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Optimal positive end-expiratory pressure (PEEP) settings in differential lung ventilation during simultaneous unilateral pneumothorax and laparoscopy

An experimental study in pigs

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Abstract
Background: A combined thoraco-laparoscopic technique for esophageal resection is technically possible, but it requires special attention to ventilation. The positive insufflation pressure normally used in laparoscopy will, when communication between thorax and abdomen is established, create a pneumothorax.
Methods: We performed an experimental study of differential lung ventilation with different levels of positive end-expiratory pressure (PEEP) settings during thoraco-laparoscopy in anesthetized pigs.
Results: Positive pressure insufflation of carbon dioxide (CO_2) resulted in elevated pulmonary capillary wedge pressure, hypercarbia, and respiratory acidosis. Hypoxemia, however, developed only at lower settings of PEEP. Heart rate, mean arterial pressure, and cardiac output remained relatively stable.
Conclusion: Pneumopleuroperitoneum under positive CO_2 insufflation pressure had adverse effects on blood gases. Hypercarbia, respiratory acidosis, and hypoxemia were early manifestations that occurred even in the presence of hemodynamic stability. The application of PEEP equal to or above CO_2 insufflation pressure improved blood gases; in particular, the hypoxia could be avoided. No beneficial effects of differential lung ventilation were documented.

Key words: Laparoscopy — Pneumothorax — Positive end-expiratory pressure (PEEP) — Cardiopulmonary effects

Following its description in 1992 [5, 6] several investigators reported their experience with esophagectomy using thoracoscopy-assisted techniques combined with laparotomy for gastric mobilization. Some clinics have added this technique to their standard methods [12], but the results of endoscopic esophagectomy are not yet generally regarded as convincing [7]. Refinements in technical equipment—i.e., the introduction of a hand port system—has made dissection and gastric mobilization easier to perform laparoscopically [15]. A combined thoracoscopic and laparoscopic technique therefore would seem to be a logical development. However, once communication to the pleural space is established, the insufflation of carbon dioxide (CO_2) under positive pressure (necessary for laparoscopy) may have adverse effects on cardiopulmonary function and a tension pneumothorax may develop.

One experimental study on the effects of positive pressure insufflation during thoracoscopy documented hemodynamic compromise with a decrease in cardiac index, mean arterial pressure (MAP), and stroke volume [10]. Reduced arterial saturation (SaO_2) and oxygen tension (PaO_2) and increased carbon dioxide tension ($PaCO_2$) have been reported during CO_2 insufflation and pneumothorax in laparoscopic surgery in pigs [16]. In the same study, the adverse effects on gas exchange could be corrected, at least temporarily, by applying positive end-expiratory pressure (PEEP) during ventilation. The therapeutic benefits of PEEP have been reported both in other experimental studies [4] and when it is applied in a clinical setting during laparoscopy, with pneumothorax as a complication [11].

However, no one has yet established which combination of insufflation pressure and PEEP is least harmful for the circulation and gas exchange. Theoretically, PEEP can improve the ventilation of collapsed and poorly ventilated alveoli, decrease intrapulmonary shunt volumes, and finally increase systemic oxygen saturation [18]. On the other hand, compliance can be reduced during PEEP as a result of overdistention of the alveoli, alveolar rupture, and surfactant inactivation [18]. A PEEP-induced increase in lung

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volume may cause compression of the small intraalveolar vessels and thereby increase pulmonary resistance [2, 9] and decrease cardiac output (CO). During laparoscopy and communicating unilateral pneumothorax, the ipsilateral lung will be partly compressed due to the positive insufflation pressure.

We assumed that the positive intrathoracic pressure had to be counteracted by application of an intrapulmonary pressure of equal or higher degree. PEEP applied in this lung had to be equal to, or exceed, the CO_2 insufflation pressure. We hypothesized that the optimal respiratory technique would be differential lung ventilation, and, to avoid harmful effects that might be related to prolonged and elevated PEEP, we decreased PEEP on the lung with no pneumothorax. For this purpose, we designed an experimental model applying differential ventilation with varying combinations of PEEP.

Materials and methods

Animal preparation and instrumentation

The experimental protocol was reviewed and approved by the Institutional Review Board of Uppsala University, Sweden, in conformity with the Helsinki convention for the use and care of animals. Eleven Swedish Landrace piglets, both male and female, 11–14 weeks of age, with a mean weight of 25 ± 1 kg (range, 22–28) were studied. The animals were fasted overnight with free access to water and delivered by the same supplier directly to the laboratory on the morning of the experiment.

Anesthesia was induced with an intramuscular injection of 3 mg/kg tiletamin plus 3 mg/kg zolazepam (Zoletil; Virback Labs., Carros, France), 2 mg/kg xylazine (Rompun; Bayer, Levercausen, Germany) and atropine 0.04 mg/kg combined with 1 mg/kg morphine intravenously as a bolus injection. Anesthesia was maintained with a continuous infusion of 20 mg/kg/h ketaminol (Ketaminol; Veterinary AB, Zurich, Switzerland), 0.24 mg/kg/h pancuronium (Pavulon; N.V. Organon Oss, Boxtel, The Netherlands), and 0.48 mg/kg/h morphine [3]. The animals were placed in supine position before instrumentation. After the induction of anesthesia, 6 mg pancuronium was administered for muscle relaxation.

Tracheotomy was performed and an 8-mm outer diameter endotracheal tube (Mallinckrodt, Glen Falls, NY, USA) introduced. Mechanical ventilation was initiated. To compensate for fluid losses throughout the study, an intravenous bolus of 50–200 ml dextran (Macrodex 6%; Fresenius Kabi AB, Upsala, Sweden) was administered before the interventions started, aiming at a pulmonary capillary wedge pressure (PCWP) of 12 mmHg. This was followed by a continuous intravenous infusion of a crystalloid solution with 2.5% glucose 10 ml/kg/h (Rehydrex; Pharmacia, Stockholm, Sweden).

Technique for separate lung intubation and differential lung ventilation

Each lung was intubated separately. In the pig, the bronchial tree differs from human anatomy [17]. Pigs have a separate bronchial branch to the right upper lobe, and this branch goes directly from the trachea, proximal to the carina. The right lung is also larger than the left and therefore needs a larger tidal volume.

First, the larger 8-mm endotracheal tube was positioned with its tip immediately above the bronchial branch to the right upper lobe. To achieve separate intubation and differential ventilation, the left bronchus was intubated through the wide-bore tube with a 5-mm (outer diameter) endotracheal tube. This tube was advanced over a 3.5-mm bronchoscope to ensure optimal placement. After the cuffs were inflated, both tubes were connected to separate ventilators (Servo Ventilator 900C; Siemens-Elema, Stockholm, Sweden) on volume-controlled ventilation with an I:E ratio of 1:1, F_iO_2 of 0.4, respiratory rate of 25 per min, and PEEP of 5 cm H_2O. Correct position of the tubes was confirmed when volumes of inspiration and expiration were equal. A respiratory profile monitor (CO_2SMO+; Novametrix Medical Systems, Inc, Wallingford, CT, USA) was connected to each ventilator for differential monitoring of end-tidal carbon dioxide levels ($ETCO_2$), static compliance (C stat), and CO_2 elimination (VCO_2). During the initial stabilization period, the tidal volume for each lung was adjusted to maintain $PaCO_2$ between 5.0 and 5.5 kPa and, if possible, to keep $ETCO_2$ levels within a limit of 10% difference between the two lungs.

Catherization and monitoring

Intravascular catheters were placed surgically. A catheter (16-G) was inserted via a branch of the right external carotid artery into the aortic arch for recording of MAP and sampling for arterial blood gas analysis. A pulmonary artery catheter (7-Fr) was introduced via the right external jugular vein into the pulmonary artery for measurements of CO and PCWP and sampling for gas analysis (saturation and partial pressure of oxygen and carbon dioxide in mixed venous blood: SvO_2, PvO_2, and $PvCO_2$). Another venous catheter (20-G) was positioned in the right atrium and used for injection of indicator fluid for measurements of cardiac output by thermodilution. Two trocars (5-mm) were inserted to accommodate gas insufflation, one intraperitoneally and the other into the right pleural space. The trocars were connected to a common gas insufflator (Pelvi-Pneu CO_2; WISAP, Saurlach, Germany); the CO_2 insufflation pressure, when applied, was always 10 mmHg. The pressure was monitored with an aneroid manometer. ECG and intravascular pressures were monitored continuously (Solar 8000; Marquette Electronics Inc., West Yorkshire, UK). Blood samples were analyzed in a blood gas analyzer (ABL5; Radiometer, Copenhagen, Denmark) and in a hemoxymeter (OSM3; Radiometer).

Experimental design

After completion of the preparations, a nonintervention period of $\geq$ 30 minutes was undertaken. During this time, final adjustments of the ventilators were performed and the animals were observed for hemodynamic and respiratory stability. After baseline values were achieved, minute ventilation and tidal volumes were held constant for the remainder of the experimental cycle. During the nonintervention periods, PEEP was 5 cm H_2O on both ventilators. Initial baseline values were recorded for each pig and used as a comparison and standard for stabilization after each single test interval.

An experimental cycle of 20 mins was initiated by insufflating CO_2 with a pressure of 10 mmHg applied to both trocars; thereby, a right pneumothorax and a pneumoperitoneum under equal positive pressure were created. During this period, right and left PEEP settings of 10 (right)\5 (left), 10\10, 15\5, and 15\10 cm H_2O were used by random assignment. The same PEEP setting was applied for the next 20 mins with no adjustments of the ventilators. At the end of each cycle, various parameters were measured.

The CO_2 gas insufflation was then turned off. The pneumothorax and the pneumoperitoneum were evacuated, and a period of hemodynamic and respiratory stabilisation was allowed. Stable conditions were defined as $PaCO_2$ ± 10% of baseline measurement, PCWP 10–15 mmHg, and a constant CO_2 elimination. If the animal did not recover within 10 min, adjustments of tidal volumes were done to normalize arterial blood gases. Intravenous volume supplementation with crystalloid solution was given if PCWP was < 10 mmHg. Thereafter, a new experimental cycle was started with another combination of PEEP. A cycle, including a normal stabilization period, took ~60 mins.

In a pilot study with a PEEP setting of 5\5 cm H_2O, extremely high airway pressures, exceeding 90 cm H_2O, were recorded, and there was a severe deterioration of blood gases that was hard to overcome in spite of long recovery periods. Therefore, the PEEP combination of 5\5 was always applied at the end of the experiment. During the study, it became clear that higher PEEP on both lungs was favorable, and for the last four animals an additional cycle with PEEP setting of 15\15 was inserted prior to 5\5.

After the completion of the experiment, the animals were killed via potassium chloride injection.

Statistical analysis

For the four procedures in which the order was randomized, a Randomised Block Design two-way analysis of variance (ANOVA) was performed for

Table 1. *p* values for results that were statistically significant at an overall level of 0.05 Where values are missing, the corresponding statistical tests were not significant.

Outcome variable	Mean outcomes of randomized procedures (PEEP 10/5, 10/10, 15/5, 15/10)	Comparison to PEEP 5/5	Comparison to baseline
HR		.0012	.0001
MAP			
PCWP			.0001
CO		.0002	.0001
SvO_2	.0001	.0001	.0001
PvO_2	.0002	.0001	.0001
$PvCO_2$	.0002	.0001	.0001
pH	.0032	.0001	.0001
BE			.0001
PaO_2	.0001	.0001	.0001
$PaCO_2$		.0001	.0001
SaO_2	.0001	.0001	.0004
$ETCO_2$ sin	.0001	.0001	
VCO_2 sin	.0001		
C stat sin	.0093	.0084	.0001
$ETCO_2$ dx			
VCO_2 dx	.0026		.0001
C stat dx	.0001	.0001	.0001

$ETCO_2$, VCO_2, and C stat were measured separately for right and left lung, as indicated by “dx” or “sin”

each outcome variable to determine if the mean outcomes of the four treatments (combinations of PEEP) differed significantly from one another. A residual analysis was performed for each ANOVA to confirm that the underlying model assumptions were met by the data. A Bonferroni correction was used to ensure that the overall type I error rate was ≤ 0.05. In addition, comparisons were made between the mean outcome averaged over the four randomized procedures, the mean outcome at baseline, and the mean outcome for procedure 5\5. A *p* values of < 0.05 was considered to be significant.

For treatment 15\15, which was applied only for the four last animals, a purely descriptive comparison of the average outcomes was applied.

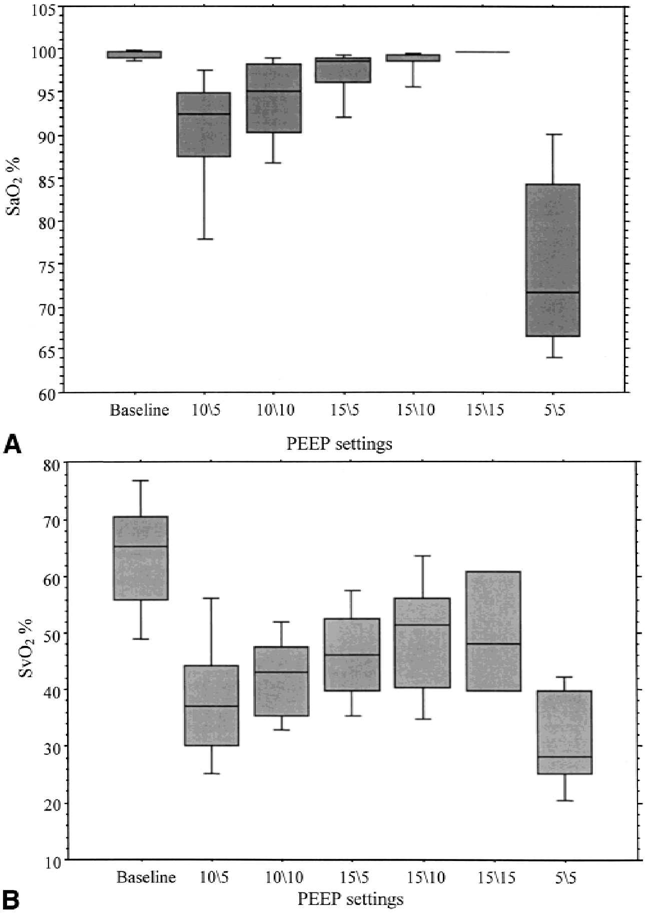

Fig. 1. **A** Arterial and **B** mixed venous oxygen saturation at baseline and at different combinations of PEEP in the right and left lungs (dz§in).

Results

Blood gases deteriorated when intrapleural pressure exceeded PEEP on either lung (Table 1 and Figs. 1 and 2). With low settings of PEEP, hypoxia became apparent. At the completion of procedure 5\5, SaO_2 had dropped from 99% to 75%. The changes were also reflected in mixed venous blood, with SvO_2 as low as 31%. The differences among the randomized treatments were significant ($p < 0.0001$); lower PEEP resulted in hypoxemia.

$PaCO_2$ increased and pH decreased when CO_2 insufflation was applied. Among the randomized treatments, however, $PaCO_2$ was not statistically different. The changes in $PaCO_2$ and pH were most pronounced at 5\5 ($p < 0.0001$), where all the animals developed severe respiratory acidosis. When 15\15 was applied, values close to normal were obtained. No drift in baseline values for blood gases or pH were observed between initial and final recordings.

Changes in hemodynamic variables were less predictable. Cardiac output decreased when CO_2 insufflation under positive pressure was applied, but there were no differences among the randomized treatments (Table 1 and Fig. 3). Mean arterial pressure did not show any significant variations when comparisons were made among the randomized treatments, the 5\5 treatment, and baseline (Fig. 3). A decrease in heart rate (HR), MAP, and CO was noted for all pigs between initial baseline measurements and the first recovery or nonintervention period, regardless of which PEEP setting we tested first. Thereafter, when compared during the nonintervention periods, HR, MAP, and CO became more stable from cycle to cycle. Concomitant to the positive insufflation pressure of 10 mmHg, PCWP increased and stayed elevated at a constant level as long as gas insufflation continued. The elevation was 5–8 mmHg and it was independent of PEEP setting.

CO_2 elimination increased when CO_2 insufflation was applied, but the total CO_2 elimination did not differ significantly among the randomized treatments or 5\5. The right lung always eliminated more CO_2, and the difference in $ETCO_2$ between right and left lung increased with a larger difference in PEEP setting. The difference in ET CO_2 values was most evident with PEEP 15\15 (Fig. 4).

The static compliance of the lung was reduced if PEEP was less than the intrapleural pressure. This was true no matter which lung was studied. Compliance decreased with decreasing PEEP. During the 15\15 treatment, compliance was comparable to baseline values (Fig. 4). If there was a difference in PEEP between the lungs, there was a tendency to simultaneous decrease in C stat, $ETCO_2$, and VCO_2 in the lung with lower PEEP, even in the presence of hypercarbia as measured by blood gas analysis (Fig. 4).

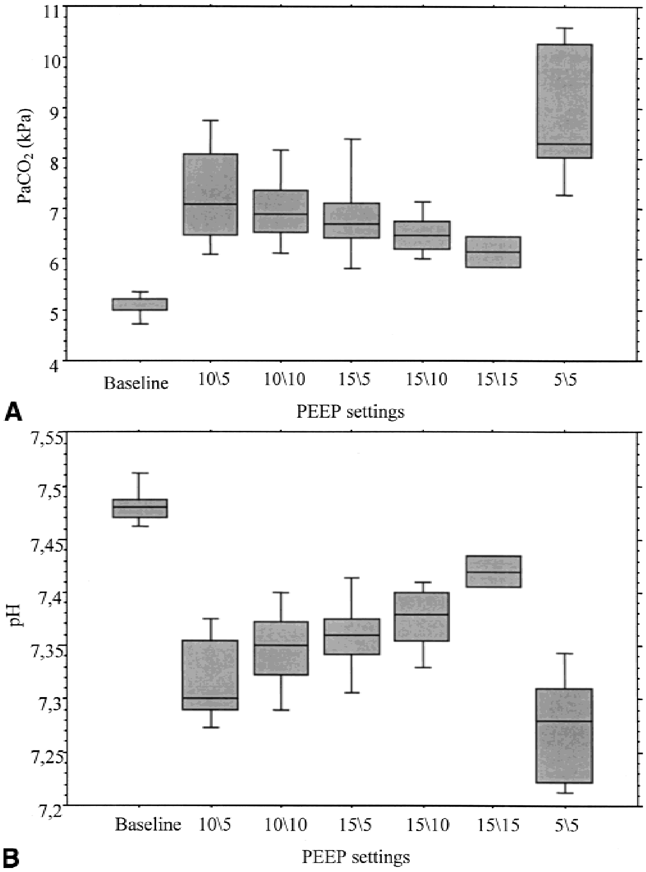

Fig. 2. **A** PaCO$_2$ (kPa) and **B** pH at baseline and at different combinations of PEEP in the right and left lungs (dz§in).

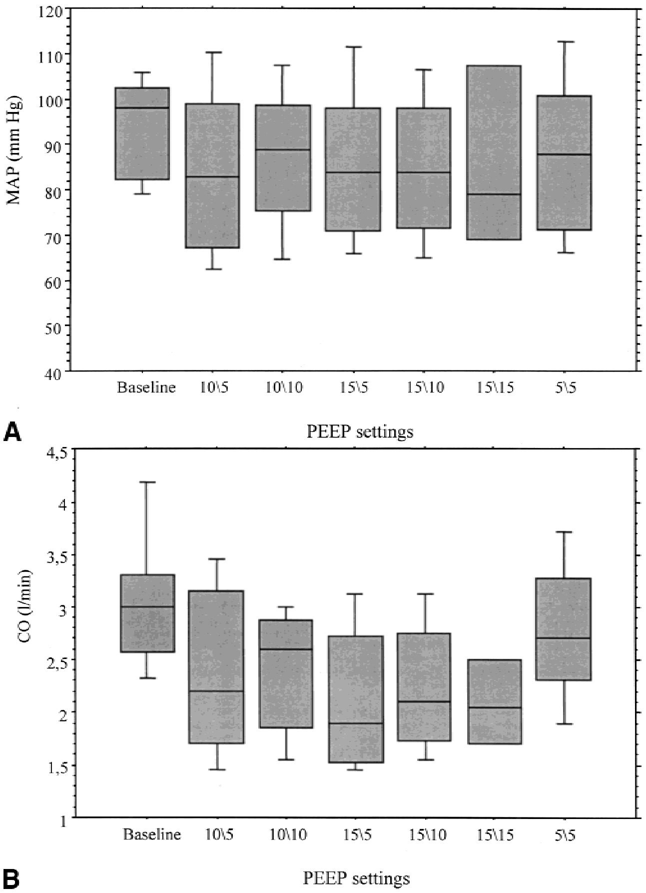

Fig. 3. **A** Mean arterial pressure (MAP) (mmHg) and **B** cardiac output (CO) (L/min) at baseline and at different combinations of PEEP in the right and left lungs (dz§in).

Discussion

In our study, when a communicating pneumopleuroperitoneum was established with CO$_2$ insufflation pressure of 10 mmHg, the following major observations were noted: PCWP increased with 5–8 mmHg followed by hypercarbia, respiratory acidosis, increased CO$_2$ elimination, and finally hypoxemia. The hypoxemia could be reversed and the acidosis considerably reduced if adequate PEEP was used on both lungs. Even if PEEP was 15 cm H$_2$O on the side with pneumothorax (right), deterioration of the blood gases was observed when PEEP on the other lung was 5 or 10 can H$_2$O. The blood gases were close to normal when PEEP in both lungs exceeded the CO$_2$ insufflation pressure. Among the different PEEP values we tested, the combination of 15\15 seemed to be the most optimal, but this combination was applied only in the last four animals. Hemodynamic outcomes were remarkably stable.

It is well known that CO$_2$ is absorbed transperitoneally during laparoscopy [13, 14]. The degree of hypercarbia and acidemia induced depends on CO$_2$ insufflation pressure, peritoneal area available for gas exchange, and respiratory compensation [18]. At an insufflation pressure of 10 mmHg, the changes are usually moderate and are clinically well tolerated both in humans [18] and in pigs [8]. With a communicating pneumopleuroperitoneum, a larger surface area will be exposed. More CO$_2$ may be absorbed and it will thus increase hypercarbia and acidosis. With positive pressure in the pleural cavity, as in our model, a tension pneumothorax may further aggravate the condition because of the collapse of lung parenchyma and impaired gas exchange. Shunting of blood in collapsed lung parenchyma may result in hypoxemia and CO$_2$ retention [1, 16]. Carbon dioxide is more soluble and crosses from the blood into the alveoli 25 times faster than oxygen [18]. Impaired gas exchange is therefore first noted in reduced oxygenation rather than increasing hypercarbia, even in the presence of an abnormal amount of systemically dissolved carbon dioxide. We observed a significant difference in PaO$_2$ among the four randomized treatments but not in PaCO$_2$.

The pathophysiology of tension pneumothorax is not fully established. It is unclear whether cardiovascular collapse is secondary to a direct compressive effect on central venous structures with reduced preload to the heart, or secondary to grave hypoxemia caused by lung parenchymal collapse with shunting of desaturated blood [1, 18]. In our study, serious hypoxemia, hypercarbia, and respiratory acidosis developed without significant changes in HR and MAP. We concluded that hypoxemia is an early sign of impaired gas exchange in ventilated pigs with pneumothorax. This condition has been described previously in an experiment with progressive pneumothorax where SaO$_2$ decreased immediately and continued to decline to levels below 50% prior to cardiovascular collapse [1].

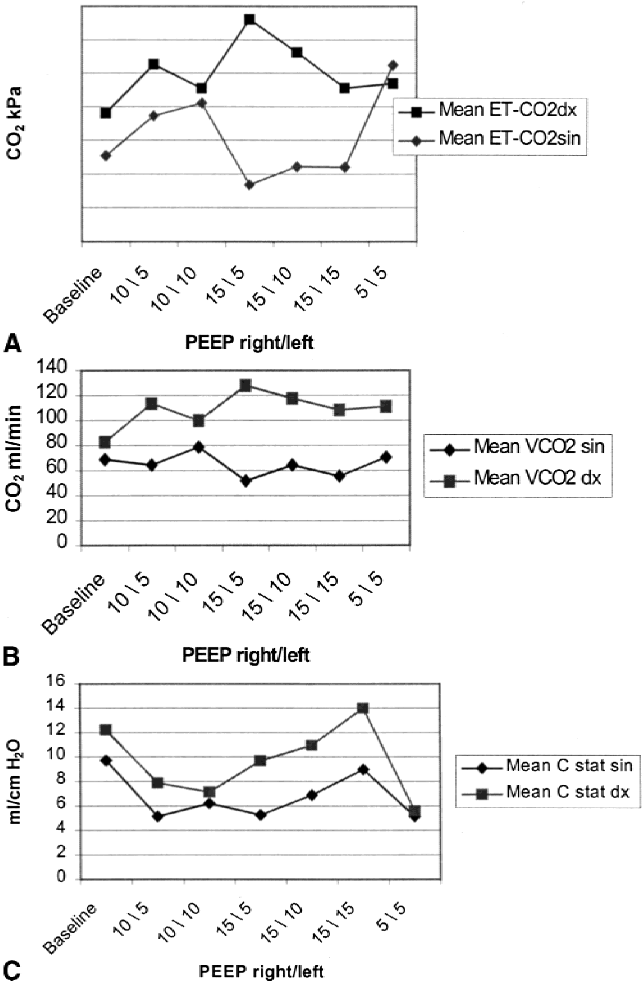

Fig. 4. **A** Variations in $ETCO_2$, **B** VCO_2, and **C** stat at baseline and at different combinations of PEEP in the right and left lungs (dz§in).

During laparoscopy, the positive insufflation pressure can have adverse effects on venous return and cardiac preload [18]. When the pressure is also extended into the thorax, as in our model, it may impair venous return even further. We noted an elevation in PCWP of 5–8 cm H_2O with the application of CO_2 insufflation. At the same time, CO decreased. These changes in PCWP and CO were independent of PEEP settings. One explanation is that positive insufflation pressure within the abdomen and thorax decreases venous return more than moderately elevated PEEP does.

We initially hypothesized that the optimal respiratory technique during laparoscopy with communicating unilateral pneumothorax would be differential lung ventilation with different PEEP on left and right lungs. On the side of the pneumothorax, we thought that PEEP had to equal or exceed the CO_2 insufflation pressure to avoid lung parenchymal collapse and poor oxygenation. To minimize harmful side effects, we tried to keep a lower PEEP on the contralateral lung.

Our hypothesis did not prove to be completely correct. To counterbalance the positive insufflation pressure, it seemed to be necessary to apply an equal or larger PEEP. However, this PEEP had to be applied to both lungs to avoid deterioration of blood gases. The 15/5 treatment was significantly worse than the 15/10 one, which in turn was worse than 15\15 when arterial blood gases were compared. In spite of severely elevated $PvCO_2$, we noted lower values of $ETCO_2$ and VCO_2 in the lung with the lower PEEP. Analysis of simultaneous changes in $ETCO_2$, VCO_2, and compliance demonstrated that these parameters decreased in the lung that had a PEEP that was exceeded by intrapleural pressure (Fig. 4). As in humans, the pig also has a mobile mediastinum, and mediastinal shift with compression of the contralateral lung and shunting of desaturated blood may, at least to some extent, explain this.

Thus, the use of differential ventilation with unequal PEEP applied to different lungs was not shown to yield any benefits. Equal PEEP on both lungs is easier to apply with one ventilator through the same endotracheal tube. In clinical application, during combined thoracoscopic and laparoscopic esophagectomy, a double-lumen endotracheal tube can be used. This also facilitates one-lung ventilation during thoracoscopy. During the thoracoscopic part of the operation, the right lung is not ventilated and is allowed to collapse. The thoracic trocars are open, with no valve mechanism. Gas insufflation is not used; the intrapleural pressure on the right side is therefore zero. During laparoscopy, ventilation can occur through a double-lumen tube or can be changed to a single-lumen tube.

Based on these observations, we recommend using CO_2 insufflation pressures within the normal range (10–12 mmHg) as long as no communication to the pleural space is established. However, when an opening to the mediastinum and the pleural space is created, the insufflation pressure has to be kept as low as possible, and PEEP should be applied to both lungs at a pressure equal to or exceeding insufflation pressure. With these maneuvers, the adverse effects of pneumothorax can be minimized.

We conclude that communicating pneumopleuroperitoneum under positive CO_2 insufflation pressure had adverse effects on arterial blood gases. Hypercarbia, respiratory acidosis, and hypoxemia were early manifestations and could be demonstrated even in the presence of hemodynamic stability. The application of PEEP improved blood gases so that hypoxemia, especially, could be avoided. No beneficial effects of the differential application of PEEP were documented. To achieve optimal ventilation, the PEEP applied to both lungs had to counterbalance the insufflation pressure.

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References

1. Barton ED, Rhee P, Hutton KC, Rosen P (1997) The pathophysiology of tension pneumothorax in ventilated swine. J Emerg Med 15: 147–153
2. Benumof JL (1985) One-lung ventilation and hypoxic pulmonary vasoconstriction: implications for the anaesthetic management. Anesth Analg 64: 821–833

3. Boschert K, Flecknell PA, Fosse RT, Framstad T, Ganter M, Sjöstrand U, Stevens J, Thurmon X (1996) Ketamine Consensus Working Group. Ketamine and its use in the pig. Lab Anim. 30: 209–219
4. Chiche JD, Joris J, Hamoir E, Lamy M (1995) PEEP corrects haemodynamic changes induced by pneumothorax during laparoscopy [Abstract]. Br J Anesth 74: A175
5. Cuschieri A (1994) Thoacoscopic subtotal oesophagectomy. Endosc Surg 2: 21–25
6. Gossot D, Ghnassia MD, Debiolles H (1992) Thoracoscopic dissection of the esophagus: an experimental study. Surg Endosc 6: 59–61
7. Gossot D, Toledo L, Cortes A (2000) Minimal access esophagectomy: where are we up to? Semin Laparosc Surg 7: 2–8
8. Ho HS, Gunther RA, Wolfe BM (1992) Intraperitoneal carbon dioxide insufflation and cardiopulmonary functions: laparoscopic cholecystectomy in pigs. Arch Surg 127: 928–933
9. Horswell JL (1993) Anesthetic techniques for thoracoscopy. Ann Thorac Surg 65: 624–629
10. Jones DR, Graeber GM, Tanguilig GG, Hobbs G, Murray GF (1993) Effects of insufflation on hemodynamics during thoracoscopy. Ann Thorac Surg 55: 1379–1382
11. Joris JL, Chiche J-D, Lamy ML (1995) Pneumothorax during laparoscopic fundoplication: diagnosis and treatment with positive end-expiratory pressure. Anesth Analg 81: 993–1000
12. Law S, Fok M, Chu KM, Wong J (1997) Thoracoscopic esophaectomy for esophageal cancer. Surgery 122: 8–14
13. Leighton T, Pianim N, Liu S-Y, Kono M, Klein S, Bongard F (1992) Effectors of hypercarbia during experimental pneumoperitoneum. Am Surg 58: 717–721
14. Lister DR, Rudston-Brown B, Warriner B, McEwen J, Chan M, Waaley KR (1994) Carbon dioxide absorption is not linearly related to intraperitoneal carbon dioxide insufflation pressure in pigs. Anesthesiology 80: 129–136
15. Litwin DEM, Darzi A, Jakimowicz J, Kelly JJ, Arvidsson D, Hansen P, Callery MP, Denis R, Fowler DL, Medich DS, O'Reilly MJ, Atlas H, Himpens JM, Swanstorm LL, Arous EJ, Pattyn P, Yodd SM, Ricciardi R, Sandor A, Meyers W (2000) the HALS study group Hand-assisted laparoscopic surgery (HALS) with the HandPort system: initial experience with 68 patients. Ann Surg 231: 715–723
16. Marcus DR, Lau WM, Swanstrom LL (1996) Carbon dioxide pneumothorax in laparoscopic surgery. Am J Surg 171: 464–466
17. Nickel R, Schummer A, Seiferle E (1979) Respiratory system. The viscera of the domestic mammals. 2nd rev. edition by Schummer A, Nickel R (eds) Verlag Paul Parey, Berlin, pp 211–281
18. Stegerwald S, Bockhorn H, Denhardt R (1998) Respiratory changes during carbon dioxide pneumoperitoneum. In: Rosenthal RJ, Friedman RL, Phillips EH (eds) The pathophysiology of pneumoperitoneum. Springer, Berlin, pp 70–77