ORIGINAL ARTICLE

Arterial pCO_2 changes during thoracoscopic surgery with CO_2 insufflation and one lung ventilation

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ABSTRACT

Introduction: The respiratory effects (changes in pH and PaCO2) of carbon dioxide insufflation in thoracoscopic surgery in adult patients with pulmonary disease were not documented previously.
Methods: In this observational study 21 patients scheduled for elective thoracoscopic surgery with one lung ventilation using a double lumen tube and intraoperative carbon dioxide insufflation were studied. Arterial blood gas findings were correlated with demographic and intraoperative variables.
Results: When compared to baseline (10-15 minutes of one lung ventilation before carbon dioxide insufflation), carbon dioxide insufflation lowered the pH, 7.31 ± 0.08 vs 7.40 ± 0.05 ($p < 0.001$) caused increased PaCO2, 53 ± 12 vs 42 ± 6.0 ($p < 0.001$) at 40-60 minutes after carbon dioxide insufflation. These derangements in arterial blood gases persisted in the post-anesthetic care unit with pH 7.33 ± 0.04 vs 7.40 ± 0.05 ($p < 0.001$) and PaCO2 51 ± 6.7 vs 42 ± 6.0 ($p < 0.001$). Moderate hypercarbia defined as PaCO2 > 50 mmHg, developed in 12 of 21 patients (57%) and was associated to lower FEV1/FVC ratios 60 ± 21 vs 81 ± 3%, older age 69 ± 9 vs 56 ± 17 years, and history of smoking, 43 ± 30 vs 16 ± 21 pack years, $p < 0.05$.
Conclusion: Intrathoracic carbon dioxide insufflation causes significant derangements in pH and PaCO2 which is worse in patients with lower FEV1/FVC, increased age and smoking history.

Keywords: *thoracic surgery, one lung ventilation, CO2 insufflation.*

INTRODUCTION

The physiological effects of carbon dioxide (CO_2) insufflation in laparoscopic surgery have been well studied (1). The pneumoperitoneum causes cardiovascular and respiratory pathophysiology which includes a decrease in venous return, ejection fraction, stroke volume and functional residual capacity, as well as an increase in ventilatory pressures, hypoxemia and hypercarbia (2). Most of these effects will revert back to normal upon deflation of the abdomen. Intrathoracic insufflation of CO_2 increases central venous pressure and pulmonary capillary wedge pressure, decreases cardiac index and cause tachycardia (3). Due to these changes the use of high insufflation pressures (8-12 mm Hg) during thoracoscopy has been cautioned against in hypovolemic patients and those with poor left ventricular function (4). The effect of CO_2 insufflation in thoracoscopic surgery on respiratory physiology has however not been extensively documented. Studies in

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either patients with minimal pulmonary disease undergoing cardiac surgery (5,6) or healthy patients undergoing thymectomy (7) or thoracoscopy (8), either found no abnormalities in arterial or end tidal CO_2 ($ETCO_2$) or a rise of 10 mm Hg or less but focused on the hemodynamic effects.

The use of CO_2 insufflation is not considered the standard surgical exposure for thoracoscopic surgery for thoracic resections. More commonly passive lung deflation is used with the exception of pediatric patients (9-11).

The respiratory effects of passive lung deflation with one lung ventilation (OLV) for thoracic resections have been documented previously (12,13). These studies noted problems with oxygenation but no difficulties with ventilation. The surgeons at our institution however feel strongly that the use of thoracic CO_2 insufflation provides better visualization in adults and it is the predominant method used by them (14).

As anaesthesiologists we were unsure of the ventilatory effects of CO_2 insufflation in an older patient population with coexisting lung pathology undergoing thoracoscopic surgery. Our goal was therefore to evaluate perioperative $ETCO_2$ and $PaCO_2$ as well as capture any clinically significant complications of hypercarbia in patients undergoing thoracoscopic surgery with CO_2 insufflation.

METHODS

After obtaining University of Western Ontario ethics review board approval and written informed consent, patients booked for elective video assisted thoracoscopic surgery (VATS) scheduled for longer than two hours at the Victoria Hospital, London Health Sciences Centre were recruited (from October 2007 to December 2008). Patients were excluded for age less than 18 years, pregnancy and inability to insert an arterial line. Patients enrolled in the study had the following demographic data collected: age, gender, height, weight, ASA class, comorbidities, preoperative arterial blood gases and pulmonary function tests.

The patients underwent their elective VATS procedure under general anaesthesia (GA) with one lung ventilation using a double lumen tube (DLT). As per routine, a radial arterial line was placed either before or immediately after intravenous induction of GA and intubation.

The patient was then placed in the 90-degree lateral decubitus position with the operative side up. Proper positioning of the DLT was confirmed with bronchoscopy before and after repositioning of the patient. OLV was initiated and anaesthesia was maintained with a potent inhalational agent in air/oxygen supplemented by a thoracic epidural or intermittent boluses of opioid.

Intraoperatively arterial blood gas (ABG) samples with corresponding vital signs and ventilation parameters were collected at 10-15 minutes of OLV before CO_2 insufflation (T1), 15 (T2) and 40-60 minutes of OLV after CO_2 insufflation (T3) and 15 minutes after CO_2 deflation on two lung ventilation (TLV) (T4), and after post-anesthetic care unit (PACU) arrival.

As this was an observational study, ventilatory and anesthestic management including respiratory rate and/or ventilation pressures were left to the discretion of the attending anesthesiologist, however these variables were recorded for subsequent analysis.

Any intraoperative or postoperative complications as well as patient length of stay in hospital were also noted.

Statistical analysis was performed using Student's t tests, ANOVA and chi-squared analysis. Statistically significant differences were considered with a $p < 0.05$.

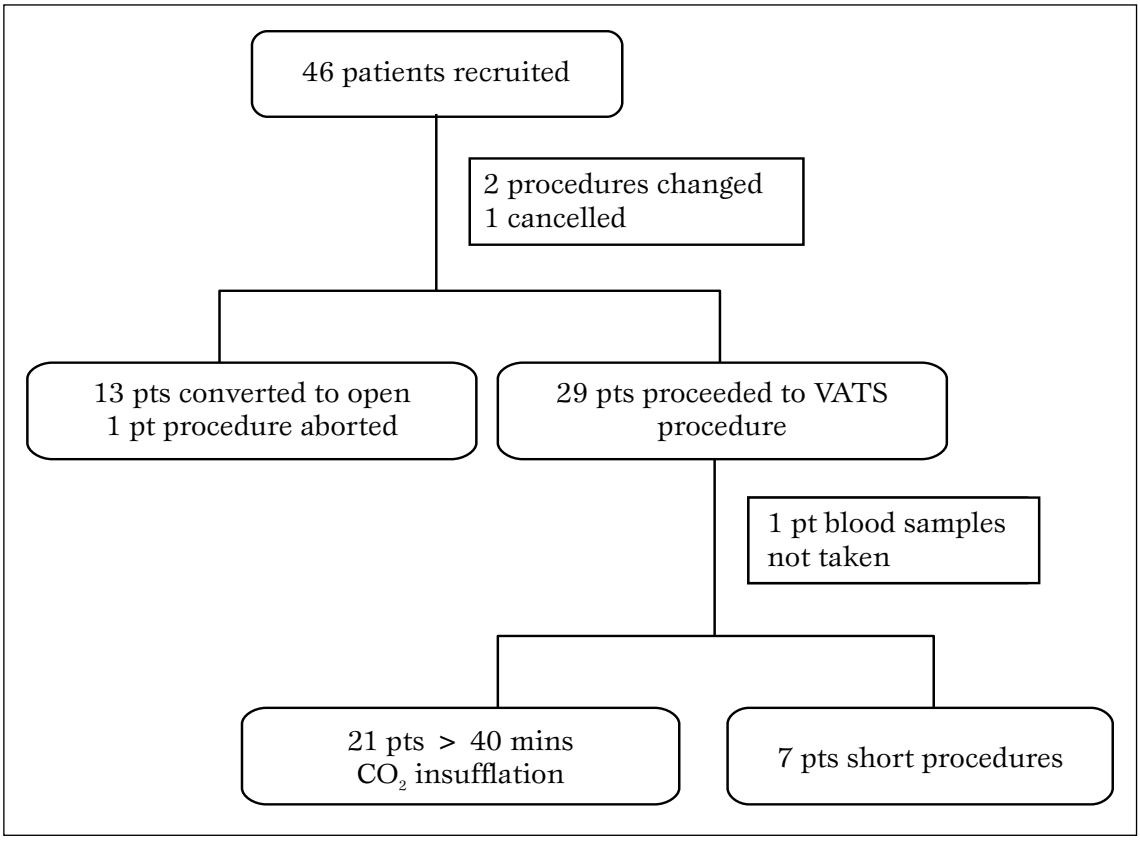

Figure 1 - *Patient recruitment and allocation.*

RESULTS

Forty-six patients gave written informed consent and were enrolled into the study. They are depicted in *Figure 1*. Three patients were immediately excluded from the study when two had their procedure changed to open thoracotomy and one was cancelled. The remaining 43 patients proceeded to have their VATS procedure as scheduled. One patient was found to have unresectable disease and the procedure was aborted.

Thirteen patients had their thoracoscopic surgery switched to an open thoracotomy for various surgical indications and as such were excluded. Three patients had their VATS procedure converted to open thoracotomy after reaching the primary endpoint and their ABG analyses were included in the study. Of the 29 patients who had minimally invasive surgery, one patient was omitted due to lack of appropriate blood samples drawn. Seven of these procedures involved less than 40 minutes of CO_2 insufflation time and they were also eliminated from the analysis.

A total of 21 patients reached our primary endpoint of 40 minutes of CO_2 insufflation. Their procedures included video-assisted wedge resections (8 patients), lobectomies (9 patients), decortications (2 patients) and other (2 patients). Their demographic data, preoperative PFTs and ABGs are listed in *Table 1*. The average thoracic CO_2 insufflation pressures were 12 $\pm$ 1.4 mm Hg for a

Table 1 - Summary of Baseline Characteristics

	All patients (21 total)	$PaCO_2$ < 50mm Hg (9 pts)	$PaCO_2$ > 50 mm Hg (12pts)
Age (years)	64 ± 15	56 ± 17	69 ± 9.1*
Gender	13F:8M	6F:3M	7F:5M
Height (m)	1.66 ± 0.09	1.64 ± 0.09	1.67 ± 0.08
Weight (kg)	80.1 ± 17	78.8 ± 11.8	77.9 ± 22.8
BMI	28 ± 6.6	29 ± 6.9	28 ± 7.0
ASA II/III/IV	2/15/4	2/7/0	0/8/4
Smokers	16/21	5/9	11/12
Pack-year History	31 ± 29	16 ± 21	43 ± 30*
PFT's			
FEV_1 (%)	74 ± 24	87 ± 4.9	71 ± 27
FEV_1/FVC (%)	68 ± 20	81 ± 13	60 ± 21*
DLCO (%)	77 ± 22	85 ± 25	75 ± 22
ABG			
pH	7.44 ± 0.03	7.44 ± 0.042	7.44 ± 0.030
$PaCO_2$ (mm Hg)	39 ± 4.0	38 ± 4.4	40 ± 3.6
HCO_3^- (mEq/L)	27 ± 2.6	28 ± 2.3	27 ± 2.9

Values are presented in mean ± standard deviation. p value is comparison of moderate hypercarbic versus mild hypercarbic groups. * denotes $p < 0.05$.

mean duration of 103 ± 74 (40-285) minutes. When compared to T1, CO_2 insufflation significantly lowered the pH, 7.31 ± 0.08 ($p < 0.001$) and caused $PaCO_2$ elevation, 53 ± 12 mm Hg ($p < 0.001$) at T3 as shown in *Table 2*.

Ten of the twenty-one patients (47%) were moderately hypercarbic (defined as an arterial CO_2 > 50 mm Hg) at 15 minutes and 12 of 21 (57%) patients had become moderately hypercarbic by 50 minutes of CO_2 insufflation despite increases in respiratory rate. Nine of the 21 patients remained only mildly hypercarbic (defined as an arterial CO_2 < 50 mm Hg). *Table 3* compares the ventilation values for the two groups. The

Table 2 - Ventilation Summary for All Patients

	Baseline	T1 - OLV 10-15 mins	T2 - CO_2 + 15 mins	T3 - CO_2 + 40-60 mins	T4 - TLV 15mins	PACU
pH	7.44 ± 0.03	7.40 ± 0.05	7.33 ± 0.07*	7.31 ± 0.08*	7.35 ± 0.06*	7.33 ± 0.04*
$PaCO_2$ (mmHg)	39 ± 4.0	42 ± 6.0	53 ± 13*	53 ± 12*	48 ± 5.3*	51 ± 6.7*
HCO_3^- (Meq/L)	27 ± 2.6	27 ± 2.2	27 ± 2.5	26 ± 2.0	26 ± 1.8	27 ± 2.7
$ETCO_2$ (mmHg)		33 ± 4.3	40 ± 4.3*	41 ± 5.9*	40 ± 5.9*	
Respiratory Rate/minute		11 ± 1.8	11 ± 2.2	12 ± 2.2	13 ± 4.0	
Tidal Volume (mL/kg)		6.2 ± 1.6	6.0 ± 1.4	5.8 ± 1.4	5.5 ± 2.7	
Peak Airway Pressure (mmHg)		30 ± 5.8	33 ± 5.1	33 ± 5.2	26 ± 6.1	

Values are presented in mean ± standard deviation. * denotes p value < 0.001 when compared with T1.

Table 3 - Ventilatory Comparison Between Mild Hypercarbic and Moderate Hypercarbic Groups

	Baseline	T1- OLV 10-15 mins	T2 - CO_2 + 15 mins	T3 - CO2 + 40-60 mins	T4 - TLV 15mins	PACU
Mild Hypercarbic Group (9 pts)						
pH	7.44 ± 0.04	7.43 ± 0.03	7.38 ± 0.03	7.38 ± 0.04	7.38 ± 0.04	7.34 ± 0.06
$PaCO_2$ (mm Hg)	38 ± 4.4	40 ± 5.0	45 ± 4.5	44 ± 4.2	44 ± 4.3	51 ± 7.4
HCO_3^-(mEq/L)	28 ± 2.3	27 ± 2.4	26 ± 1.8	26 ± 1.3	26 ± 2.0	27 ± 2.5
$ETCO_2$ (mm Hg)		32 ± 3.8	38 ± 2.8	37 ± 3.2	36 ± 1.8	
Respiratory Rate/minute		11 ± 2.0	11 ± 2.1	12 ± 1.8	12 ± 2.4	
Tidal Volume (mL/kg)		6.4 ± 1.7	5.9 ± 1.1	5.9 ± 1.1	5.9 ± 1.2	
Peak Airway Pressure (mmHg)		30 ± 5.0	33 ± 5.7	34 ± 5.9	27 ± 4.4	
	Baseline	**OLV 10-15 mins**	**CO_2 + 15min**	**CO_2 + 50min**	**TLV 15min**	**PACU**
Moderate Hypercarbic Group (12 pts)						
pH	7.44 ± 0.03	7.38 ± 0.06	7.29 ± 0.07*	7.26 ± 0.07*	7.32 ± 0.05*	7.32 ± 0.03
$PaCO_2$ (mm Hg)	40 ± 3.6	44 ± 6.1	58 ± 14 #	60 ± 11 *	51 ± 4.2 +	51 ± 6.6
HCO_3^-(mEq/L)	27 ± 2.9	26 ± 2.0	28 ± 2.9	27 ± 2.3	27 ± 1.8	26 ± 2.8
$ETCO_2$ (mm Hg)		34 ± 4.5	42 ± 4.5 #	44 ± 5.5 +	42 ± 6.5 #	
Respiratory Rate/minute		11 ± 1.7	12 ± 2.4	13 ± 2.45	13 ± 3.0	
Tidal Volume (mL/kg)		6.1 ± 1.6	6.1 ± 1.7	5.7 ± 1.6	6.8 ± 1.6	
Peak Airway Pressure (mmHg)		30 ± 6.7	33 ± 4.9	32 ± 4.7	25 ± 7.2	

Values are presented in mean ± standard deviation. p-values comparing the two groups are indicated in parentheses in the bottom half of the chart. * denotes $p < 0.001$, + $p < 0.01$, # $p < 0.05$.

moderately hypercarbic group had a pH of 7.26 ± 0.07 and $PaCO_2$ 61 ± 11 mm Hg vs 7.38 ± 0.04 and 44 ± 4.2 in the mildly hypercarbic group at T3. In the moderate hypercarbic patient group, the arterial-ET-CO_2 difference widened to 16 ± 8.3 mm Hg ($p < 0.05$) compared to 7.4 ± 3.0 mm Hg in the mildly hypercarbic patients at T3.

Comparison of baseline demographic data, preoperative PFTs and ABGs between the moderate hypercarbic and mildly hypercarbic groups is also shown in *Table 1*.

Moderate hypercarbic patients had lower FEV_1/FVC ratios 60 ± 21 vs 81 ± 13 %, ($p < 0.05$), but no other differences in baseline PFTs and blood gases or intraoperative management including respiratory rate or ventilatory pressures.

Moderate hypercarbic patients were significantly older 69 ± 9 vs 56 ± 17 yrs, ($p < 0.05$) and had a longer history of smoking 43 ± 30 vs 16 ± 21 pack-years, ($p < 0.05$).

One moderately hypercarbic patient developed rapid atrial fibrillation and 2mm of ST segment depression in the PACU which responded to intravenous diltiazem treatment.

No other clinically significant complications arose secondary to the use of CO_2 insufflation. Postoperatively two patients required the insertion of a Heimlich valve for persistent air leak.

Two patients had a prolonged length of stay; one patient required discharge with home oxygen and another had subcutaneous emphysema requiring repeated chest tube insertions. In hospital length of stay (LOS) in these VATS patients was 5.7 ± 3.4 days and no difference in LOS was found between the two groups.

DISCUSSION

Our study documented that adult patients undergoing pulmonary resection with pre-existing pulmonary disease and the use of CO_2 insufflation develop significant changes in their arterial blood gases leading to acidosis and hypercarbia. More importantly a significant number of patients (57%) developed moderate hypercarbia defined as a $PaCO_2$ > 50 mm Hg.

This finding is a marked difference from Ohtsuka et al who found minimal respiratory changes in cardiac patients undergoing internal mammary artery harvesting (5). Ohtsuka et al however included patients with relatively normal preoperative pulmonary function. Byhahn et al did find an increase in $PaCO_2$ to a mean of 44.6 mm Hg after 2 hours of CO_2 insufflation despite adjusting their ventilation to maintain normal pH and $PaCO_2$ (32-45 mm Hg) (6).

This rise in carbon dioxide was still less than our finding and again is likely due to our study population having a significant presence of pulmonary impairment. Other studies of the effect of CO_2 insufflation concentrated on the hemodynamic effects and recorded few if any respiratory details (3, 4, 7, 8).

Previously reported studies in similar patients undergoing similar VATS procedures with OLV did not utilize CO_2 insufflation and also did not report similar changes in $ETCO_2$ or $PaCO_2$ (12, 13).

We did note demographic factors correlated with the development of moderate hypercarbia during OLV with CO_2 insufflation were the presence of lower FEV_1/FVC, increased age and increased smoking history. None of these factors are particularly surprising and are common in thoracic surgery patients. More interesting was our finding that peak airway pressures, minute ventilation, and the use of PEEP were not different between the two study groups suggesting that altering ventilation may not be that successful. One option may be the use of permissive hypercarbia as long as oxygenation is not compromised since no clinically relevant perioperative complications were noted. As the ventilatory parameters were not proscribed these results must be treated cautiously.

We also found that the arterial-$ETCO_2$ gradient was substantially widened from normal to 7.4 mm Hg in the mild hypercarbic group and to 16 mm Hg in the moderate hypercarbic group after 50 minutes of capnothorax.

This is similar but larger than the difference found by Byhahn et al of an arterial-$ETCO_2$ gradient of 4.7 mmHg at baseline which increased to 10 mm Hg after 120 minutes of CO_2 insufflation (6). As noted however, these patients did not have significant pulmonary disease.

This is also larger than that found in laparoscopic colorectal surgery patients reported by Tanaka et al who found an arterial to $ETCO_2$ difference that went from 5.8 at baseline to 7.1, 8.1 and 6.4 mm Hg after 10, 60 and 120 minutes of pneumoperitoneum (15). In light of our findings we note that using end-tidal CO_2 is not a reliable monitor of $PaCO_2$ and we recommend the use of frequent arterial blood gas sampling in high risk patients (low FEV1/FVC, increased age and smoking histories > 40 pack-years) undergoing VATS with CO_2 insufflation to avoid hypercarbia and acidosis.

Limitations of our study include the fact that it was a small observational study. It may have been due to this small sample size that we did not detect any clinically relevant complications.

We also did not control intraoperative ventilation nor did we blind the anaesthesiologists to the arterial blood gas results. In spite of this information being available however the majority of patients still developed significant hypercarbia indicating our

results underestimated the severity of the problem. Future study of the clinical impact of CO_2 insufflation in thoracosopic surgery patients should involve a larger sample size and control of ventilation parameters.

CONCLUSIONS

We found that adult patients with coexisting respiratory morbidity undergoing VATS with CO_2 insufflation develop hypercarbia and acidosis which is more likely in patients with advanced age, lower preoperative FEV1/FVC and greater smoking histories.

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