
7	69	Male	57	COPD and lung fibrosis
8	64	Female	63	COPD
Mean ± SD	69 ± 6		69 ± 7	

COPD = chronic obstructive pulmonary disease.

ied the effect of an ASPIDS system when tidal volume (V_T) was reduced in chronic obstructive pulmonary disease (COPD) patients suffering respiratory failure.

Methods

We compared conventional mechanical ventilation, low-tidal-volume (V_T) ventilation (permissive hypercapnia), phasic tracheal gas insufflation (PTGI), and ASPIDS with 8 hemodynamically stable, normothermic, adult, ventilated patients who presented to our respiratory intensive care unit with severe chronic obstructive pulmonary disease (Table 1). The study was approved by our institutional review board and conducted between February and August of 2001.

The patients were intubated with cuffed endotracheal tubes (ETTs, inner diameters of 7–8.5 mm). The cuff was frequently checked to detect and avoid air leakage. We applied volume-controlled ventilation with a constant inspiratory flow pattern (Servo 900C, Siemens-Elema, Sweden). On average, V_T was 13.5 mL/kg (range 12–14 mL/kg), respiratory rate was 16.5 breaths/min (range 14–18 breaths/min), applied PEEP was 6 cm H_2O (range 4–8 cm H_2O), and fraction of inspired oxygen (F_{IO_2}) was 0.45 (range 0.4–0.5). Inspiratory time was set at 25% of respiratory cycle time and post-inspiratory pause time was 10% of cycle time. A moisture exchanger and bacterial/viral filter (AC53/FE62NST, Europe Medical, Bourgen Bresse, France) and a connector to the Y-piece were in place. Monitoring included electrocardiogram, noninvasive arterial blood pressure measurement, end-tidal capnometry, and body temperature measurement (54S monitor, Hewlett-Packard, Palo Alto, California). To inhibit spontaneous breathing the patients received norcuron (Organon Teknika, Boxtel, Netherlands) at about 4 mg every 90 min. We measured respiratory variables, including peak pressure and plateau pressure (Servo 940 monitor system, Siemens-Elema, Sweden), and PEEPi was measured with an end-expiratory hold technique. Blood gas analysis was performed with an AVL Omni analyzer (Global Medical Instrumentation, Albertville, Minnesota).

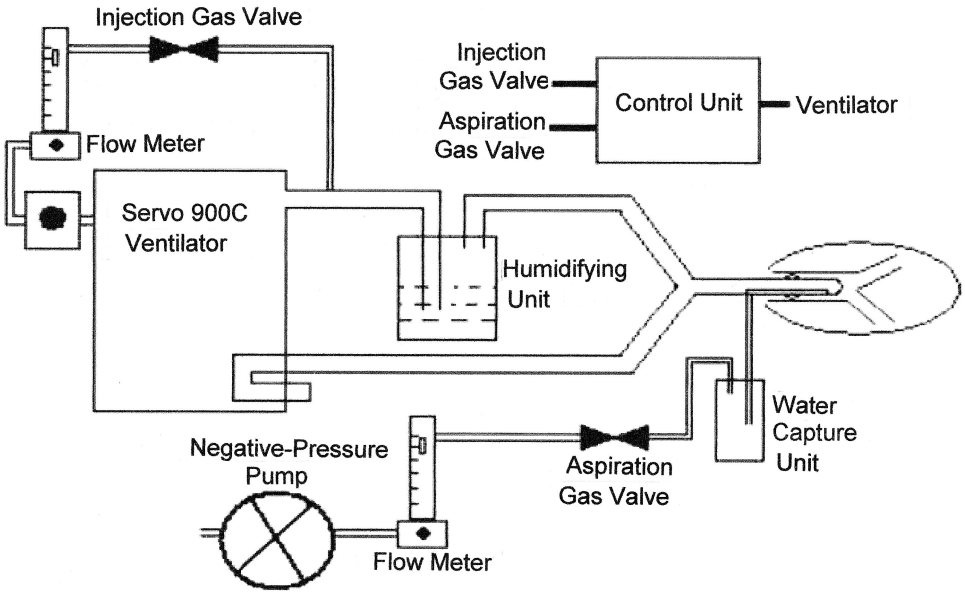

Fig. 1. The aspiration of dead space system consisted of a control unit and lines for aspiration and injection of gas. The catheter was inserted into the tracheal tube through a swivel adapter that connected the tube to the ventilator circuit. Its tip was positioned 2 cm proximal to the tip of the tracheal tube. During expiration the aspiration solenoid valve opens to allow the negative-pressure pump to aspirate gas from the dead space, with the flow meter regulating the volume of aspirated gas. Simultaneously with aspiration, fresh gas is injected into the inspiratory line. The injected gas is tapped from a second outlet of the gas mixer (at the ventilator's set fraction of inspired oxygen) and controlled by a flow meter that allows adjustment of injected volume to match aspirated volume.

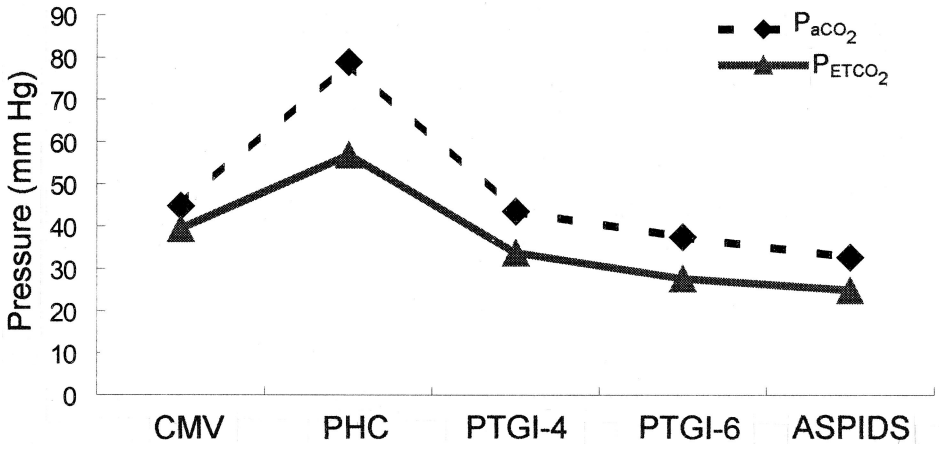

Fig. 2. P_{aCO_2} and end-tidal carbon dioxide (P_{ETCO_2}) were higher during permissive hypercapnia (PHC, which was induced by a 30% tidal-volume reduction) than during conventional mechanical ventilation (CMV). Both P_{aCO_2} and P_{ETCO_2} were reduced by phasic tracheal gas insufflation at 4 L/min (PTGI-4) and at 6 L/min (PTGI-6), and by aspiration of dead space (ASPIDS).

Aspiration of Dead Space System

The ASPIDS system consisted of a control unit and aspiration and injection gas lines (Fig. 1). The aspiration circuitry included a polyethylene end-hole catheter (inner diameter 2 mm, outer diameter 3 mm), a water-capture bottle, an aspiration solenoid valve, a flow meter, and a negative-pressure pump (or the centralized hospital vacuum source). The injection gas circuitry included a flow meter, an injection solenoid valve, and a tube connected to the inspiratory limb of the ventilator circuit.

The ASPIDS system's working principle involves the control unit integrating with the ventilator to gate the aspiration and injection gas solenoid valves to coordinate gas flows with the patient's respiratory pattern. During expiration the aspiration solenoid valve opens to allow the negative-pressure pump to aspirate gas from the dead space, with simultaneous replacement with the same amount of fresh gas into the inspiratory limb. PTGI was delivered by advancing the polyethylene end-hole catheter to the carina and connecting the injection line to the catheter. The PTGI system insufflated fresh gas into the trachea at a set flow during expiration.

Protocol

To enter the study a patient had to be medically stable. An initial blood gas sample was obtained and data from the baseline ventilation state were recorded to document stability. V_T was then adjusted to establish a P_{aCO_2} of about 45 mm Hg, and respiratory variables were recorded after a steady state of 30 min of conventional mechanical ventilation. To induce permissive hypercapnia, V_T was decreased by 30% while maintaining a constant respiratory rate, and respiratory variables were recorded after 30 min. With each subject the effect of ASPIDS was studied during permissive hypercapnia. ASPIDS was administered at 4 L/min, and PTGI was administered at 4.0 L/min (PTGI-4) and 6.0 L/min (PTGI-6), with sufficient intervals between the experimental periods to assure independent intervention effects.

Statistical Analysis

All data are expressed as mean ± SD. Analysis of variance was used to compare treatments. Data were analyzed with commercially available statistical software (STATA 4.0 for Windows, StataCorp, College Station, Texas). Differences were considered statistically significant when $p < 0.05$.

Results

P_{aCO_2} and end-tidal carbon dioxide (P_{ETCO_2}) were significantly lower during ASPIDS and PTGI than during conventional mechanical ventilation, and were lowered more by ASPIDS than by PTGI (Fig. 2). With permissive hypercapnia the peak, plateau, and mean airway pressures were significantly lower during conventional mechanical ventilation. PTGI caused flow-dependent increases in peak, plateau, and mean airway pressures. ASPIDS reduced peak, plateau, and mean airway pressures (Fig. 3). PTGI increased PEEPi, but ASPIDS had no obvious influence on PEEPi (Fig. 4). There were no significant changes in heart rate or arterial blood pressure during PTGI or ASPIDS (Table 2).

Discussion

ASPIDS significantly decreased P_{aCO_2} and P_{ETCO_2} in stable COPD patients suffering respiratory failure. ASPIDS' effect on P_{aCO_2} and P_{ETCO_2} was greater than that of PTGI-4

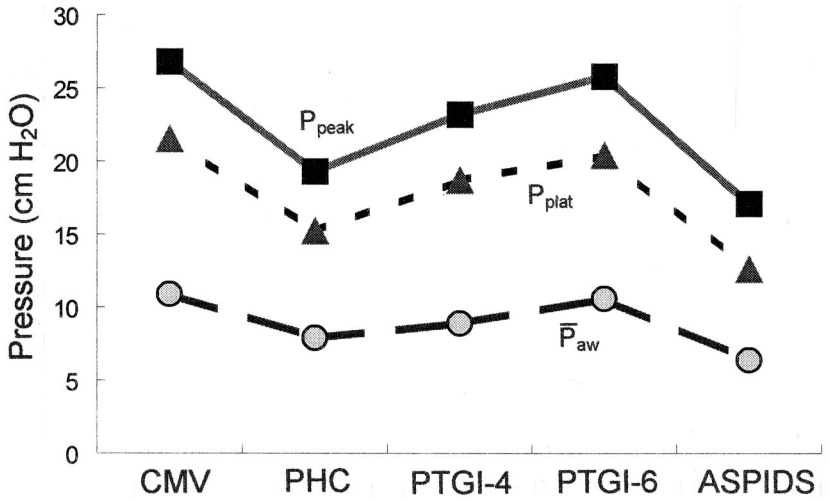

Fig. 3. Airway pressures were lower during permissive hypercapnia (PHC) than during conventional mechanical ventilation (CMV). Phasic tracheal gas insufflation at 4 L/min (PTGI-4) and at 6 L/min (PTGI-6) caused flow-dependent increases in airway pressure. Aspiration of dead space (ASPIDS) lowered airway pressures to that of conventional mechanical ventilation (CMV). P_{peak} = peak pressure. P_{plat} = plateau pressure. $\bar{P}_{aw}$ = mean airway pressure.

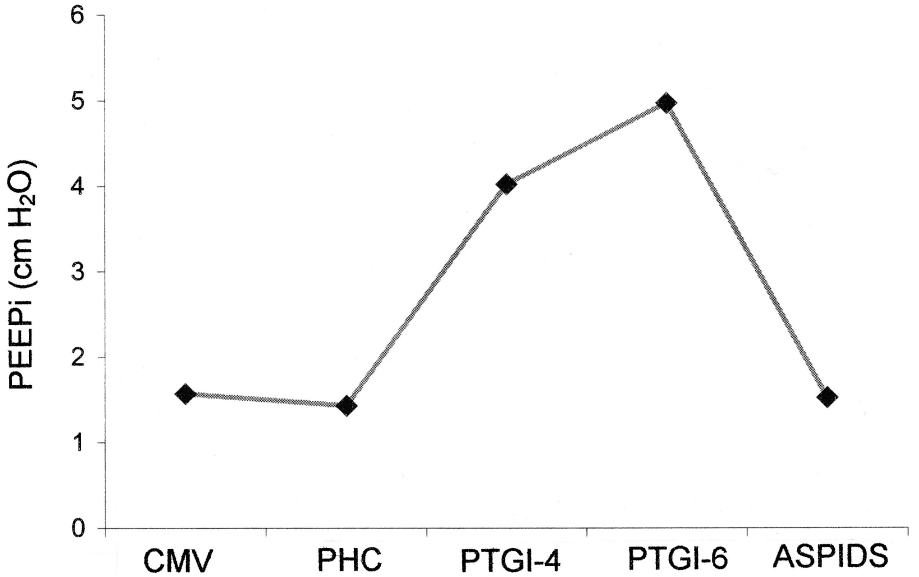

Fig. 4. Intrinsic positive end-expiratory pressure (PEEPi) was higher during phasic tracheal gas insufflation at 4 L/min (PTGI-4) and at 6 L/min (PTGI-6) than during conventional mechanical ventilation (CMV), permissive hypercapnia (PHC), or aspiration of dead space (ASPIDS).

(see Fig. 2). Thus, ASPIDS and PTGI allow lower V_T, enhance ventilation efficiency, and improve carbon dioxide washout.

We speculate that clearance of dead space is the primary mechanism of ASPIDS. V_T equals alveolar gas volume plus total dead-space volume. Total dead-space volume equals alveolar dead space plus anatomic dead space. Since the gas in the anatomic dead space has a high concentration of carbon dioxide at the end of expiration, ASPIDS reduces the carbon dioxide level by reducing anatomic dead space through the aspirating catheter during expiration, thus reducing the volume of carbon dioxide that would re-enter the lungs during the following inspiration. During ASPIDS, gas is simultaneously injected into and drawn out of the inspiratory line to clear the carbon-dioxide-laden tracheal gas and replace it with fresh gas while avoiding the possibility of end-expiratory alveolar collapse.

We propose 3 possible explanations why ASPIDS has a greater carbon-dioxide-clearing effect than PTGI:

1. With ASPIDS the clearance of dead-space carbon dioxide is via the aspirating catheter, which is a more

Table 2. Heart Rate and Arterial Blood Pressure During Conventional Mechanical Ventilation, Permissive Hypercapnia, Aspiration of Dead Space, and Phasic Tracheal Gas Insufflation

	CMV (mean + SD)	PHC (mean + SD)	PTGI-4 (mean + SD)	PTGI-6 (mean + SD)	ASPIDS (mean + SD)	f*	p†
Heart Rate (beats/min)	99 ± 12	104 ± 15	103 ± 14	108 ± 16	106 ± 16	1.27	0.29
Arterial Blood Pressure (mm Hg)	87 ± 9	94 ± 9	88 ± 9	89 ± 9	88 ± 9	2.39	0.08

CMV = conventional mechanical ventilation.
PHC = permissive hypercapnia.
PTGI-4 = phasic tracheal gas insufflation at 4 L/min.
PTGI-6 = phasic tracheal gas insufflation at 6 L/min.
ASPIDS = aspiration of dead space.
*f is the analysis-of-variance equivalent of p.
†There were no significant differences in heart rate or arterial blood pressure between the control (CMV), PHC, and the experimental interventions (PTGI-4, PTGI-6, and ASPIDS).

complete clearance mechanism than PTGI's dilution of tracheal gas by the insufflating flow.

2. The ASPIDS system might clear carbon dioxide (that has moved retrograde) from the portion of the inspiratory line that connects the Y-piece to the tip of the tracheal tube, whereas PTGI clears carbon dioxide mainly from the ETT and common tubing or connectors.

3. The degree and effect of turbulence caused by ASPIDS or PTGI in the Y-piece and adjacent tubes are unknown; a different type of mixing of inspired and expired gas certainly occurs in the tubes. During ASPIDS the gas injected into the airway is aspirated by the catheter within the ETT, whereas during PTGI the insufflated gas jettisons carbon dioxide out the expiratory limb but also possibly retrograde into the inspiratory limb *and/or* toward the lungs, in which case it would subsequently be rebreathed. Therefore, differences in turbulence dynamics in the upper airways and circuitry could explain differences between ASPIDS and PTGI.

Peak, plateau, and mean airway pressures were significantly lower during permissive hypercapnia than during conventional mechanical ventilation, but those pressures increased significantly with PTGI. The increases in peak and plateau pressure were greater than the increase in mean airway pressure. ASPIDS significantly decreased peak, plateau, and mean airway pressure, even during permissive hypercapnia. ASPIDS avoids certain potential problems with PTGI. By reducing airway pressures ASPIDS lessens the risk of ventilator-induced lung injury and decreases intratracheal pressure that can impede expiration.

In COPD and severe asthma, elevated PEEPi reflects increased lung pressure at end-expiration, most often due to dynamic hyperinflation. PEEPi increase can affect hemodynamics and increase the work of breathing. The patients in the present study had low PEEPi before PTGI. PTGI increased PEEPi, but ASPIDS did not increase PEEPi. These results are consistent with the reports of De Robertis et al,[14,15] one of which proposed that the higher PEEPi was because the PTGI catheter decreased the inside diameter of the ETT, thus increasing resistance in the circuitry. Another cause may be that the PTGI flow itself produced a direct resistance to exhalation. Though the ASPIDS system's aspiration catheter also decreases the ETT diameter, the ASPIDS flow assists expiratory flow rather than (as in PTGI) opposing it. Therefore, flow direction, rather than lumen diameter, would seem to be the dominant factor, since ASPIDS was associated with significantly lower PEEPi.

ASPIDS and PTGI had no obvious effects on blood pressure or heart rate, which suggests that ASPIDS and PTGI have no important effect on hemodynamics at relatively low PEEPi in COPD patients.

Conclusions

We have shown that ASPIDS is technically feasible in a clinical setting. However, this preliminary study of only 8 COPD patients suffering respiratory failure does not provide adequate evidence for expanded use of ASPIDS in clinical practice, especially for patients who can't tolerate permissive hypercapnia. Further studies are required to examine certain specific aspects of ASPIDS. For example, we are working on improving the triggering sensitivity for spontaneously breathing patients and preventing occlusions of the aspiration system by airway secretions.

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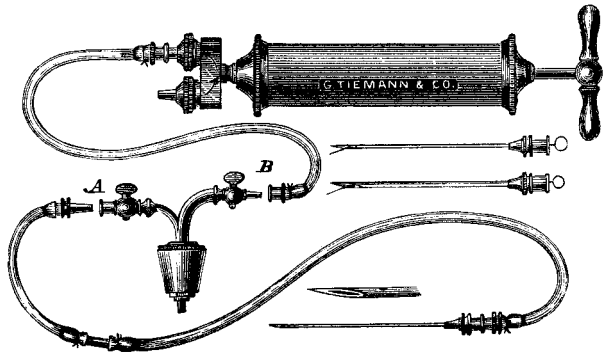

Codman's Aspirator and Injector
The American Armamentarium Chirurgicum.
New York: George Tiemann & Co; 1879.
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