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Ventilation–perfusion distributions and gas exchange during carbon dioxide-pneumoperitoneum in a porcine model

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Key points

- Pneumoperitoneum (PP) with carbon dioxide used for laparoscopic surgery results in conflicting ventilation effects.
- In a pig model, PP improved *V/Q* match with better oxygenation and gas exchange.
- This may be due to increased Pa_{CO_2} causing enhanced hypoxic pulmonary vasoconstriction.

Background. Carbon dioxide (CO_2)-pneumoperitoneum (PP) of 12 mm Hg increases arterial oxygenation, but it also promotes collapse of dependent lung regions. This seeming paradox prompted the present animal study on the effects of PP on ventilation–perfusion distribution (*V/Q*) and gas exchange.

Methods. Fourteen anaesthetized pigs were studied. In seven pigs, single photon emission computed tomography (SPECT) was used for spatial analysis of ventilation and perfusion distributions, and in another seven pigs, multiple inert gas elimination technique (MIGET) was used for detailed analysis of *V/Q* matching. SPECT/MIGET and central haemodynamics and pulmonary gas exchange were recorded during anaesthesia before and 60 min after induction of PP.

Results. SPECT during PP showed no or only poorly ventilated regions in the dependent lung compared with the ventilation distribution during anaesthesia before PP. PP was accompanied by redistribution of blood flow away from the non- or poorly ventilated regions. *V/Q* analysis by MIGET showed decreased shunt from 9 (SD 2) to 7 (2)% after induction of PP ($P<0.05$). No regions of low *V/Q* were seen either before or during PP. Almost no regions of high *V/Q* developed during PP (1% of total ventilation). Pa_{O_2} increased from 33 (1.2) to 35.7 (3.2) kPa ($P<0.01$) and arterial to end-tidal P_{CO_2} gradient ($Pa\varepsilon'_{CO_2}$) increased from 0.3 (0.1) to 0.6 (0.2) kPa ($P<0.05$).

Conclusions. Perfusion was redistributed away from dorsal, collapsed lung regions when PP was established. This resulted in a better *V/Q* match. A possible mechanism is enhanced hypoxic pulmonary vasoconstriction.

Keywords: blood flow; gas exchange; laparoscopy; lung; measurement techniques; model; pig; respiratory; surgery

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During general anaesthesia and mechanical ventilation, lungs are compressed by a cranial shift of the diaphragm, promoting atelectasis formation.[1] Intra-abdominal insufflation of carbon dioxide (CO_2) for laparoscopic surgery (pneumoperitoneum, PP) causes further shift of the diaphragm and increased lung collapse, decreased respiratory compliance, and increased airways pressure, as shown in clinical and experimental studies.[2–5] In addition, CO_2 is absorbed across the peritoneal epithelium. This is a likely cause of the recurrent findings of acidaemia, hypercapnoea, and cardiovascular instability during PP.[6 7] CO_2 may also strengthen hypoxic pulmonary vasoconstriction (HPV), either by causing hypercapnoea or acidosis, or both.[6 8]

Perfusion of non-ventilated alveoli causes shunt and impaired oxygenation of blood.[9 10] CO_2 elimination may also be impaired.[11 12] Despite the increase in atelectasis by PP, shunt need not increase and arterial oxygenation need not decrease,[13] although a decrease in Pa_{O_2} has also been reported.[9 10] This seeming paradox of more atelectasis and less shunt has not yet been explained.

The present animal experiment was initiated to study the effects of PP on ventilation–perfusion distribution and gas exchange by isotope technique (single photon emission computed tomography, SPECT) and multiple inert gas elimination technique (MIGET).

Methods

After approval by the local animal ethics committee, 14 2-month-old healthy piglets [mean body weight 30 (2) kg] of the Hampshire, Yorkshire, and Swedish country breeds from a local breeder were studied.

Anaesthesia and mechanical ventilation

All pigs were anaesthetized by an i.m. injection of xylazine (2.2 mg kg^{-1}, Rompun®; Bayer, Leverkusen, Germany), tiletamine/zolazepam (6 mg kg^{-1}, Zoletil®; Virbac, Carros, France), and atropine (0.04 mg kg^{-1}, NM Pharma, Stockholm, Sweden). The pigs' lungs were mechanically ventilated after intubation with an ID 7.0 mm cuffed tracheal tube (Mallinckrodt, Athlone, Ireland). Anaesthesia was maintained by continuous infusion of fentanyl (5 μg kg^{-1} h^{-1}, Leptanal®; Janssen-Cilag AB, Sweden), ketamine (25 mg kg^{-1} h^{-1}, Ketaminol vet.®; Intervet, Boxmeer, The Netherlands), and propofol (3 mg kg^{-1} h^{-1}, Diprivan®; Astra, Södertälje, Sweden). Pancuronium was given as infusion for muscle relaxation (0.3 mg kg^{-1} h^{-1}, Pavulon®; Organon, Oss, The Netherlands). Ringer's acetate (Pharmacia AB; Stockholm, Sweden) was infused with an average rate of 5 ml kg^{-1} h^{-1} to maintain a constant haemoglobin concentration and stable systemic arterial pressure. Bolus doses of fentanyl were given if systemic arterial pressure increased or there were signs of awareness.

Mechanical ventilation was initiated in the volume-controlled mode (intermittent positive pressure ventilation) (Servo i; Maquet Critical Care AB, Solna, Sweden). Ventilatory frequency was adjusted to achieve normocapnia (Pa_{CO_2}=4.7–6 kPa). Tidal volume (V_t), airway pressures (P_{aw}), and flow were continuously recorded. Static compliance (C_{rs}) of the total respiratory system was calculated as $C_{rs}=V_t$ [$P_{aw\ plateau}-P_{aw\ end\ expiration}$], where airway pressures were measured after end-inspiratory and end-expiratory halts of 3 s.

Monitoring

For pressure measurements and arterial blood sampling, an 18 G catheter was inserted in the left carotid artery. A thermistor-tipped Swan–Ganz catheter (CritiCath™ SP5107H-14 TD; Becton Dickenson, Franklin Lakes, NJ, USA) and another 18 G catheter were introduced into the left external jugular vein. Systemic, pulmonary arterial, and central venous pressures were displayed on a monitor (SC 9000 XL; Maquet Critical Care AB) and were recorded with reference to the mid-thoracic level at end-expiration. End-expiratory carbon dioxide tension ($P_{E'_{CO_2}}$) was measured by capnography implemented in the ventilator (Servo i). Arterial and mixed venous blood samples were analysed with ABL 300 blood gas analyzer and OSM 3 oximeter (Radiometer, Copenhagen, Denmark). $Pa_{E'_{CO_2}}$ was calculated. Cardiac output (Qt) was measured by thermodilution with 10 ml of saline boluses injected into the right atrium. The first measurement was ignored, and the cardiac output was derived from the mean of the three consecutive measurements. The injections were evenly distributed over the respiratory cycle.

SPECT technique

SPECT was used to analyse the spatial ventilation and perfusion distributions during anaesthesia and PP.[14] Ventilation distribution was assessed by inhalation of krypton (^{81m}Kr; $t_{1/2}$: 12 s), being produced by a rubidium generator on site (Mallinkrodt, The Netherlands).[15] Lung blood flow was assessed by i.v. injection of ^{99m}Tc-labelled macroaggregated albumin (^{99m}Tc-MAA) (Pulmocis; CISbiointernational, Gif sur Yvette, France). The animals were put in the supine position with their front legs stretched cranially. Since PP caused a cranial shift of the diaphragm, a new SPECT was done directly after the insufflation of the abdominal cavity, measuring the radioactivity from the first isotope injection. This enabled a background subtraction of the radioactivity when the second injection was done during PP. In order to obtain high pulmonary emission activity in relation to the contribution of activity from preceding measurements, the injected activity was increased from 50 MBq ^{99m}Tc-MAA for the first SPECT scan to 100 MBq ^{99m}Tc-MAA for the next SPECT. Images were acquired on a dual-head gamma camera (Millenium; General Electric Systems, Milwaukee, WI, USA) equipped with all-purpose, medium-energy collimators. SPECT acquisition was made in 60 projections (30 per head) and stored in a 128 by 128 matrix, resulting in a pixel size of 4.42 mm^2. A low-resolution CT scan (covering the same volume as the SPECT) was performed immediately after each SPECT to evaluate lung borders and to enable attenuation correction. The overall scan time for SPECT and CT was ∼40 min.

Data were first reconstructed on an eNTEGRA workstation and later on a Xeleris workstation (General Electric Systems). The reconstruction was performed with an iterative model (OSEM, four iterations and eight subsets) and a Hann filter (cut-off 0.85) for the post-reconstruction filtering on both workstations. The reconstructed volumes were then corrected for radiation spillover using a HERMES workstation (Hermes Medical Solution, Stockholm, Sweden). For each reconstructed slice, the contents were analysed by commercial (HERMES) and custom-made software. After evaluation of the lung borders with the CT, the left and the right lungs were chosen as the regions of interest by drawing the external boundaries of the lung along the inside of the ribs and the internal boundaries along the mediastinal organs. The lungs were divided into 35 equally thick portions in the dorsal to ventral direction, for assessment of the vertical ventilation–perfusion distribution. Similarly, the lungs were divided into 48 equally thick slices from caudal to cranial lung regions for analysis of ventilation–perfusion distribution in that plane.

MIGET technique

Determination of the V_A/Q distribution was undertaken with the MIGET.[16] Six inert gases of different solubilities in blood were dissolved in isotonic saline and infused into a peripheral vein. Arterial and mixed venous blood samples were tonometered with gas and analysed together with an expired gas sample by gas chromatography (Model 5890, Series II; Hewlett-Packard, Waltham, MA, USA). These data enable the construction of a virtually continuous distribution of

V_A/Q ratios against blood flow or ventilation, with separation of shunt ($V_A/Q<0.005$) from regions of low V_A/Q ratios ($0.005<V_A/Q<0.1$; poorly ventilated lung units in relation to their perfusion), and also separation of regions of high V_A/Q ratios ($10<V_A/Q<100$) from dead space (V_D) ($V_A/Q>100$). The mean V_A/Q of the ventilation and perfusion distributions (V_{mean}, Q_{mean}) was calculated. Moreover, the standard deviation of the logarithmic distribution of perfusion (LogSDQ) and ventilation (LogSDV) was calculated as measures of the dispersion (mismatch) of blood flow and ventilation. Finally, the Pa_{O_2} that can be predicted from the V_A/Q distributions was compared with measured Pa_{O_2} (by blood gas analysis).

Study protocol

In both groups (MIGET and SPECT; $n=14$), pigs were ventilated with a V_T of 10 ml kg^{-1}, PEEP 5 cm H_2O, and $F_{I_{O_2}}$ 0.5. On the basis of previous results from our laboratory, $F_{I_{O_2}}$ was increased to 1.0 for 30 min in order to induce atelectasis in the range of 3–5%.[3] $F_{I_{O_2}}$ was then decreased to 0.5 before creation of PP.

PP was created by insufflation of CO_2 into the abdominal cavity *via* a VERRES needle with a common CO_2 insufflator (7060-Insufflator Pelvi Pneu Semm Systems; Wisap, Munich, Germany) until the abdominal pressure (P_{abd}) reached 12 mm Hg. Mechanical ventilation was maintained with the same respirator settings (unaltered ventilation) as before induction of PP. SPECT and MIGET could not be done in the same pig because of technical and logistic reasons. Therefore, in seven pigs, the distributions of ventilation and perfusion were studied by SPECT, and in the other seven pigs, the V_A/Q relationship was studied by MIGET. Measurements of gas exchange, haemodynamics, and SPECT/MIGET were made during anaesthesia and 60 min after induction of PP. At the end of the experiment, pigs were killed by an overdose of potassium chloride.

The investigations were performed in the experimental laboratories of the Department of Clinical Physiology, and in the Department of Nuclear Medicine at the University Hospital in Uppsala.

Statistics

Statistical analysis was performed with the Prism 4 software package (GraphPad Software Inc., San Diego, CA, USA) on a Macintosh computer. Power calculations using a two-sided design at a significance level of 5% ($\alpha=0.05$) and a probability of 80% ($\beta=0.20$) to detect a difference of at least 35% in the development of atelectasis (and subsequent change in ventilation) revealed that a minimum of seven pigs were needed in each group.

Data were tested for normal distribution with the Shapiro–Wilks *W*-test. Normally distributed data are presented as mean and standard deviation (cardiopulmonary, ventilation, and gas exchange variables) and were analysed by repeated-measures one-way analysis of variance (ANOVA) with the *post hoc* Bonferroni correction. Non-normally distributed data were analysed by Friedman's ANOVA and Tukey's HSD. MIGET data were tested with an unpaired *t*-test. Differences were considered statistically significant if $P<0.05$.

Results

Respiration and haemodynamics

Respiratory and haemodynamics data were similar in the MIGET and SPECT groups, and data have therefore been pooled in Table 1. Pa_{O_2}, $P_A-a_{O_2}$, Pa_{CO_2}, and $Pa_{E'_{CO_2}}$ increased during PP, and pH decreased. Peak airway pressure and airway plateau pressure almost doubled, and respiratory compliance decreased to less than half the value before PP.

Central venous, mean pulmonary arterial, and pulmonary capillary wedge pressures increased during PP. No changes in cardiac output were seen.

Ventilation and perfusion distributions (SPECT group)

The distributions of ventilation and blood flow in the caudal–cranial direction during anaesthesia before and during PP are shown in Figure 1 and in the dorsal–ventral direction in Figure 2. The ventilation and perfusion distributions along the caudal–cranial axis were similar to each other, indicating a rather good match of ventilation and perfusion. During PP, a shift of ventilation and blood flow along the *x*-axis away

Table 1 Respiratory and haemodynamic data. No differences were seen in any variable between the MIGET and SPECT groups; pooled data from both groups are therefore shown ($n=14$). Baseline, ventilation with 50% O_2; PP, pneumoperitoneum with abdominal pressure of 12 mm Hg by CO_2 insufflation; Pa_{O_2}, arterial oxygen tension; $P_A-a_{O_2}$, difference between alveolar and arterial PO_2; Pa_{CO_2}, arterial carbon dioxide tension; $P_{E'_{CO_2}}$, end-expiratory carbon dioxide tension; $Pa_{E'_{CO_2}}$, difference between arterial and end-expiratory PCO_2; pHa, arterial pH; MV, minute ventilation; $P_{aw\ peak}$, peak airway pressure; $P_{aw\ plateau}$, plateau airway pressure; C_{rs}, respiratory compliance; MAP, mean arterial pressure; CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; MPAP, mean pulmonary arterial pressure; CO, cardiac output. Data given as mean (SD) ($n=14$). *P-value ($P<0.05$) is calculated as unpaired *t*-test in comparison with PP to baseline

	Baseline	PP 60 min
Pa_{O_2} (kPa)	33.1 (1.2)	35.7 (3.2)*
$P_A-a_{O_2}$ (kPa)	6.2+1.4	1.9+3.2*
Pa_{CO_2} (kPa)	5.3 (0.4)	6.5 (0.4)*
$P_{E'_{CO_2}}$ (kPa)	5.0 (0.4)	5.9 (0.4)*
$Pa_{E'_{CO_2}}$ (kPa)	0.35 (0.1)	0.61 (0.2)*
pHa	7.46 (0.05)	7.35 (0.04)*
MV (litre min^{-1})	6.2 (0.9)	6.2 (0.9)
$P_{aw\ peak}$ (cm H_2O)	19 (2)	33 (4)*
$P_{aw\ plateau}$ (cm H_2O)	16 (2)	29 (4)*
C_{rs} [ml (cm H_2O)$^{-1}$]	28 (5)	12 (2)*
MAP (mm Hg)	93 (14)	95 (10)
CVP (mm Hg)	7 (3)	9.5 (4)*
MPAP (mm Hg)	18 (2)	20 (4)*
PCWP (mm Hg)	8 (2)	10 (3)*
CO (litre min^{-1})	4.1 (0.7)	3.9 (0.8)

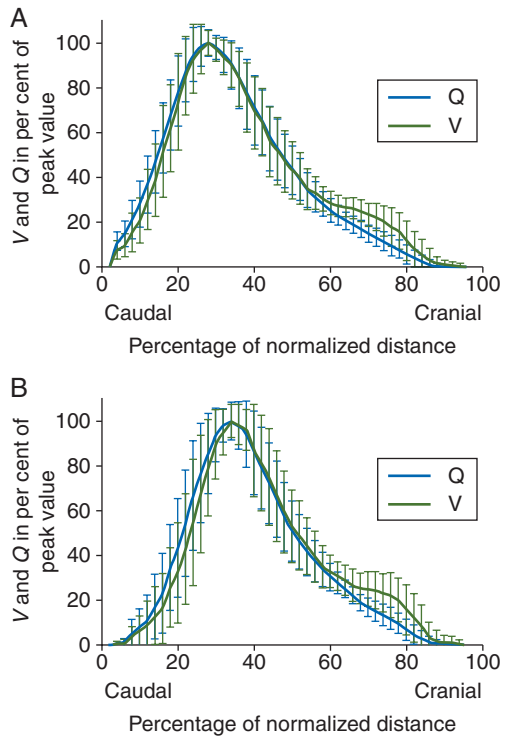

Fig 1 SPECT: ventilation and blood flow distributions in the caudal-to-cranial direction (horizontal axis) during anaesthesia before (A) and during PP (B). Ventilation (V) and blood flow (Q) are shown in per cent of their peak values. Note the shift of ventilation and perfusion away from the caudal region after induction of PP and the balanced V/Q distribution. (Mean+SD of all seven pigs studied with SPECT.)

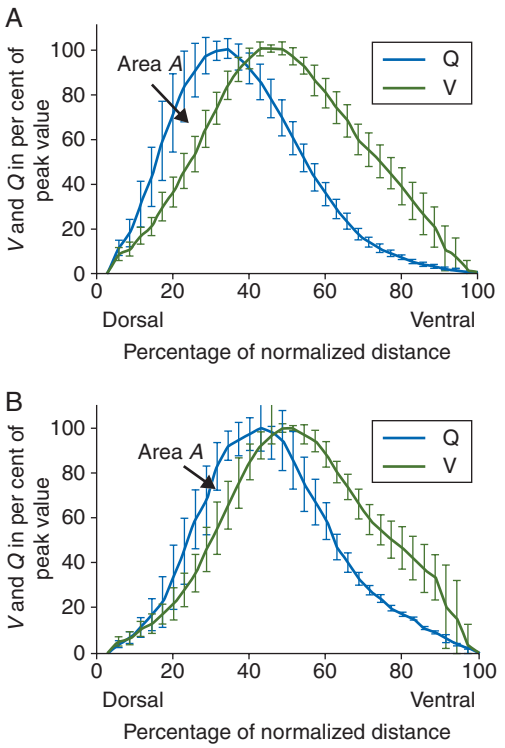

Fig 2 SPECT: ventilation and blood flow distributions from dorsal to ventral (vertical axis) during anaesthesia before (A) and during PP (B). Ventilation (V) and blood flow (Q) are shown in per cent of their peak values. 'Area A', as described in the Results section, is shown in both panels. Note the larger difference in ventilation and perfusion distributions than in the caudal-to-cranial direction shown in Figure 1. Note also the smaller area A during PP, indicating less of 'over-perfused' areas relative to their ventilation. This should improve oxygenation when compared with the recording during baseline before PP. [Mean (SD) of all seven pigs studied with SPECT.]

from caudal towards cranial regions was seen. The starting point on the x-axis was kept constant relative to the spine. The shift of ventilation and perfusion can therefore be explained both by a cranial displacement of the diaphragm and by increase of atelectasis in juxtadiaphragmatic regions (Figs 1 and 2). The displacement along the caudal–cranial axis was similar for ventilation and blood flow. Thus, no worsening of the matching of ventilation and blood flow along the horizontal (caudal–cranial) axis occurred with PP.

The distributions of ventilation and blood flow along the vertical (dorsal–ventral) axis showed a larger difference between them than along the caudal–cranial axis. Thus, ventilation was distributed to ventral regions to a much larger extent than perfusion (Fig. 2). This indicates that near the diaphragm, there is reduced ventilation but persistence of blood flow. With PP, there were further shifts of ventilation and blood flow towards ventral regions with more marked redistribution of perfusion than of ventilation.

The matching of the ventilation and blood flow can also be roughly estimated by analysing the area inscribed by the ventilation and perfusion curves. We used the following mathematical calculation: the area inscribed by the ventilation and perfusion curves for the part where perfusion is larger than ventilation, $A=\sum\Delta(Q-V)$ (also shown in Fig. 2), and the area inscribed by the perfusion curve, $B=\sum Q$, and calculated the percentage of perfusion going to less ventilated than perfused regions as $C=A/B\times100$. The area A decreased in the dorsal to ventral direction during PP as did C [C before PP: caudal–cranial: 4 (2), dorsal–ventral: 19 (3); C during PP: caudal–cranial: 5 (2), dorsal–ventral: 15 (3), $P<0.05$]. Moreover, a simplified estimation of the V/Q matching was made by calculating the mean of ventilation and perfusion distributions (V_{mean}, Q_{mean}) and the scatter around the mean (similar to logSDV and logSDQ by MIGET). V_{mean} was unaltered by PP [1.36 (0.13) and 1.40 (0.09)] and Q_{mean} increased from 0.81 (0.11) to 0.96 (0.15) ($P<0.05$). Furthermore, logSDV decreased [0.52 (0.04) to 0.47 (0.03); $P<0.05$] as did logSDQ [0.45 (0.07) to 0.34 (0.04); $P<0.05$] during PP. These findings describe a better match of ventilation and blood flow during PP.

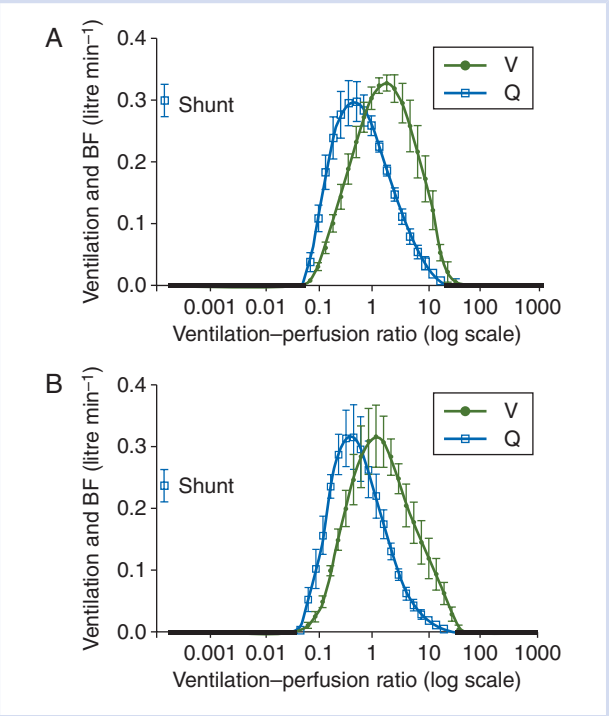

Fig 3 MIGET: ventilation and blood flow plotted against V_A/Q ratios during anaesthesia (A) and after creation of PP (B). Note the slight decrease in shunt and closer matching of ventilation and blood flow distributions during PP (see also V_{mean} and Q_{mean} in Table 2).

Table 2 Ventilation–perfusion matching (MIGET group; $n=7$). Baseline, ventilation with 50% O_2; PP, pneumoperitoneum with abdominal pressure of 12 mm Hg by CO_2 insufflation; Shunt, perfusion of non-ventilated areas ($V_A/Q<0.005$); low V_A/Q, low ventilation to perfusion ratio ($0.005<V_A/Q<0.1$); regions of normal ventilation to perfusion ratios ($0.1<V_A/Q<1$); high V_A/Q, high ventilation to perfusion ratio ($1<V_A/Q<10$); Dead space, ventilated but non-perfused areas ($10<V_A/Q<100$); Log SDV, log standard deviation of ventilation distribution; log SDQ, log standard deviation of perfusion distribution; V_{mean}, mean of ventilation distribution; Q_{mean}, mean of blood flow distribution; $(V-Q)_{mean}$, difference between V_{mean} and Q_{mean}. Data given as mean (SD) ($n=7$). *P-value ($P<0.05$) is calculated as unpaired t-test of PP in comparison with baseline

	Baseline	PP 60 min
Shunt (% QT)	9.0 (2.0)	7.0 (2.0)*
Low V_A/Q (% QT)	0	0
$0.1<V_A/Q<1$ (% QT)	53 (6)	55 (3)
$1<V_A/Q<10$ (% QT)	38 (6)	37 (4)
Dead space (% VE)	38 (2)	39 (3)
Log SDV	0.86 (0.03)	0.83 (0.03)
Log SDQ	0.84 (0.02)	0.80 (0.03)
V_{mean}	1,89 (0.37)	1.58 (0.41)
Q_{mean}	0.87 (0.16)	0.99 (0.2)
$(V-Q)_{mean}$	1.03 (0.45)	0.58 (0.24)*

Ventilation–perfusion matching (MIGET group)

The V_A/Q distributions are shown in Figure 3 and Table 2. The distributions had a remaining sum of squares of 1.24 (0.56), which indicates high methodological accuracy. Shunt decreased after creation of PP. No low V_A/Q was seen either before or during PP. A small amount of high V_A/Q appeared during PP, whereas V_D was unaltered. V_{mean} and Q_{mean} expressed as V_A/Q ratio came closer to each other during PP ($P<0.05$; Table 2). This suggests an improved match of ventilation and perfusion. LogSDQ and logSDV were not significantly altered by PP (Table 2). The difference in Pa_{O_2} that can be predicted from the V_A/Q distribution and measured from blood gas analysis was small [1.6 (0.8) kPa] for the measured Pa_{O_2} of around 35 kPa, which is a further support of good methodological accuracy.

Discussion

The main findings of the present study are that pulmonary blood flow shifts away from the dorsal to ventral regions to a higher extent than ventilation during PP. Ventilation is most likely shifted away because of atelectasis formation, as shown previously both in clinical and experimental studies.[2 3] Thus, atelectasis was about 4% during anaesthesia in our porcine model and increased to 10% during PP.[2 3] These redistributions result in improved oxygenation and gas exchange during PP.

It is well known that humans develop atelectasis in dependent lung regions after induction of anaesthesia.[17] The magnitude of the pulmonary shunt correlates with the size of the atelectasis.[1 14] In supine subjects, PP to an abdominal pressure of 12 mm Hg caused a cranial shift of the diaphragm of 1–3 cm, decreased lung volumes and increased airway pressures,[2 5] and increased the formation of atelectasis but did not, in most studies, increase the shunt.[2 3] In the present study, pulmonary shunt decreased during PP and areas with low V_A/Q were not seen. This is in line with previous results using MIGET in humans.[13] Also, haemodynamic responses to PP are similar to studies gathered in the European Association for Endoscopic Surgery guidelines.[18] The dead space measured by the multiple inert gas technique showed normal values for pigs throughout the investigation. It was not altered by the insufflation of CO_2, again in keeping with previous findings in humans.[13] Acidosis and hypercarbia were seen in similar previous studies.[6] Such changes may affect HPV and enhance redistribution of blood flow.[19–22] Use of another gas for creation of PP, or abdominal lift, caused no changes in acid–base balance and oxygenation.[6 23]

What is new in the present study is the demonstration of a shift of blood flow away from dorsal, dependent regions during PP, a redistribution that was larger than the decrease in ventilation that was reasonably caused by the lung collapse. A possible explanation may thus be more efficient HPV.

Either an increase in Pa_{CO_2} or a decrease in $P_{E'_{CO_2}}$ or changes in both can explain the finding of an increased $Pa_{E'_{CO_2}}$. A decrease in $P_{E'_{CO_2}}$ would be seen with an increase in dead space (V_D).

However, the opposite, an increase in $P_{E'_{CO_2}}$ (Table 1), was seen and V_D was not altered, as mentioned above (Table 2). Pa_{CO_2}, on the other hand, increased (Table 1) and this may be caused, after ruling out an increase in V_D, by an increase in shunt. The shunt enables mixed venous blood to pass through non-ventilated lung tissue and increase Pa_{CO_2}. An increase in $Pa_{E'_{CO_2}}$ has even been nicknamed 'shunt-dead space', although it need not reflect a real V_D.[20]

The increase in $Pa_{E'_{CO_2}}$ has been shown to be a good predictor of the increase in atelectasis during PP (assuming constant dead space),[3] whereas Pa_{O_2} and shunt do not guide in estimating atelectasis.[2 10] Furthermore, the absorption of CO_2 during PP, which should increase both Pa_{CO_2} and $P_{E'_{CO_2}}$ at constant ventilation, showed no effect on $Pa_{E'_{CO_2}}$, as judged from the maintained $Pa_{E'_{CO_2}}$ with increasing CO_2 levels in blood and expired air in a previous study from our group.[3] Thus, the increased $Pa_{E'_{CO_2}}$ may reasonably be explained by enhanced atelectasis formation.

PP shifted ventilation and perfusion away from caudal regions along the horizontal axis but to a similar extent, so that a fairly good matching of ventilation and blood flow was maintained. Further support of a better matching during PP, besides reduced shunt and $P_A-a_{O_2}$, comes from the more closely located V_{mean} and Q_{mean} on the *V/Q* axis, both with SPECT and MIGET data.

Anaesthetic drugs may modulate the HPV. Ketamine, as used in our study, may act as a bronchodilator,[24] but no effect on HPV has been reported. It should also be mentioned that total i.v. anaesthesia with ketamine is common in respiratory studies.[25 26] HPV can be blunted by vagal stimulation,[27] but atropine, as used in our study, should protect against this effect.[28]

Limitations of the study include that HPV is well developed in pigs[29 30] that may have enhanced the redistribution of perfusion. Still, findings in this study on pulmonary vascular pressures, shunt, and gas exchange are comparable with findings in humans.[2 13 31] Another limitation of the study is that the MIGET and SPECT studies could not be done in the same animals, because of technical and logistic reasons. Study protocols were comparable, only time schedules (SPECT longer than MIGET) of the measurements were different.

In conclusion, we have shown an improved gas exchange and oxygenation, caused by redistribution of blood flow away from collapsed lung tissue during PP. A likely, but not yet proven, explanation is enhanced HPV, possibly mediated via increased Pa_{CO_2}. This may be of clinical interest when giving patients anaesthesia for laparoscopic surgery.

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Conflict of interest

None declared.

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