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Oxygen insufflation through the bronchoscope channel for sedation-induced hypoxia: safe and effective.

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Oxygen insufflation through the bronchoscope channel for sedation-induced hypoxia: safe and effective

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Abstract

Objectives: To evaluate both efficacy and safety parameters for insufflation through the bronchoscope as a method of recovery from sedation-induced hypoxia. To explore parameters applicable to use in human beings using an animal model.

Materials and methods: Two adult pigs were sedated enough to depress respiratory drive. The effects of insufflation at 15 l/min (the upper limits of flow that might be used clinically) were then evaluated. Pressure and volume responses to bronchoscopy during intubation and without an endotracheal tube in place were recorded. Several assays were performed for each scenario, with each animal acting as its own control. Recovery from hypoxemia using insufflation was compared with recovery using mechanical ventilation.

Results: Insufflation was effective, with rapid increases in fraction of inspired oxygen (FIO_2), saturation, and partial pressure of arterial oxygen (PaO_2). The rate of recovery using insufflation was faster than that from institution of mechanical ventilation. Insufflation in an intubated animal with cuff inflated led to a rapid and dangerous rise in pressure. With balloon deflated, there were no adverse pressure consequences from insufflation *via* the endotracheal tube at a rate of 15 l/min.

Conclusion: Insufflation through the bronchoscope for episodes of sedation-induced hypoxia should be safe and effective as long as not delivered within a closed system.

Keywords: bronchoscopy, hypoxemia, insufflation, sedation

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Introduction

Bronchoscopy under sedation can be associated with hypoxemia.[1–4] This is largely due to sedation-induced central nervous system depression, with upper airway obstruction and decreased central respiratory drive the two dominant factors.[1,2,4] Suctioning and procedures such as bronchoalveolar lavage also can contribute.[5,6] If a patient is being bronchoscoped through an endotracheal tube, the degree of obstruction and impedance of inspiratory and expiratory flow can be physiologically significant and can contribute to compromise.

When hypoxemia is defined simply as desaturation to an oxygen saturation (SaO_2) ⩽90%, desaturation is very frequent, occurring in up to 75% of bronchoscopies in a recent review.[3] These data are complicated by the fact that transient dips below 90% are not clinically important; it is reasonable to focus specifically upon oxygenation only when desaturation persists or when the SaO_2 drops below 85%. Also, many are not true desaturations – the first intervention should be a pulse oximeter check to be sure that the oximeter is reading correctly. Even with these considerations, clinically relevant desaturation is a relatively frequent occurrence during bronchoscopy, with interventions ranging from increasing FIO_2 to interruption of the procedure with scope removal and the invasive or noninvasive institution of positive pressure ventilation.

One of our approaches to sedation-induced hypoxemia during bronchoscopy has been tracheal insufflation, with oxygen delivery *via* the

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suction channel of the bronchoscope. While this sounds 'intuitive' and there is a moderate literature on insufflation, bronchoscopic tracheal insufflation (BTI) for sedation-induced hypoxia has not been studied in the literature. One reason for this could be the steps involved; historically, BTI has required intermittent manual alterations of the tubing connected to the bronchoscope. A foot pedal that allows toggling back and forth from suction to oxygen administration rapidly and without the need to change tubings has been developed (BronchO2, Thoracent, Inc., Long Island, NY, USA). This would simplify the applications of insufflation during bronchoscopy, and its availability led to this animal study of the physiology of insufflation. The goals were to establish efficacy and safety parameters for BTI to treat sedation-induced hypoxia. For comparative studies, BTI was compared with institution of mechanical ventilation. Whereas boundaries of pressure and hypoxia were being explored, this study was performed on adult pigs.

Study design

The studies were performed on two pigs, which will heretofore be labeled as animal A and animal B. Pigs were chosen with the belief that extrapolation of the results of the study to human beings would be reasonable. The pigs, two Yorkshire cross females, were housed per US Department of Agriculture guidelines. Pig A weighed 49 kg and pig B weighed 48 kg. All studies involved sedation deep enough such that the animals would have had no proprioceptive capacity and no awareness of discomfort. The animals were euthanized upon termination of all studies using pentobarbital plus sodium phenytoin as per the guidelines of the University of Minnesota's Institutional Animal Care and Use Committee.

An intravenous (IV) catheter was placed in the ear vein. Normal saline at 5–10 ml/kg/h was given for fluid maintenance. Propofol (1–6 mg/kg, IV) was given for intubation. Ongoing sedation consisted of a combination of ketamine 15 mg/kg/h, valium 1 mg/kg/h, and buprenorphine 0.5 mcg/kg/h. This sedation was chosen both for animal comfort and to intentionally depress respiratory drive. All pressure studied were performed on animal A intubated with a #7.5 (internal diameter) Shiley endotracheal tube (ETT) (Medtronic, Minneapolis, MN, USA) in order to mimic pressure consequences of insufflation for an intubated human being. Animal B was intubated with a #10 Shiley ETT for maintenance and rescue (see below). Ventilation was effected with a volume-controlled ventilator (Narkomed 2A, Drager, Telford, PA, USA) set to a tidal volume of 8–12 ml/kg, respiratory rate 20/min, FiO_2 21%, and positive end-expiratory pressure of 5. These settings were designed to keep oxygen saturation above 90% and end-tidal carbon dioxide ($ETCO_2$) between 35 and 45 mmHg between interventions. Bronchoscopy on animal A was performed with a therapeutic bronchoscope with a diameter of 6.2 mm and a 2.8-mm working channel. Bronchoscopy on animal B was performed with a bronchoscope with a 5.5-mm diameter and a 2-mm working channel (Olympus America, Center Valley, PA, USA).

Oxygen was insufflated *via* the 'BronchO2' device. The BronchO2 is a small foot pedal with connectors for both suction and oxygen. Suction tubing connects the BronchO2 device to the suction port of the bronchoscope. Depression of the BronchO2 pedal (with the suction button on the scope depressed/activated) changes the suction channel of the bronchoscope from suction to insufflation at the specified oxygen flow rate. For all interventions in this study, the oxygen flow rate was set at 15 l/min. For all insufflation, the distal end of the bronchoscope was positioned in the distal trachea just above the carina.

Hemodynamics, electrocardiogram, $ETCO_2$, and oxygen saturation were monitored and recorded. Peak airway pressures (PAPs) and $ETCO_2$ were monitored *via* the ventilator. Fractional O_2 from the exhalation port was also recorded and is reported herein as the FIO_2, as it best represented the alveolar mixture effected by the combination of any ventilator-delivered gasses and insufflation. Blood pressure was measured directly *via* a cannula inserted into the femoral artery. A rectal temperature probe was placed, and body temperature was maintained at or above 98°F with a combination warming/cooling air unit. Blood gases were processed using a GEM 4000 ABG machine (Werfen, Bedford, MA, USA). A C-arm X-ray unit was used to check for pneumothorax. Statistical analyses were performed using Microsoft Excel.

The experiments were designed to study the physiologic responses to intermittent insufflation.

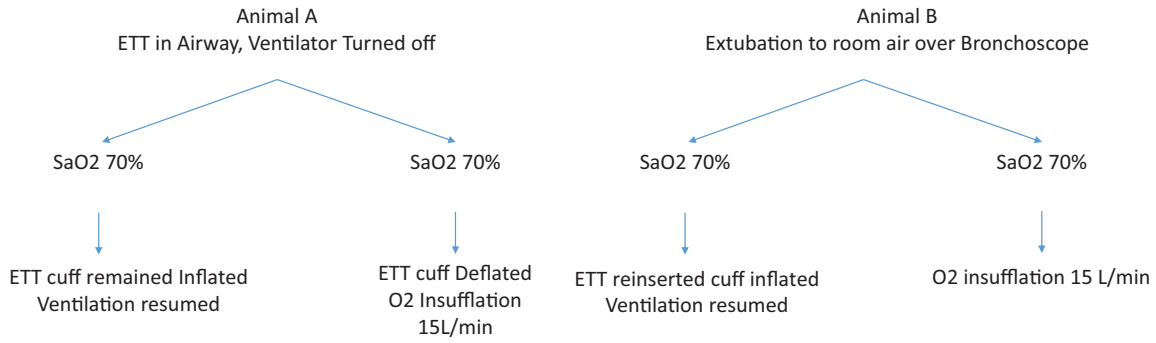

Figure 1. Sequence of events for induced desaturation with rescue. Re-institution of mechanical ventilation for rescue was compared with insufflation. For insufflation, ETT was in place with balloon deflated (animal A) or removed (animal B).

They studied two primary outcomes in each of two separate 'clinical settings'. The 'clinical settings' were intubation and the absence of intubation, both commonly encountered in the bronchoscopic evaluation and treatment of patients. The primary outcomes were the pressure and oxygenation results of insufflation at 15l/min.

All assays of the pressure sequelae of insufflation were performed on animal A, all with the #7.5 Shiley ETT, in place. An ETT, even with the cuff deflated, is a form of upper airway obstruction, and a bronchoscope in the tube further decreases cross-sectional area for gas flow (The therapeutic bronchoscope with its larger diameter was intentionally used for studies performed with the ETT in place, as it maximized obstruction to exhalation – the goal of the study was to elucidate any issues that could emerge during insufflation in clinical practice.). First, PAP was measured on the ventilator settings noted above with a bronchoscope in place in the airway. The ETT cuff was then deflated and PAP was measured after 15 s of insufflation. This was repeated three more times with a 3-min recovery between assays. Insufflation was then performed with the cuff of the ETT inflated. This was done only once given the adverse pressure results. Given the pressure results of insufflation with ETT in place and cuff deflated (see below), studies of the pressure sequelae of insufflation at 15l were not repeated for extubated animals. Chest radiographs were used to monitor for pneumothorax and were obtained after intubation, after 15 min, and after all trials had been completed.

Both animals were used for studies of the impact of insufflation on oxygenation. The first study measured the impact of insufflation on FIO_2. The subsequent studies looked at insufflation *versus* re-institution of mechanical ventilation after induction of hypoxia by cessation of ventilatory support. Interventions were initiated when desaturation to 70% had occurred. Figure 1 outlines the sequences of events for the re-oxygenation assays The assays were performed in pairs, alternating an assay using resumption of ventilatory support for rescue with an assay using insufflation for rescue. Thus, each animal served as its own control for each assay. Two pairs of assays were performed with animal A and 3 pairs with animal B, for a total of 10 assays.. For animal A, the ETT was left in place. The cuff was inflated for resumption of ventilatory support, and insufflation was performed with the cuff deflated. Animal B was extubated over the bronchoscope to allow/induce hypoxia. For re-oxygenation, the ETT was reintroduced over the bronchoscope for the ventilation arm and insufflation was performed through the bronchoscope without reintubation for the insufflation arm.

Results

Pressure studies

The results of the pressure studies are shown in Figure 2. Insufflation with the balloon deflated caused no significant change in PAP, with mean pressure 25.5 mmHg [standard deviation (SD) = 0.6 mm Hg] pre-insufflation and 25.8 mmHg (SD 1.3 mmHg) with insufflation.

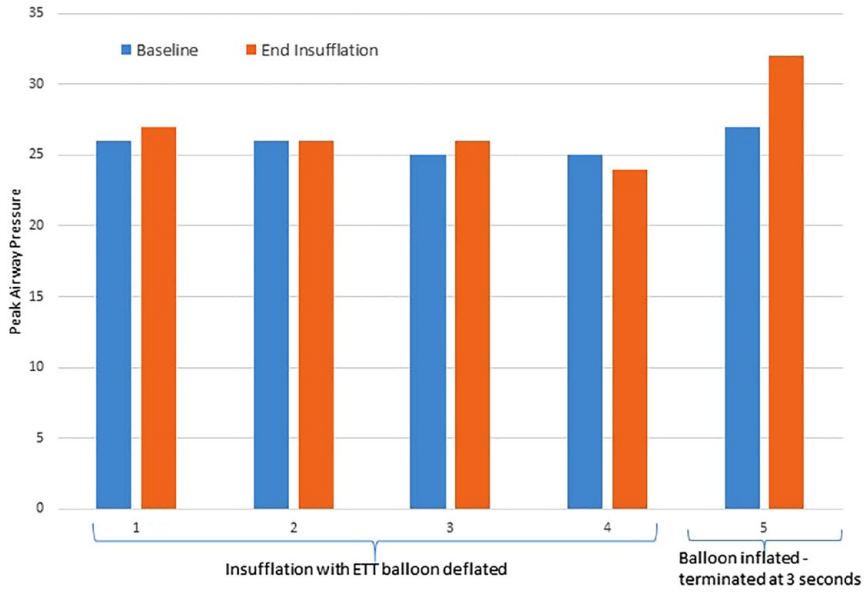

Figure 2. Pressure sequelae of insufflation using large bronchoscope within a #7.5 Shiley ETT. Baseline was obtained during mechanical ventilation with bronchoscope in ETT. Ventilation was then ceased, and insufflation values were obtained with insufflation alone and ETT balloon deflated (1–4) or inflated (5). For each assay, the animal served as its own control. Given the adverse pressure sequelae of inflation with balloon inflated, this assay was terminated at 3 s and not repeated.

Insufflation at 15 l with an ETT in place and the balloon inflated led to a precipitous rise in pressure (from 27 to 32 in 3 s). No further assays were performed with the balloon inflated, as the results were deemed definitive and physiologically dangerous. There was no hemodynamic instability during any of these assays. Chest radiographs showed lack of pneumothorax (It is assumed that persistent insufflation at 15 l with cuff inflated would have led to pneumothorax and hemodynamic instability, but it was felt that it was not necessary to induce these changes.). Given the fact that insufflation with a #7.5 Shiley in place and cuff deflated did not significantly increase intrathoracic pressure, the study was not repeated in extubated animals, as expiratory resistance is lower without an ETT in place.

Hypoxia rescue studies

The impact of insufflation upon FIO_2 (as defined above) is shown in Figure 3. In the first assay, conversion directly from mechanical ventilation with an FIO_2 of 21% to insufflation using O_2 at 15 l/min with the balloon deflated, there was a marked increase in FIO_2, with a mean of 77% after 10 s of insufflation.

Desaturation was then induced, and the pulse oximetry results of insufflation *versus* mechanical ventilation as an intervention for desaturation are shown in Figure 4. While interventions were begun in every case when saturation reached 70%, in some cases, saturations initially continued to decline, and nadirs were used for analysis. 'Recovery' for this study was the point in time from initiation of rescue (insufflation or mechanical ventilation) at which saturation postintervention had reached the prehypoxia saturation. Both interventions successfully reversed desaturation, with no significant difference between the two – for insufflation *versus* mechanical ventilation mean baseline saturations, mean nadirs, and mean recovery saturations were each within 3% between groups. Time to recovery was significantly shorter with insufflation (see Table 1); for insufflation, recovery occurred in 45 ± 21 s, while for mechanical ventilation, recovery occurred in 116 ± 47 s ($p = 0.025$).

Arterial blood gasses were collected at nadir and at time of recovery for five insufflation recoveries and four mechanical ventilation recoveries. Individual results are presented graphically in Figure 5. Mean changes are presented in Table 2. Insufflation effected greater increases in PaO_2.

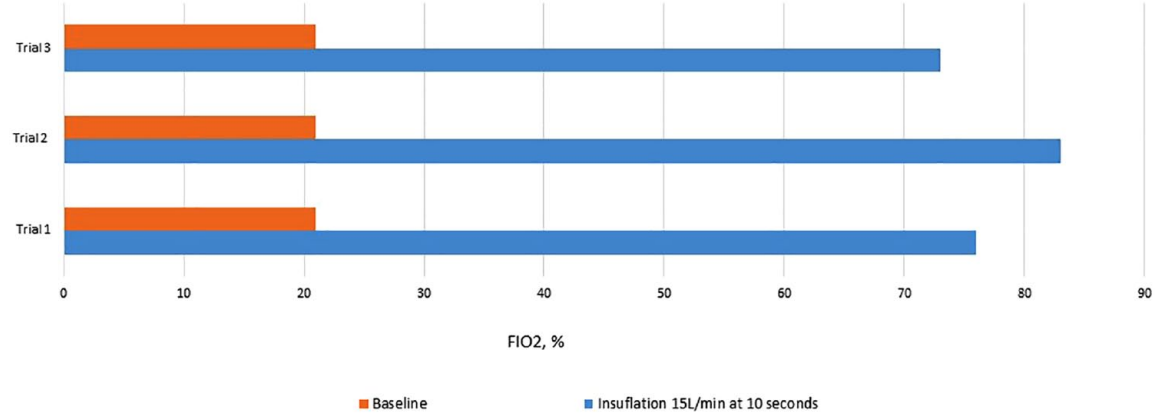

Figure 3. FIO_2 sequelae of insufflation for 10 s at 15 l/min. For each assay, the animal served as its own control.

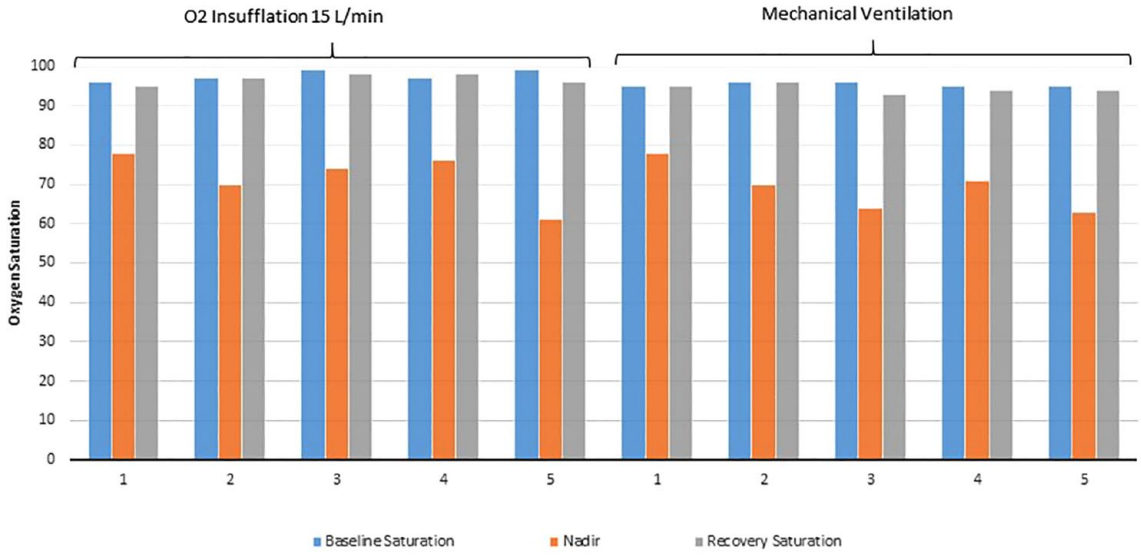

Figure 4. Rescue studies. Re-institution of mechanical ventilation (at an FIO_2 of 21%) was compared with oxygen insufflation at 15 l/min. Each animal served as its own control for each assay.

Table 1. Time to recovery (s).

Time to recovery in seconds	Insufflation	Mechanical ventilation
1	82	150
2	33	110
3	35	174
4	35	90
5	41	55
Mean ± SD	45 ± 21*	116 ± 47*

SD, standard deviation.

With insufflation, there were slight increases in pCO_2 (mean 4 mmHg) and decreases in pH (mean 0.04), while for mechanical ventilation, there were mean decreases in partial arterial pressure of carbon dioxide (mean 10 mmHg) and increases in pH (mean 0.04).

Discussion

This study demonstrates that tracheal insufflation through a bronchoscope situated just above the carina can rapidly correct hypoxemia in apneic pigs. There is a rapid rise in pO_2 which is accompanied by a small increase in pCO_2 and decrease

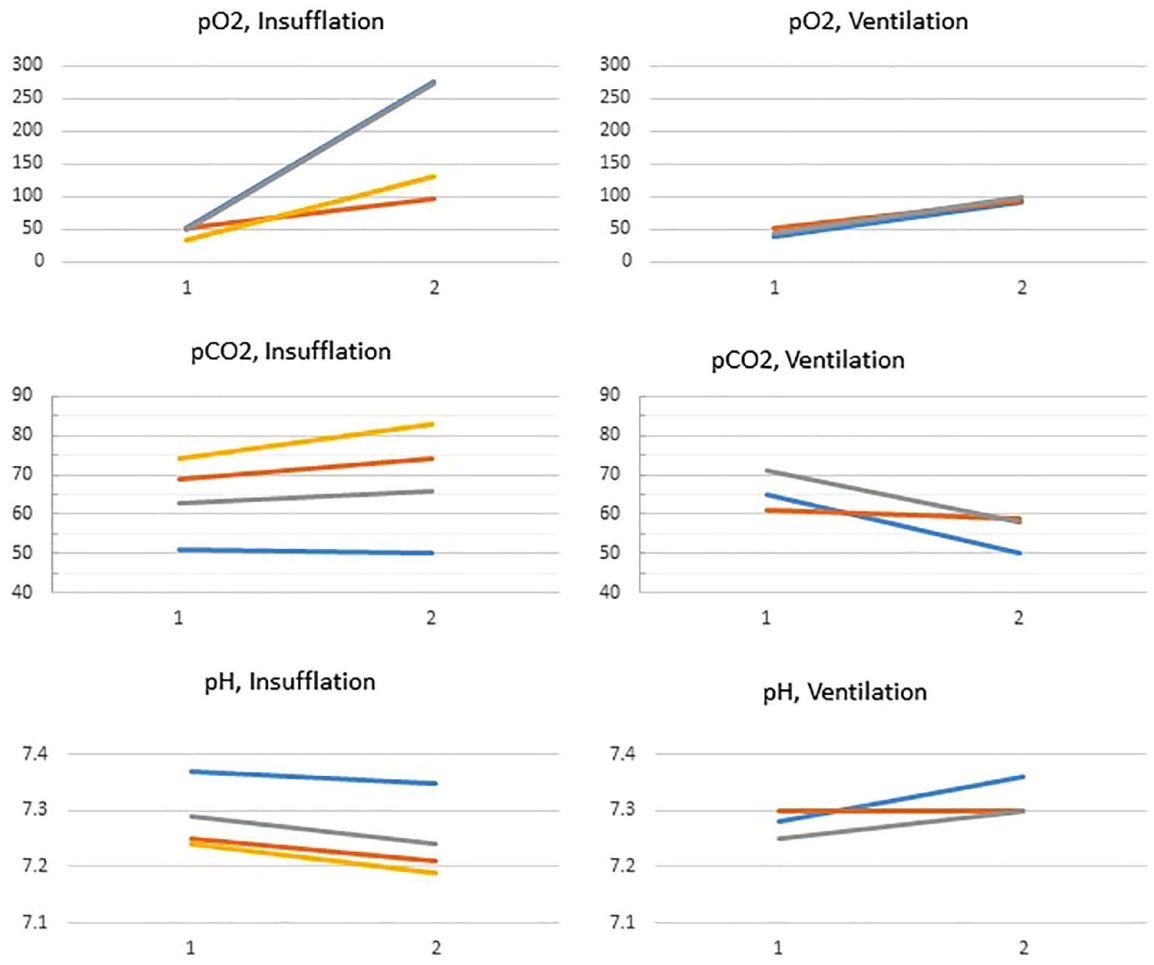

Figure 5. Arterial blood gasses were collected at approximate nadir of saturation (1) and after rescue (2) *via* insufflation (left side) *versus* mechanical ventilation (right side) (four sets were collected for insufflation and three sets for ventilation).

Table 2. Mean blood gas changes, nadir to SaO_2 recovery.

	pO_2	pCO_2	pH
Insufflation	+148	+4	−0.04
Mechanical ventilation	+49	−10	+0.04

in pH. Tracheal insufflation *via* the bronchoscope could thus be an effective and efficient intervention for transient periods of respiratory depression incurred during bronchoscopy. There is one caveat, that insufflation with flow levels as high as the 15 l used in this study should not be utilized in a closed system.

The findings of this study extend prior data; the physiology of bronchial insufflation has been studied relatively extensively both as a form of ventilatory support and as a sole form of ventilation in conditions of apnea. The studies most relevant to this study were done by Slutsky *et al.*[7] Slutsky *et al.* showed that tracheal insufflation in apneic dogs could maintain oxygenation for up to 5 h. There was a slow rise in pCO_2 and a slow decrease in pH, both of which did reach an equilibrium over time. The steady state was a respiratory acidosis from which the animals could recover completely, with no adverse sequelae. They showed that active ventilation did occur, with molecular diffusion significantly abetted by cardiogenic oscillations.[8] They also showed that

gas exchange was most effective when the distal end of the insufflation catheter was from just above to 3 cm below the main carina.[7] The goal of this study was to extend the data on insufflation with an exploration of risks and safety parameters that could be relevant to bronchoscopy in human beings. The animal model allowed exploration of implications for high flow rates, for severe desaturation, and for both intubated and extubated scenarios.

Hypoxia associated with bronchoscopy has two relatively unique features. First, the airway is already cannulated. Second, it is relatively transient – the drugs used for sedation during bronchoscopy have relatively short durations of action. As mentioned, the authors have been using insufflation *via* the bronchoscope for many years, and we assume that others too consider insufflation *via* the bronchoscope to be part of the 'bronchoscopy toolbox'. What is surprising is that insufflation *via* the bronchoscope is not an integral part of the conversation in the literature about sedation-induced hypoxia; we were able to find one report of the efficacy of bronchoscopic insufflation during intubation over a bronchoscope,[9] and two case reports of insufflation *via* the bronchoscope causing pneumothoraces due to obstructed airways, the one contraindication identified in this study.[10,11] The literature on insufflation does not discuss insufflation *via* a bronchoscope for treatment of hypoxia,[12–14] and a recent review of management of oxygenation during bronchoscopy did not even mention insufflation.[15] Insufflation for re-expansion of atelectasis has been reported.[16]

The goal of this study was to define parameters that can be extrapolated to the use of thoracic gas insufflation in bronchoscopy in clinical practice. Pigs were selected as they are relatively large mammals with airway diameters close to those of human beings. In our clinical (human) experience, flow rates of 6 l *via* the bronchoscope have usually been effective for oxygenation, which is in line (if flow rates are adjusted for weight) with the findings of Slutsky *et al.*[8] The toggle device used in this study can accommodate any flow rate up to 15 l/min; the flow rate of 15 l utilized for this study was based upon a desire to determine whether flow rates at the upper limits of what might be utilized clinically had any pressure implications, favorable or unfavorable (There is also some evidence that higher flow rates lower pCO_2 more effectively.).[17]

Some of the statistical differences between insufflation and ventilator rescue in this study can be considered to be 'artificial'; the FIO_2 of the ventilator was left at 21% for rescue in these studies and in clinical practice would have been 100% or close to 100%. Both interventions were effective. The potential advantages of insufflation consist of the capacity to intervene almost instantly and to return to the procedure without a change in airway access.

The primary indication for insufflation in the setting of bronchoscopy would be hypoxia, with insufflation an efficient bridging therapy. The availability of relatively instantaneous insufflation would, however, allow brief initiation of insufflation for other issues such as occlusion of the suction channel by a mucus or blood clot. Insufflation can at times also improve visualization. The primary contraindication to insufflation would be a closed system. Insufflation through a bronchoscope in an intubated patient with ETT balloon inflated would be one example. Another would be insufflation with the bronchoscope wedged in an airway; although insufflation for atelectasis has been reported,[16] this, when performed, should be done with caution and awareness of the pressure risks.

Conclusion

Insufflation has been studied extensively, but there is surprisingly little mention in the literature of insufflation for hypoxemia incurred during bronchoscopy. This animal study demonstrates that insufflation can be a safe and rapidly effective intervention. There is one caveat; high flow rates such as 15 l/min should not be insufflated into a closed system. This study (and prior research)[8,9] supports the concept that insufflation would be of value for the transient hypoxias encountered during bronchoscopy.

Declarations

Ethics approval and consent to participate

Approval for this study was obtained *via* University of Minnesota's IACUC (Protocol 2106A39160). No human subjects were involved.

Consent for publication
Not applicable.

Author contributions
Wissam Abouzgheib: Conceptualization; Data curation; Formal analysis; Funding acquisition; Investigation; Methodology; Project administration; Supervision; Validation; Visualization; Writing – original draft; Writing – review & editing.

Henry Miller: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Writing – original draft; Writing – review & editing.

Thaddeus Bartter: Data curation; Formal analysis; Methodology; Validation; Writing – original draft; Writing – review & editing.

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Competing interests
The authors declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: Drs HM and TB had no conflicts of interest. Dr WA participated in the development of the device used to provide tracheal insufflation. While this could be construed as a conflict of interest, this study looks at physiologic parameters, and the results would be the same regardless of which method was used to provide insufflation. These facts are nevertheless noted in the text of the paper for clarity.

Availability of data and materials
The raw data reported in this document are available from the primary author, WA.

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