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Spontaneous Breathing during General Anesthesia Prevents the Ventral Redistribution of Ventilation as Detected by Electrical Impedance Tomography

A Randomized Trial

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ABSTRACT

Background: Positive-pressure ventilation causes a ventral redistribution of ventilation. Spontaneous breathing during general anesthesia with a laryngeal mask airway could prevent this redistribution of ventilation. We hypothesize that, compared with pressure-controlled ventilation, spontaneous breathing and pressure support ventilation reduce the extent of the redistribution of ventilation as detected by electrical impedance tomography.

Methods: The study was a randomized, three-armed, observational, clinical trial without blinding. With approval from the local ethics committee, we enrolled 30 nonobese patients without severe cardiac or pulmonary comorbidities who were scheduled for elective orthopedic surgery. All of the procedures were performed under general anesthesia with a laryngeal mask airway and a standardized anesthetic regimen. The center of ventilation (primary outcome) was calculated before the induction of anesthesia (AWAKE), after the placement of the laryngeal mask airway (BEGIN), before the end

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What We Already Know about This Topic

- Controlled mechanical ventilation under general anesthesia increases ventilation in the ventral parts of the lung
- We do not know whether pressure support ventilation prevents the redistribution of ventilation

What This Article Tells Us That Is New

- Using a noninvasive and radiation-free imaging technique of lung function, electric impedance tomography, under general anesthesia in nonparalyzed adults, this study demonstrates no difference of regional ventilation distribution between the modes of pressure-controlled ventilation and pressure support ventilation, whereas spontaneous breathing prevented the redistribution

of anesthesia (END), and after arrival in the postanesthesia care unit (PACU).

Results: The center of ventilation during anesthesia (BEGIN) was higher than baseline (AWAKE) in both the pressure-controlled and pressure support ventilation groups (pressure control: 55.0 vs. 48.3, pressure support: 54.7 vs. 48.8, respectively; multivariate analysis of covariance, $P < 0.01$), whereas the values in the spontaneous breathing group remained at baseline levels (47.9 vs. 48.5). In the postanesthesia care unit, the center of ventilation had returned to the baseline values in all groups. No adverse events were recorded.

Conclusions: Both pressure-controlled ventilation and pressure support ventilation induce a redistribution of ventilation toward the ventral region, as detected by electrical impedance tomography. Spontaneous breathing prevents this redistribution.

INTUBATION and mechanical ventilation cause a redistribution of ventilation:^{1,2} compared with spontaneous breathing (SB) in the awake state in supine position, positive-

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pressure ventilation (PPV) increases the aeration in the ventral parts of the lung, whereas the dorsal parts contain less air. General anesthesia with a laryngeal mask airway allows safe and sufficient patient ventilation with both SB and PPV,³ and SB during general anesthesia could prevent the redistribution of ventilation.

Pressure support ventilation (PSV) was originally invented as a weaning tool for intensive care patients.⁴ Contrary to pressure-controlled ventilation (PCV), which replaces the patient's own breathing efforts, PSV supports the patient's breathing; when the patient inhales, the ventilator exerts a positive pressure to facilitate the patient's inspiration. Since SB remains intact during PSV, we expect PSV to cause less redistribution of ventilation than PCV.

Electrical impedance tomography (EIT) is a new tool for the assessment of regional lung ventilation.⁵ The EIT device measures the impedances between a series of electrodes placed around the thorax. The nature of the tissue between the electrodes determines the bioimpedance: high concentration of water, electrolytes, and cells reduces the impedance, whereas fat, bone, and air increase it. By means of a mathematical process (backprojection), the EIT device generates a two-dimensional map of the impedances across the transverse plane under the electrodes. EIT is noninvasive, radiation-free, and allows dynamic imaging and analysis of ventilation at the bedside and in the operating room.

The aim of the study was to assess the distribution of ventilation during SB, PCV, and PSV by means of EIT in order to elucidate the effects and the interaction of SB and PPV on ventilation distribution during general anesthesia.

We hypothesize that, compared with PCV, SB and PSV during general anesthesia reduce the extent of the redistribution of ventilation as detected by EIT.

Materials and Methods

The study was a randomized, three-armed, observational, clinical trial at a single site, conducted by the Department of Orthopedic Anesthesia at the University Hospital in Dresden, Germany, between March and October 2010. With approval from the local ethics committee (Ethikkommission der Medizinischen Fakultät Carl Gustav Carus, University Dresden, study identifier EK 375122009; ClinicalTrials.gov identifier NCT01073917), 30 patients who were scheduled for elective knee, foot, or ankle surgery were screened by the anesthesia team, and after successful screening they were included in the study after obtaining written informed consent. The exclusion criteria were contraindications to the drugs used for anesthesia, pregnancy or breastfeeding, severe cardiac or pulmonary comorbidities (defined as American Society of Anesthesiologists physical status III or greater), and contraindications to a laryngeal mask airway (LMA) or EIT. A full flowchart of the study is represented in figure 1.

After arrival in the operating room, the patients were connected to a monitoring system (Philips MP70; Philips Deutschland GmbH, Hamburg, Germany) for monitoring

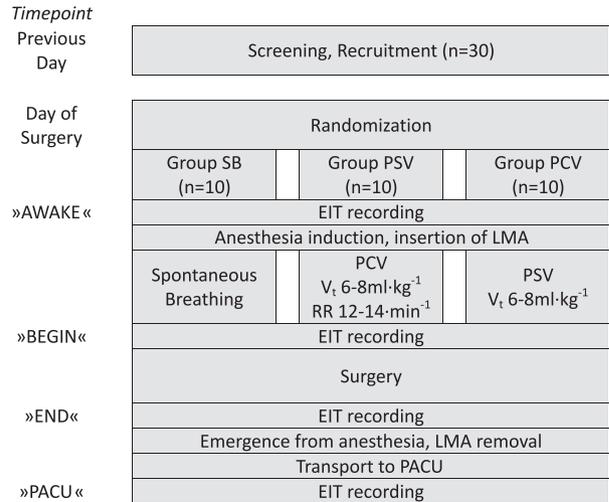


Fig. 1. Study flowchart. All patients completed the study and remained in the groups they had been randomized to. Analysis was performed based on intention to treat. EIT = electrical impedance tomography; LMA = laryngeal mask airway; PACU = postanesthesia care unit; PCV = pressure-controlled ventilation; PSV = pressure support ventilation; RR = respiratory rate; SB = spontaneous breathing; V_t = tidal volume.

according to clinical standards (heart rate, noninvasive blood pressure, and oxygen saturation). The electrode belt of the EIT device (EIT Evaluation Kit 2; Dräger Medical, Lübeck, Germany) was placed around the patient's chest at the level of the sixth intercostal space. After a resting period of 5 min, baseline values were recorded. The patient was in supine position, awake but lightly sedated with midazolam, and breathing spontaneously without supplemental oxygen administration. Regional anesthesia of the leg was done for certain procedures (*e.g.*, knee arthroplasty, hammer toe surgery, cruciate ligament repair).

For randomization, we prepared sealed opaque envelopes (10 envelopes per group). A single envelope was opened before the induction of general anesthesia. Anesthesia was induced with midazolam (2 mg), sufentanil (0.1 µg/kg) and propofol (1–2 mg/kg). No neuromuscular blocking agent was given. After the placement of the LMA, the patients were connected to a respirator (ZEUS; Dräger Medical) and manually ventilated to achieve a sevoflurane level of 0.7 minimum alveolar concentration (approximately 1.1% end-tidal concentration). Bispectral index monitoring (BIS, Philips MP70; Philips Deutschland GmbH) was used in addition to clinical judgment to ensure adequate depth of anesthesia.

According to the randomization, the patients in the SB group did not receive any mechanical ventilation. The patients in the PCV group were ventilated in pressure-control mode. The inspiratory pressure was adjusted to achieve a tidal volume of 6–8 ml/kg. The respiratory rate was set to 12–14/min. The patients in the PSV group were allowed to breathe spontaneously with no mandatory respiratory rate. Again, the pressure support was adjusted to achieve a tidal

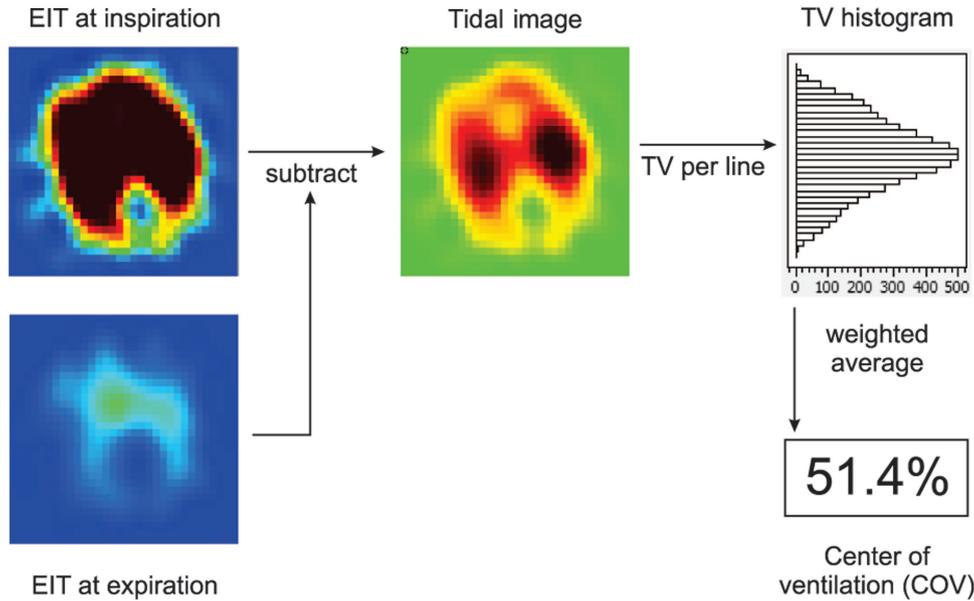


Fig. 2. Calculating the center of ventilation. The electrical impedance tomography (EIT) image at expiration is subtracted from the EIT image at inspiration, resulting in a tidal EIT image that visualizes the two-dimensional distribution of the ventilation in the thorax. Summarizing the tidal variation (TV) per line results in a dorsoventral histogram of the tidal variation. The weighted average of the histogram is the center of ventilation (COV).

volume of 6–8 ml/kg. The common settings for all of the groups were as follows: an inspired fraction of oxygen (FiO₂) of 0.8, zero positive end-expiratory pressure (PEEP), and 0.7 minimum alveolar concentration of sevoflurane. Nitrous oxide was not used. Repeated boluses of 0.1 μg/kg sufentanil were administered according to the patients’ clinical needs (defined as: respiratory rate more than 10/min in groups SB and PSV; BIS more than 60, tachycardia/hypertension or patient movement in all groups).

At the end of the surgical procedure, sevoflurane administration was stopped, and the LMA was removed after the patient regained consciousness and began to breathe sufficiently. After transport to the postanesthesia care unit (PACU), the patients were treated according to clinical standards.

Ventilation data (respiratory rate, tidal volume, end-tidal carbon dioxide, volatile anesthetics, oxygen concentration, and airway pressures) were extracted from the ZEUS ventilator by using the built-in medibus interface (MedLink, Nor-

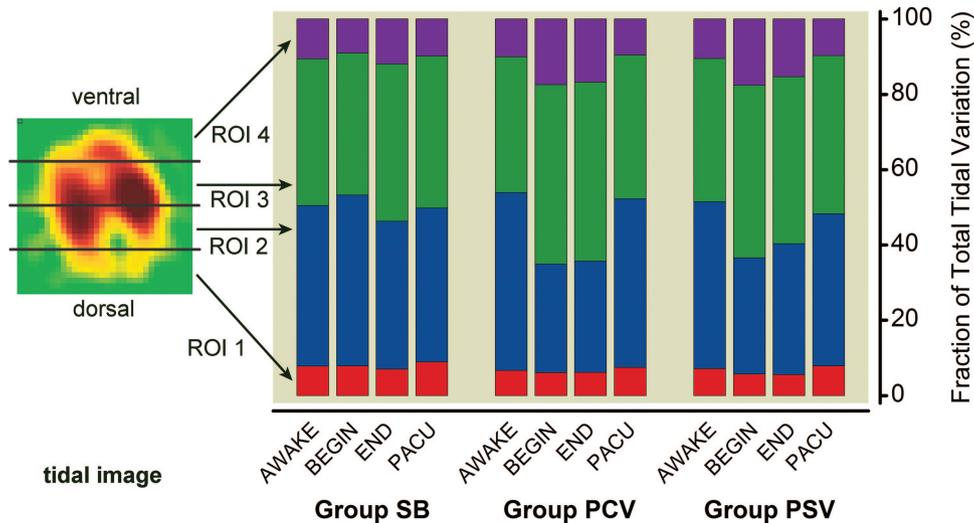


Fig. 3. Percentage of total tidal variation per region of interest. The stacked bars illustrate the changes in the ventilation distribution across the four regions of interest (ROI; also see table 3). The spontaneous breathing (SB) group is uniform throughout the case, whereas the pressure control (PCV) and pressure support (PSV) groups exhibit an increase in ventilation in ROI 3 and 4 and a decrease in ROI 2 at time-points BEGIN and END, indicating a ventral shift in the ventilation. ROI 2, 3, and 4 in the SB group are significantly different from those in groups PCV and PSV ($P < 0.05$; MANCOVA with Sidak adjustment). AWAKE = before the induction of anesthesia; BEGIN = after the placement of the laryngeal mask airway; END = before the end of anesthesia; PACU = after arrival in the postanesthesia care unit.

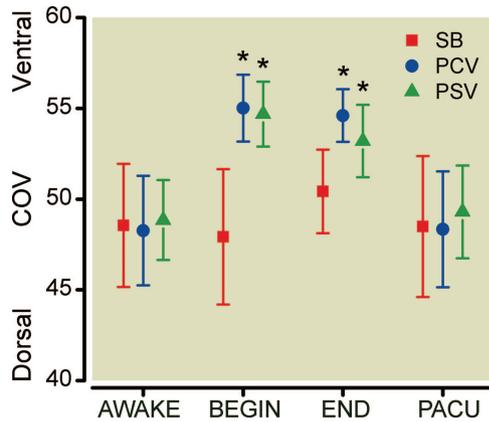


Fig. 4. Changes in center of ventilation (COV). Center of ventilation, given as a mean \pm 95% CI. * indicates $P < 0.05$ versus AWAKE. AWAKE = before the induction of anesthesia; BEGIN = after the placement of the laryngeal mask airway; END = before the end of anesthesia; PACU = after arrival in the postanesthesia care unit; PCV = pressure-controlled ventilation; PSV = pressure support ventilation; SB = spontaneous breathing.

tis Ingenieurbüro, Lübeck, Germany). The EIT data were recorded with a prototype provided by Dräger Medical. The EIT prototype has built-in software that automatically adjusts and calibrates the gain settings. It calculates the impedances in 1024 pixels across a cross-section of the thorax (fig. 2). Software that was developed by the authors was used to analyze the EIT data at each of the following time-points: before induction (AWAKE), 10 min after LMA insertion (BEGIN; in groups SB and PSV, 10 min after spontaneous ventilation had recommenced), at the end of the procedure before sevoflurane administration was stopped (END), and in the PACU, 10–15 min after arrival (PACU). For each time-point, we analyzed a sample of 5 or 6 consecutive breaths. The software analyzes the distribution of the ventilation-induced impedance changes and calculates the mean center of ventilation (COV; fig. 2), similar to the method introduced by Frerichs *et al.*^{6,7}

To analyze the regions of interest (ROI), the tidal EIT image was divided into four stacked regions, each covering 25% of the ventrodorsal diameter (fig. 3). We added the impedance changes of all of the EIT pixels per ROI and divided the sum by the total sum of the impedance changes

in the entire tidal EIT image, resulting in the fraction of the total tidal variation per ROI (given in %). An increase in the fractional tidal variation per ROI indicates a redistribution of ventilation toward this ROI.

Statistics

No *a priori* power analysis was conducted because we did not know the exact effect size that we would see. However, based on the effects seen in a previous study,⁸ we estimated that 10 patients per group would be sufficient to achieve adequate power levels.

The primary outcome was the increase in COV during anesthesia (time-points BEGIN and END) compared with baseline (AWAKE). Secondary outcome was the possible change in peripheral oxygen saturation caused by the anesthesia (at time-points AWAKE *vs.* PACU).

One-way ANOVA was used to test the differences in the patient and case characteristics; chi-square test was used for gender. $P < 0.05$ was considered to be statistically significant. Two-tailed Student *t* tests for paired samples with Bonferroni correction for multiple testing was used to compare the COV data with the baseline measurements (fig. 4). Cohen's *d* for within-subject comparisons was used to determine the effect size. Kruskal–Wallis' test was used for non-parametric data.

The changes in the COV and ROI over time in each group were analyzed by multivariate analyses of variance with Sidak α adjustments, using the baseline values as a covariate (MANCOVA). A formal data safety monitoring board was not required by the local ethics committee, but a coauthor who was not involved in the data acquisition (AH) reviewed the data for plausibility and accuracy.

Results

All of the randomized patients successfully completed the study; LMA and surgery were tolerated well, and all patients recovered quickly. There were no adverse events or complications. We lost the data for a single time-point for one patient (BEGIN for one patient in the SB group) because of a device malfunction caused by interference from the electrocautery. All patients remained in the groups they had been randomized to. The data were analyzed on an intention-to-treat basis.

Table 1. Patient and Case Characteristics

	Spontaneous Breathing (n = 10)	Pressure-controlled Ventilation (n = 10)	Pressure Support Ventilation (n = 10)	<i>P</i> Value
Height (cm)	175 \pm 9	178 \pm 11	174 \pm 10	0.62*
Weight (kg)	79 \pm 10	82 \pm 14	81 \pm 14	0.81*
Female gender (%)	40	50	60	0.67‡
Duration of anesthesia (min)	71 \pm 27	58 \pm 8	85 \pm 39	0.11*
Total sufentanil dose (μ g)	19.7 \pm 6.8	20.3 \pm 5.1	27.3 \pm 16.8	0.74†
Additional regional anesthesia	1 of 10	1 of 10	5 of 10	0.51‡

The data are presented as the mean \pm SD or %.

* ANOVA, † Kruskal–Wallis, or ‡ chi-square, when appropriate.

Table 2. Ventilation

	Spontaneous Breathing (n = 10)	Pressure-controlled Ventilation (n = 10)	Pressure Support Ventilation (n = 10)	P Value
End-tidal carbon dioxide (mmHg)				
BEGIN	46 (43–49)	38 (36–40)	43 (39–47)	<0.001†
END	47 (42–52)	36 (35–37)	42 (38–46)	<0.001†
Tidal volume (ml)				
BEGIN	397 (253–539)	522 (466–577)	487 (400–575)	0.14*
END	530 (389–670)	517 (469–565)	574 (456–693)	0.51*
Respiratory rate (per min)				
BEGIN	8 (6–10)	13 (12–13)	9 (6–12)	<0.001†
END	8 (7–9)	13 (12–14)	9 (7–12)	<0.001†
Minute ventilation (l)				
BEGIN	3.2 (2.5–3.8)	6.6 (6.1–7.1)	4.9 (3.2–5.7)	<0.001*
END	4.3 (3.5–5.0)	6.8 (6.2–7.4)	5.4 (4.1–6.8)	<0.001*
Peak pressure (mbar)				
BEGIN	3 (3–3)	11 (10–13)	12 (10–13)	<0.001†
END	3 (3–3)	12 (11–13)	11 (7–15)	<0.001†
Oxygen saturation on room air (%)				
BASELINE	100 (98–100)	99 (97–100)	100 (97–100)	0.65†
Postanesthesia care unit	98 (98–99)	99 (97–100)	98 (94–99)	0.64†

Measurements were taken at the beginning and end of anesthesia (time-points BEGIN and END). The data are presented as the mean (95% CI); the oxygen saturation is presented as median (quartiles).

* One-way ANOVA, † Kruskal–Wallis, when appropriate.

There were no significant differences in the groups' patient and case characteristics (table 1). The types of surgery were knee arthroscopy (26), metal removal (2), knee arthroplasty (1), and hammertoe surgery (1), and there were no significant differences across the groups ($P = 0.48$).

After induction of anesthesia and insertion of the LMA, spontaneous breathing in groups SB and PSV resumed after 2.4 min with no difference between these two groups. The patients in group PCV were mechanically ventilated immediately after insertion of the LMA. During anesthesia, the mean end-tidal carbon dioxide was lower in the PCV group (37 mmHg) than in the SB (47 mmHg) and PSV (43 mmHg; $P < 0.01$; table 2) groups. The mean tidal volume was approximately 500 ml, with no differences across the groups. The peak airway pressure was lower in the SB group (3 mbar) than in PCV and PSV groups (12 mbar and 11 mbar, respectively; $P < 0.01$). The peripheral blood oxygen saturation in room air measured by plethysmography was not different across the groups, either at baseline (AWAKE) or after arrival in the PACU (PACU).

Image quality of the EIT recordings was good; sample tidal EIT images and corresponding COV values are reproduced in figure 5. The histogram of the proportionate tidal variations in each row of the tidal image (fig. 6) show a pronounced shift of the tidal variations toward the ventral rows during PPV (time-point AWAKE), whereas the distribution in group SB remains unchanged. The center of ventilation (COV) before the induction of anesthesia (time-point AWAKE) was 48.7 ± 3.8 ; there were no statistically significant differences across the three groups (fig. 4). Throughout anesthesia (time-points BEGIN and END), we observed a statistically significant ventral shift in the COV of

about 10% in both of the pressure-ventilated groups (effect size BEGIN vs. AWAKE in group PCV: 1.87; in group PSV: 1.99). In the SB group, the COV remained at baseline. After extubation (time-point PACU), there was no difference in

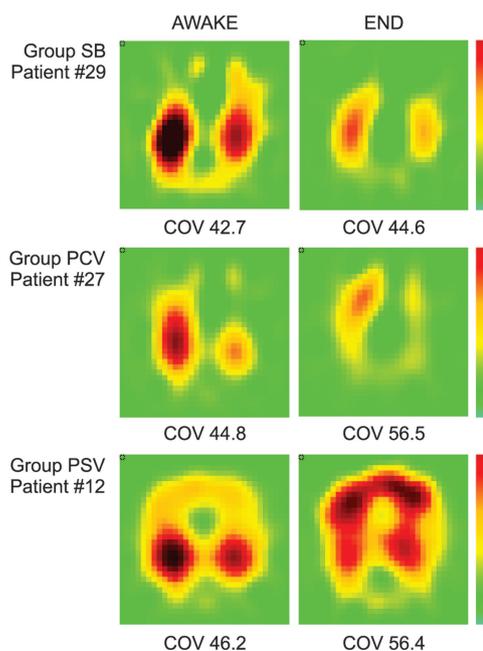


Fig. 5. Samples of electrical impedance tomography tidal images. Sample recordings of the ventilation distribution at baseline (AWAKE) and at the end of general anesthesia (END); each of the same patient per group. Green pixels indicate no impedance change, yellow through red indicate small through large changes. COV = center of ventilation; PCV = pressure-controlled ventilation; PSV = pressure support ventilation; SB = spontaneous breathing.

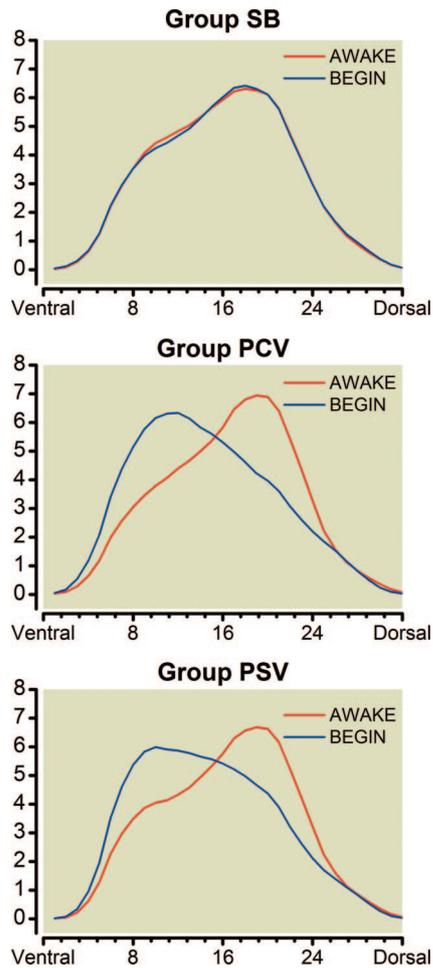


Fig. 6. Comparison of the ventilation distribution across the 32 rows of the tidal image electrical impedance tomography (EIT) (ventral = 1, dorsal = 32) before (AWAKE) and after (BEGIN) induction of general anesthesia. Groups pressure-controlled ventilation and pressure support ventilation depict a significant ventral shift of the ventilation, as indicated by the center of ventilation. COV = center of ventilation; PCV = pressure-controlled ventilation; PSV = pressure support ventilation; SB = spontaneous breathing.

the COV compared with the COV at baseline (AWAKE). MANCOVA confirmed that the COV values in the SB group were significantly different than those in the PCV and PSV groups (both $P < 0.01$) and that the PCV and PSV groups were not significantly different ($P = 0.77$).

An analysis of the four regions of interest (fig. 3, table 3) further illustrates the redistribution of ventilation during PCV and PSV. ROI 3 and 4 increase significantly (time-points BEGIN and END), whereas ROI 2 decreases ($P < 0.05$ for all differences; MANCOVA with a Sidak adjustment). The changes in ROI 1 were not significantly different across the groups.

Discussion

Our study shows that during general anesthesia with a LMA, both PCV and PSV induce a ventral redistribution of venti-

lation, whereas spontaneous breathing preserves the physiologic distribution of ventilation.

EIT is a relatively new imaging technology of lung function. It is noninvasive and radiation-free, and can easily be used at the bedside.⁵ EIT has been confirmed to be a viable tool for the assessment of regional lung ventilation at the bedside,⁹ and the accuracy of this method has been confirmed.^{10,11}

The output of the EIT device is a 32×32 matrix of impedance values that reflect a cross-section of the patient's thorax under the electrode belt. The impedances are presented in real-time with a resolution of 20 Hz. The absolute impedance values reflect not only the air content of the lung, but are influenced heavily by the characteristics of the surrounding tissue (*e.g.*, skin, moisture, fat content, bone, muscles). Subtracting the expiratory EIT matrix from the inspiratory EIT matrix un.masks the changes caused by the difference in air content during inspiration. A calibration of the EIT impedance values to calculate lung volume is technically possible, but it is not trivial during spontaneous breathing. Because we were comparing the tidal EIT images of the same individual at different time-points, a calibration to lung volume was not necessary to analyze the changes in ventilation distribution.

A parameter that quantifies the distribution of ventilation is the COV. The COV is a single number, has a good reproducibility,¹¹ and simplifies the comparison of EIT recordings. A value of 50 indicates that the ventilation is equally distributed between the ventral and dorsal halves of the thorax. Higher numbers indicate a shift toward the ventral region, and lower numbers indicate a shift toward the dorsum.

Another way to look at the tidal image is the analysis by ROI. The number calculated per ROI is the sum of the impedance changes in this ROI in relation to the sum of the impedance changes of the whole EIT image. For instance, a number of 30% indicates that 30% of the tidal variation takes part in this particular ROI.

The changes that we observed in the COV agree with the redistribution of ventilation as indicated by the changes in the tidal variation per ROI. Figure 6 illustrates the redistribution of ventilation caused by the induction of anesthesia and the beginning of PPV in groups PCV and PSV. Figure 3 shows how ventilation is redistributed from the dorsal to the ventral regions during the whole case in the PCV and PSV groups, and how the changes resolved after the patients awoke. Again, the distribution of ventilation remains unchanged throughout the study period in the SB group.

Our observation that the ventilation distribution is unchanged during general anesthesia with SB agrees with data from a volunteer study of ventilation and perfusion heterogeneity.¹² In their study, the authors compared ventilation and perfusion in healthy volunteers during SB in either awake or anesthetized state by using single-photon emission computed tomography. The authors found no significant redistribution of the ventilation when comparing the awake

Table 3. Percentage of Total Tidal Variation per Region of Interest

	AWAKE	BEGIN	END	PACU
Group SB at 4 Time-points				
ROI 4	11 (7–14)	9 (4–14)	12 (8–16)	10 (6–13)
ROI 3	39 (34–45)	38 (32–43)	42 (39–45)	40 (34–47)
ROI 2	43 (35–50)	45 (37–54)	39 (35–44)	41 (32–50)
ROI 1	8 (6–10)	8 (6–10)	7 (5–9)	9 (8–10)
Group PCV at 4 Time-points				
ROI 4	10 (7–13)	18 (14–21)	17 (14–20)	10 (6–13)
ROI 3	36 (30–42)	48 (44–51)	48 (45–50)	38 (32–44)
ROI 2	47 (38–56)	29 (25–32)	30 (27–32)	45 (35–55)
ROI 1	7 (6–7)	6 (5–7)	6 (5–7)	7 (6–9)
Group PSV at 4 Time-points				
ROI 4	11 (8–13)	18 (13–22)	15 (11–20)	10 (6–14)
ROI 3	38 (32–44)	46 (42–50)	44 (41–48)	42 (36–48)
ROI 2	44 (39–50)	31 (28–34)	35 (32–38)	40 (34–47)
ROI 1	7 (5–9)	6 (5–7)	6 (4–7)	8 (6–10)

The data are presented as the mean (95% CI). Region of interest (ROI) 1 is dorsal, whereas ROI 4 is ventral. ROI 2, 3, and 4 in the SB group are significantly different from those in groups PCV and PSV ($P < 0.05$; MANCOVA with Sidak adjustment).

AWAKE = before the induction of anesthesia; BEGIN = after the placement of the laryngeal mask airway; END = before the end of anesthesia; PACU = postanesthesia care unit; PCV = pressure controlled ventilation; PSV = pressure support ventilation; SB = spontaneous breathing.

with the anesthetized state. An interesting difference between their study and ours is the fact that Nyrén *et al.*¹² used a sevoflurane monoanesthesia with considerably higher sevoflurane concentrations ($2.9 \pm 1.4\%$), but no opioids. Consecutively, their volunteers had a lower tidal volume (292 ± 158) than our patients (table 2) and a higher respiratory rate (24 ± 5). Apparently, the preservation of ventilation distribution during SB does not depend on the tidal volume or respiratory rate.

A study by Keller *et al.* used a similar anesthetic regimen in a similar setting to compare the patient outcomes after PPV versus SB during general anesthesia with a LMA.¹³ Although they confirmed that both PPV and SB are equally safe in this setting, their data on tidal volume and respiratory rate differs from our data in the same way as the Nyren study does: the patients in their SB groups had lower tidal volumes and higher respiratory rates than our patients. Again, this can be attributed to the fact that Keller *et al.* used nitrous oxide, but no opioids during the case.

The redistribution of ventilation observed during PPV in our study can be explained by two pathophysiological concepts: the altered diaphragm movement during mechanical ventilation, and the positive pressure itself. In a thorough analysis of the diaphragm's movement during anesthesia, Reber *et al.* observed a cephalad movement of the diaphragm in anesthetized patients who were ventilated mechanically.¹⁴ During spontaneous breathing, the dependent part of the diaphragm moves the most, whereas during PPV, the ventilation lacks the physiologic movement of the diaphragm,^{15,16} reducing the ventilation in the dorsal parts of the lung. With the patient in prone position, the transdiaphragmal pressure caused by the abdominal content is highest in the dorsal parts.¹⁶ Therefore, during PPV, the air moves predomi-

nantly to the ventral regions of the lung, since the regional compliance in the ventral region is higher.

Although these mechanisms explain the difference between SB and PCV, it was surprising to find no difference between PCV and PSV. We expected to see less redistribution of ventilation during PSV than during PCV, because the movement of the diaphragm was not suppressed. However, when we analyze the distribution of ventilation, we examine the static state at the end of the inspiration. At the peak of the inspiration, the pressure and the tidal volume was the same in both PSV and PCV groups (table 2), and it appears logical that we would see the same extent of redistribution in both groups. During PSV, the movement of the diaphragm takes place at the beginning of the respiratory cycle, so a detailed analysis of the ventilation dynamics throughout the whole cycle might reveal a difference between PCV and PSV.

Another possible explanation for the lack of a difference between PCV and PSV in our study is the low PSV trigger setting (2.0 l/min). A sensitive trigger setting results in a very short interval between the start of the patients' own inhalation effort and the beginning of the positive-pressure support provided by the respirator. Because both the PCV and PSV groups had identical tidal volumes and peak pressures (table 2), it is likely that the proportion of spontaneous breathing in the PSV group was small. This would have reduced the beneficial effects of spontaneous breathing.

Limitations

Even though the patients in our study were randomly assigned to the three groups, we did observe some imbalances regarding the addition of regional anesthesia. Even though the difference is not statistically significant, five patients in group PSV had regional anesthesia, whereas in groups SB and PCV, respectively, only one patient received a peripheral

nerve block (table 1). In addition, the total dose of sufentanil in group PSV was higher than in the other groups (27.3 μg vs. 19.7 μg and 20.3 μg). However, the mean respiratory rate in group PSV was not different from the respiratory rate in group SB, indicating that the effect of the sufentanil doses was probably similar in both groups.

Another interesting difference is the fact that in group SB, the variation of the tidal volumes was considerably higher than in groups PCV and PSV. This is because of the study protocol that defines the target tidal volume for patients in groups PCV and PSV as 6–8 ml/kg, whereas the tidal volume in group SB reflects inter- and intraindividual variability. With our study protocol, we cannot determine the relationship between tidal volume and redistribution of ventilation. However, we did see a significant difference in the COV in groups SB versus PSV/PCV not only at time-point BEGIN but also at time-point END, where the means of the tidal volume are much closer together than at time-point BEGIN. In addition, the COV in group SB did not change during the whole study period, with and without anesthesia. It is conceivable that the redistribution of the ventilation is more strongly related to the PPV than to the difference in tidal volumes.

In our study we decided not to use PEEP in the PCV and PSV groups, because it would have introduced another confounder that makes comparisons between PCV/PSV and SB difficult. Use of PEEP during PPV increases the ventilation in the dorsal parts of the lung and reduces the amount of redistribution caused by induction of anesthesia.¹⁷ It would be interesting to see how PPV with and without PEEP compares with SB with or without continuous positive airway pressure.

Generalizability

The study protocol was closely modeled after the actual clinical practice in our hospital, and all three types of ventilation during general anesthesia (SB, PCV, and PSV) are used in our department. The results of our study could aid anesthesia-providers in the future when they are planning the mode of ventilation for a case.

Even though the difference in the COV was substantial in our study, it appears to have little clinical relevance to healthy patients. The possibility of clinical relevance for patients with risk factors for atelectasis and postoperative hypoxemia needs to be addressed in future studies.

Conclusion

According to the changes in COV in our study, both PCV and PSV induce a redistribution of ventilation toward the ventral region. Spontaneous breathing prevents this redistribution.

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