The Effects of Preperitoneal Carbon Dioxide Insufflation on Cardiopulmonary Function in Pigs

CPT Michael W. Blaney, MD, William C. Calton, MD, LTC James H. North Jr, MD

ABSTRACT

Background and Objectives: Although considerable experimental and clinical knowledge exists on the physiology of pneumoperitoneum, insufflation of the preperitoneal space has not been extensively studied. The purpose of this study is to evaluate the physiology associated with preperitoneal carbon dioxide (CO₂) insufflation in a porcine model.

Methods: Eleven pigs weighing 35 to 45 kg were anesthetized and placed on mechanical ventilation. A pulmonary artery catheter and an arterial line were inserted. Balloon dissection of the preperitoneal space and insufflation to 10 mm Hg for 1.5 hours, followed by an increase to 15 mm Hg for an additional 1.5 hours, was performed. Hemodynamic and arterial blood gas values were determined every 15 minutes throughout the stabilization and three-hour insufflation period. Hemodynamic parameters and blood gas values were analyzed using one-way analysis of variance with respect to insufflation time and pressure.

Results: Analysis of hemodynamics (CO, CVP, PAD, PAS, PCWP) did not demonstrate statistical significance with respect to time. However, there was a statistical difference in CO (p=0.01), CVP (p<0.01), and PCWP (p=0.034) when comparing a pressure of 15 mm Hg to a pressure of 10 or 0 mm Hg. The other parameters did not demonstrate significant differences among the three pressure groups. Arterial PCO₂ and pH were highly significant with respect to time (p<0.01 and P<0.01, respectively) and among the pressure groups (p<0.01 and P<0.01, respectively).

Conclusions: Insufflation of the preperitoneal space with CO₂ gas does not cause significant alterations in hemodynamics and blood gas changes at a pressure of 10 mm Hg. However, when a pressure of 15 mm Hg is used to insufflate this space, there is evidence of decreased pH and cardiac output, with elevated CVP and CO₂ retention. This correlates with greater pneumodissection of the gas within the layers of the abdominal wall when elevated pressures are used.

Key Words: Pneumoperitoneum, Preperitoneal, Laparoscopic, Physiology.

INTRODUCTION

With the advent of minimally invasive surgery, there has been a growth of surgical procedures requiring the insufflation of a distending gas for surgical exposure. The physiologic effects of insufflating the intra-abdominal cavity (pneumoperitoneum) have been well described in the literature. It is recognized that although laparoscopic surgery is associated with a low morbidity, there are significant cardiopulmonary and acid-base alterations that must be considered.

As more experience was gained, new procedures were developed, some of which included the use of a distending gas outside the confines of the peritoneal cavity. Such is the case in laparoscopic herniorrhaphy in which the preperitoneal space is insufflated with carbon dioxide. Additionally, other procedures, such as laparoscopic anti-reflux and colon surgery, that require a pneumoperitoneum involve violation of the peritoneal lining in order to perform the dissection. The opening of the peritoneum allows for the dissection of gas into the local tissue planes. Although this is helpful with the dissection, it also affords an opportunity for significant pneumodissection outside the operative field. The dissection of gas into the extraperitoneal space creates a more dynamic environment when compared to the relatively static space of the intra-abdominal cavity. This may become a significant problem as the complexity and length of laparoscopic surgical procedures increase.

The preperitoneal approach to laparoscopic hernia repair provides a model to evaluate the physiology of extraperitoneal CO₂ (carbon dioxide) insufflation. The purpose of this study is to answer the following questions: 1) To what extent is preperitoneal CO₂ absorbed? 2) Does dis-
tention of the preperitoneal space result in cardiovascular changes? and 3) To what extent is there dissection of gas within these extraperitoneal tissue planes? In order to evaluate these questions, a porcine model was developed for the insufflation of the preperitoneal space utilizing insufflation pressures commonly employed for laparoscopic hernia repair.

MATERIALS AND METHODS

After a ten-day acclimation period, 11 adult male pigs weighing between 36 and 45 kg were anesthetized with an IM injection of ketamine hydrochloride (ketaset, 20 mg/kg, Fort Dodge Laboratories, Fort Dodge, Iowa) and Xylazine (2mg/kg, Butler Co., Columbus, OH). The animal was then placed on mechanical ventilation (Ohio V5A Modulus Anesthesia Gas Machine) with an initial tidal volume of approximately 15 cc/kg and maintained under general anesthesia with 1-2% isoflurane (Floran, Anaquest, Madison, WI) while receiving a continuous infusion of lactated ringers at 80 cc/hr.

A common carotid arterial line was placed and monitored continuously via an ICU monitor (model HP66, Hewlett Packard, Waltham, MA). A pulmonary artery catheter was then floated into the pulmonary artery wedge position and monitored continuously. Initial arterial blood gas values were obtained prior to the start of the study period, and the animals minute ventilation adjusted to obtain levels of PCO$_2$ between 38 and 44 with pH between 7.40 and 7.45. Once the blood gas was normalized during this initial stabilization period, the minute ventilation was fixed for the remainder of the study. In addition to arterial blood gas values, hemodynamic parameters were also recorded during this initial stabilization period.

Balloon dissection of the preperitoneal space was accomplished through a small infraumbilical incision. The anterior fascia of the rectus abdominus was incised and the rectus split in the direction of its fibers for exposure of the posterior sheath. A 10 mm Origin™ balloon dissector was then passed along the posterior sheath into the preperitoneal space and insufflated with 15 pumps of the bulb inflator. A 10 mm Hasson trocar was placed and secured for insufflation of CO$_2$ gas, and a midline trans-abdominal port was placed in the subxyphoid position in order to monitor for rupture of the peritoneum.

Once the blood gas was normalized to the target range, the 180-minute study period began. The preperitoneal space was first insufflated with 10 mm Hg CO$_2$ gas for 90 minutes. Arterial blood gas values (pH, PCO$_2$, and PO$_2$) and hemodynamic parameters (CO - cardiac output, PCWP - pulmonary capillary wedge pressure, CVP - central venous pressure, and PAS/PAD - pulmonary artery systolic and diastolic pressures) were recorded every 15 minutes. End tidal CO$_2$ (EtCO$_2$) was also recorded using a Hewlett-Packard continuous in-line CO$_2$ monitor. Cardiac output was determined using the thermodilution technique with the mean of three injections per data point calculated by the hemodynamic module. CVP, PCWP, Pulmonary artery pressures, and arterial blood pressures were transduced directly. At the end of the initial 90 minutes at 10 mm Hg, the preperitoneal pressure was increased to 15 mm Hg for an additional 90 minutes.

During the insufflation period, animals were monitored for the development of gross pneumodissection outside the confines of the pelvis, which was arbitrarily defined as palpable subcutaneous emphysema above the umbilical port. At the end of the 180-minute study period, each animal was sacrificed using a lethal dose of Beuthanasia and necropsy of the abdominal wall performed in order to determine the extent of auto-dissection of gas within the tissue planes of the abdominal wall. The data was analyzed using analysis of variance. Statistical signficance was considered to be p value less than .05; error bars were calculated using standard error of the mean.

RESULTS

The arterial blood gas and hemodynamic data collected during the stabilization period and 180-minute study period was analyzed with respect to 1) the insufflation time (180 minutes) and 2) the insufflation pressure (0, 10, 15 mm Hg). While the data was collected, the peritoneum was monitored for rupture via the transabdominal subxyphoid port. Two animals demonstrated peritoneal rupture and subsequent pneumoperitoneum shortly after increasing the pressure to 15 mm Hg. The data from these animals at 15 mm Hg was excluded from the analysis.

A. Data Analysis with Respect to Time

The mean of each hemodynamic parameter was calculated at each 15-minute data point for all the animals. Table 1 represents the mean of each parameter at the
beginning of the study, at the end of the first insufflation period with 10 mm Hg (after 90 minutes of insufflation), and, finally, the mean at the end of the study period after insufflation with 15 mm Hg pressure. During the 180-minute study period, there was no statistically significant change in hemodynamic parameters when analyzed with respect to time (Table 1).

Analysis of arterial blood gas values with respect to time demonstrated statistically significant rises of PCO$_2$ and decreases in pH (Table 2). There was an increase in the slope of the PCO$_2$ curve and a corresponding decrease in the pH after increasing the pressure to 15 mm Hg (Figure 1, 2).

B. Data Analysis with Respect to Pressure

The mean value for each measured parameter at a pressure of zero (prior to insufflation) and at each pressure of 10 and 15 mm Hg was analyzed with respect to one another. The average CO, CVP, and PCWP at a pressure of 15 mm Hg was statistically changed when compared to pressures of 10 and 0. Other hemodynamic parameters did not differ significantly (Table 3).

Similarly, we noticed that at a pressure of 10 mm Hg, there was no statistically significant difference in PCO$_2$ and pH when compared to blood gas values at a pressure of zero. However, when the pressure was elevated to 15 mm Hg, there was significant increase in the absorption of carbon dioxide with a corresponding acidemia (Table 4).

C. Data Analysis of Animals with Clinical Evidence of Pneumodissection

Necropsy demonstrated that all animals had pneumodissection beyond the confines of the pelvis into the layers of the abdominal wall. However, six animals were noticed to have gross clinical evidence of pneumodissection outside the surgically dissected field during the study period. In all instances, this did not occur until increasing the insufflation pressure to 15 mm Hg and was manifested by palpable subcutaneous emphysema of the entire anterior abdominal wall. The animals with clinical evidence of pneumodissection demonstrated greater degrees of blood gas changes as evidenced by the pH and PCO$_2$ (Table 5).

DISCUSSION

Significant pneumodissection in laparoscopic procedures is not uncommonly observed, particularly when the CO$_2$ is exposed to extraperitoneal tissue planes. Clinically this may present with extensive subcutaneous emphysema, pneumomediastinum and pneumothorax. These sequelae are usually self-limiting once insufflation cease-
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**Table 1.**
Hemodynamic Analysis with Respect to Time.

| Hemodynamic parameter (mean value) | Stabilization period | 90 minutes (end of pressure at 10 mm Hg) | 180 minutes (end of study period and pressure at 15 mm Hg) | P value |
|-----------------------------------|----------------------|----------------------------------------|--------------------------------------------------------|--------|
| CO                                | 4.02 +/- .13         | 3.89 +/- .30                           | 3.56 +/- .35                                           | .64    |
| CVP                               | 5.65 +/- .54         | 5.38 +/- .86                           | 8.00 +/- 2.17                                          | .47    |
| PAD                               | 12.96 +/- .72        | 12.22 +/- 1.06                         | 13.50 +/- 1.02                                         | .97    |
| PAS                               | 22.52 +/- .72        | 21.78 +/- .78                          | 23.50 +/- 1.48                                         | .99    |
| PCWP                              | 6.96 +/- .75         | 7.11 +/- 1.21                          | 8.85 +/- 1.33                                          | .86    |

**Table 2.**
Arterial Blood Gas Analysis with Respect to Time.

| Blood Gas Value | Stabilization period | 90 minutes (end of pressure 10 mm Hg) | 180 minutes (end of study period and pressure 15 mm Hg) | P value |
|-----------------|----------------------|----------------------------------------|--------------------------------------------------------|--------|
| PCO$_2$         | 42.68 +/- .77        | 43.56 +/- 1.68                         | 54.17 +/- 2.93                                         | <.01   |
| pH              | 7.43 +/- .01         | 7.43 +/- .01                           | 7.37 +/- .02                                           | <.01   |
| EtCO2           | 34.56 +/- .67        | 36.56 +/- 1.75                         | 43.17 +/- 3.20                                         | .065   |

es; however, during insufflation, rapid rises in end-tidal CO$_2$ can be observed in this setting.

Carbon dioxide has traditionally been the insufflation gas of choice, although the use of other gases has been proposed. Results from several studies indicate that hypercarbia, with its associated decrease in blood pH, is primarily due to transperitoneal absorption of the carbon dioxide. Seed et al observed that anesthetized patients undergoing laparoscopic surgery developed increases in EtCO$_2$. Ho et al, using a porcine model, were able to directly measure increased carbon dioxide production, with secondary acidemia, after insufflation of CO$_2$ gas during laparoscopic cholecystectomy. They found that transperitoneal absorption of CO$_2$, not increased dead-space and impaired ventilatory function, was responsible for the development of hypercapnia and acidemia.

The current study showed that at 10 mm Hg pressure in the preperitoneal space, there was no significant absorption of carbon dioxide. However, after increasing the pressure to 15 mm Hg, there is a statistically significant increase in PCO$_2$ and decrease in pH with much greater degrees of change observed in the animals with clinical evidence of pneumodissection. It is possible that the development of hypercarbia and acidemia that occurred toward to end of the study period may still have developed if the pressure was left at 10 mm Hg for the entire 180 minutes. This is difficult to answer unless the animals were randomized to two separate pressure groups (one group at 10 mm Hg and a second at 15 mm Hg). However, when the pressure was increased to 15 mm Hg, there was an almost immediate trend in CO$_2$ retention with a corresponding acidemia, and in six of the eleven animals, a gross evidence of pneumodissection developed that was absent when the pressure was at 10 mm Hg. It is our contention that the process of pneumodissection markedly increases the effective surface area for gas diffusion. Anecdotally, we have observed similar phenomena in humans with significant hypercar-

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Table 3.
Hemodynamic Analysis with Respect to Pressure.

| Hemodynamic Parameter | Pressure = 0 | Pressure = 10 | Pressure = 15 | P value |
|-----------------------|-------------|--------------|---------------|---------|
| CO                    | 4.04 +/- .11| 3.96 +/- .09 | 3.62 +/- .10  | .011    |
| CVP                   | 5.37 +/- .41| 5.61 +/- .35 | 7.44 +/- .52  | <.01    |
| PAD                   | 12.64 +/- .58| 12.47 +/- .43| 13.29 +/- .41 | .43     |
| PAS                   | 22.08 +/- .59| 22.05 +/- .42| 22.57 +/- .51 | .71     |
| PCWP                  | 6.75 +/- .63| 7.06 +/- .36 | 8.56 +/- .56  | .034    |

Table 4.
Arterial Blood Gas Analysis with Respect to Pressure.

| Blood Gas Value | Pressure = 0 | Pressure = 10 | Pressure = 15 | P value |
|-----------------|--------------|--------------|---------------|---------|
| PCO\textsubscript{2} | 42.41 +/- .60| 43.32 +/- .60| 48.90 +/- .96 | <.01    |
| pH              | 7.44 +/- .01 | 7.44 +/- .00 | 7.40 +/- .01  | <.01    |
| EtCO\textsubscript{2} | 34.55 +/- .56| 35.90 +/- .54| 39.21 +/- .99 | <.01    |

Table 5.
pH and PCO\textsubscript{2} in Animals With and Without Clinical Evidence of Pneumodissection.

| Clinical Evidence of Pneumodissection | Mean PCO\textsubscript{2} at end of 10 mm Hg (90 minutes) | Mean PCO\textsubscript{2} at end of 15 mm Hg (180 minutes) | Mean pH at end of 10 mm Hg (90 minutes) | Mean pH at end of 15 mm Hg (180 minutes) |
|--------------------------------------|----------------------------------------------------------|----------------------------------------------------------|----------------------------------------|----------------------------------------|
| Yes                                  | 44.45 +/- 2.1                                           | 50.94 +/- 2.0                                           | 7.42 +/- .016                           | 7.37 +/- .018                           |
| No                                   | 41.56 +/- 1.1                                           | 44.62 +/- .52                                           | 7.45 +/- .012                           | 7.42 +/- .006                           |
| P value                              | .25                                                      | .028                                                    | .21                                    | .03                                    |

Reports of acute cardiovascular compromise during abdominal insufflation is well documented. During insufflation of the abdomen for laparoscopy, it is believed that at lower pressure levels there is augmentation of venous return with a corresponding increase in CVP and right heart filling. This results in an associated increase in cardiac performance as would be predicted.
The mechanical effect of increased abdominal pressure, however, higher levels of insufflation cause significant IVC compression that results in compromised venous return and, hence, a decrease in cardiac performance.\textsuperscript{10,11} Kelman et al. demonstrated that progressive increases in abdominal pressure to 20 cm H\textsubscript{2}O was accompanied by increases of CVP and cardiac output. Greater increases to around 40 cm H\textsubscript{2}O resulted in measurable decreases in CVP and cardiac output.\textsuperscript{10} Kashtan confirmed these findings in a canine model with the additional caveat that the magnitude and direction of change not only depends on the level of abdominal pressure but also on the intravascular volume status.\textsuperscript{11}

The mechanical effect of increased abdominal pressure, however, alone may not explain the complex physiologic changes that occur during laparoscopic surgery. In addition to the direct mechanics that insufflation has on the cardiovascular system, there are also changes in acid-base homeostasis and reflex sympathetic activity that must also be considered. In a porcine model by Ho et al., they found that at standard insufflation pressures of 15 mm Hg, the CVP and cardiac output remained unchanged. However, there was a measurable depression in the stroke volume with a compensatory increase in heart rate, thereby maintaining CO. They go on to state that the reduction in stroke volume may be the result of a cardiac depressant effect that CO\textsubscript{2} has on myocardium and that this depressant effect may be partially masked by the sympathetic stimulation on the heart rate from high PCO\textsubscript{2} levels and the stress of abdominal insufflation.\textsuperscript{12} In addition, the cardio-depressant effects of anesthesia are also confounding factors that can alter intraoperative cardiovascular physiology. For these reasons, it is impossible to explain a single mechanism for the cardiovascular changes noted during laparoscopic surgery.

This study demonstrated no significant changes in hemodynamic parameters over the entire 180-minute study period when analyzed with respect to time. But when these values were compared with respect to the different insufflation pressures, there was an increase in CVP and PCWP, with a decrease in CO after increasing the insufflation pressure to 15 mm Hg. The elevated CVP and PCWP that was seen may possibly be explained by augmented venous return secondary to increased pressure and pneumodissection of gas that occurred at 15 mm Hg. Although, one would normally expect augmented cardiac performance with elevated venous return as predicted by the Starling Curve, it is possible that this increase was negated by the cardiac depressant of the anesthetic and hypercarbia, resulting in the decrease in CO that was observed.

Although laparoscopic surgery with insufflation of CO\textsubscript{2} gas is safe and generally associated with minor physiologic consequences, there is the potential for significant CO\textsubscript{2} retention to develop in certain situations. In the case of extraperitoneal laparoscopic surgery, there is a potential plane for gas to dissect, thereby increasing the effective absorptive surface area for gas diffusion with a less predictable physiology than the fixed surface area of the intra-abdominal cavity. At the commonly used pressure of 10 mm Hg for preperitoneal hernia repairs, this pneumodissection is minimal. However, the current study suggests that when higher pressures are used, there is a threshold above which significant pneumodissection can occur, resulting in dynamic changes in CO\textsubscript{2} absorption.

**CONCLUSIONS**

Insufflation of the preperitoneal space with CO\textsubscript{2} gas does not cause significant alterations in hemodynamics and blood gas changes at a pressure of 10 mm Hg. However, when a pressure of 15 mm Hg is used to insufflate this space, there is evidence of decreased pH and cardiac output, with elevated CVP and CO\textsubscript{2} retention. This correlates with greater pneumodissection of the gas within the layers of the abdominal wall when elevated pressures are used.

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