ANESTHESIOLOGY

Pressure Support *versus* Spontaneous Ventilation during Anesthetic Emergence—Effect on Postoperative Atelectasis: A Randomized Controlled Trial

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EDITOR'S PERSPECTIVE

What We Already Know about This Topic

- Pressure support ventilation modalities are now standard on newer anesthesia machines and are commonly used during emergence from anesthesia
- Their benefits in preventing postoperative atelectasis have not been well studied

What This Article Tells Us That Is New

- A randomized trial in patients undergoing laparoscopic colectomy or robot-assisted prostatectomy compared pressure support ventilation to spontaneous ventilation with intermittent manual assistance during anesthetic emergence
- The outcome was atelectasis in the postanesthesia recovery unit, using lung ultrasound
- The incidence of atelectasis was significantly lower and the Pa_{O_2} was significantly higher with pressure support ventilation; however, in the 48-h postoperative observation period, the incidence of oxygen saturation measured by pulse oximetry less than 92% was not different between groups

ABSTRACT

Background: Despite previous reports suggesting that pressure support ventilation facilitates weaning from mechanical ventilation in the intensive care unit, few studies have assessed its effects on recovery from anesthesia. The authors hypothesized that pressure support ventilation during emergence from anesthesia reduces postoperative atelectasis in patients undergoing laparoscopic surgery using the Trendelenburg position.

Methods: In this randomized controlled double-blinded trial, adult patients undergoing laparoscopic colectomy or robot-assisted prostatectomy were assigned to either the pressure support (n = 50) or the control group (n = 50). During emergence (from the end of surgery to extubation), pressure support ventilation was used in the pressure support group *versus* intermittent manual assistance in the control group. The primary outcome was the incidence of atelectasis diagnosed by lung ultrasonography at the postanesthesia care unit (PACU). The secondary outcomes were Pa_{O_2} at PACU and oxygen saturation measured by pulse oximetry less than 92% during 48 h postoperatively.

Results: Ninety-seven patients were included in the analysis. The duration of emergence was 9 min and 8 min in the pressure support and control groups, respectively. The incidence of atelectasis at PACU was lower in the pressure support group compared to that in the control group (pressure support *vs.* control, 16 of 48 [33%] *vs.* 28 of 49 [57%]; risk ratio, 0.58; 95% CI, 0.35 to 0.91; $P = 0.024$). In the PACU, Pa_{O_2} in the pressure support group was higher than that in the control group (92 ± 26 mmHg *vs.* 83 ± 13 mmHg; $P = 0.034$). The incidence of oxygen saturation measured by pulse oximetry less than 92% during 48 h postoperatively was not different between the groups (9 of 48 [19%] *vs.* 11 of 49 [22%]; $P = 0.653$). There were no adverse events related to the study protocol.

Conclusions: The incidence of postoperative atelectasis was lower in patients undergoing either laparoscopic colectomy or robot-assisted prostatectomy who received pressure support ventilation during emergence from general anesthesia compared to those receiving intermittent manual assistance.

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Although there have been many studies regarding ventilatory techniques to reduce postoperative pulmonary complications,[1–4] only a few studies have focused on the period of recovery from anesthesia. The benefits obtained from the protective ventilation techniques may be lost during this emergence process. Whalen *et al.*[5] found that recruitment maneuver and the application of positive end-expiratory pressure (PEEP) improved intraoperative oxygenation, but the effect dissipated promptly after extubation. Many studies have observed the development of

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atelectasis during the emergence period.[6,7] Furthermore, it is estimated that the emergence period contributes to approximately 39% of the total amount of postoperative atelectasis.[7]

Currently, we allow patients to breathe spontaneously and assist their respiration intermittently during the transition from controlled ventilation to spontaneous respiration while assessing whether the patients have enough power to breathe without assistance. However, patients who are spontaneously breathing remain under the influence of residual anesthetic agents and neuromuscular blockers and may not have restored their functional residual capacity,[8,9] subsequently developing atelectasis.[10] In addition, pain-induced respiratory restriction or respiratory muscle fatigue during spontaneous respiration may increase the risk of atelectasis. Postoperative atelectasis is one of the most common pulmonary complications noted in surgical patients,[10,11] and a fair majority of studies have suggested that postoperative atelectasis is harmful. It increases the risk of hypoxemia and forms the pathophysiologic basis for other postoperative pulmonary complications.[12–14] Atelectasis can last for several days after surgery,[15] impairing respiratory function, and ultimately delaying patient discharge.[16,17]

Pressure support ventilation is widely used for weaning from the ventilator in the intensive care unit (ICU) and is recently available in anesthesia machines. Pressure support ventilation applies a fixed amount of pressure the physician selects to the patients throughout each breath to augment their own respiration and is one of the most comfortable ventilation modes for patients. In these aspects, pressure support ventilation during recovery from anesthesia may reduce postoperative atelectasis compared to spontaneous respiration with intermittent manual assistance. To date, few studies have assessed the effect of pressure support ventilation on postoperative atelectasis.

Therefore, we compared the incidence of postoperative atelectasis in patients who received pressure support ventilation with that in patients who received spontaneous respiration with intermittent manual assistance, postlaparoscopic colectomy, or robot-assisted laparoscopic prostatectomy. In recent times, laparoscopic colectomy and robot-assisted laparoscopic prostatectomy have gained wide acceptance for their better or noninferior outcomes and more enhanced recovery compared to open surgery. However, these procedures are associated with a higher risk of postoperative atelectasis due to the high intra-abdominal pressure and Trendelenburg position, which pushes the diaphragm upward and subsequently results in the collapse of the alveoli.[9,18]

We hypothesized that pressure support ventilation reduces the incidence of postoperative atelectasis compared to spontaneous respiration with intermittent manual assistance in patients undergoing laparoscopic colectomy or robot-assisted laparoscopic prostatectomy.

Materials and Methods

Study Design

This was a single-center, randomized, controlled, patient- and evaluator-blinded trial with a two-arm parallel design to assess the possible superiority of pressure support ventilation. The study protocol was approved by the Samsung Medical Center (Seoul, Korea) Institutional Review Board (approval No. SMC 2020-02-117-002; date of approval, April 16, 2020). It was prospectively registered with the Korean Clinical Research Information Service (registration No. KCT0004944; principal investigator, Hyun Joo Ahn; date of registration, April 20, 2020; https://cris.nih.go.kr). Our study was conducted in accordance with the ethical principles of the 1964 Declaration of Helsinki and its later amendments and was performed at Samsung Medical Center. The trial was conducted in accordance with the original protocol. Written informed consent was obtained from all participants.

Participants

Between April 2020 and September 2020, 108 patients scheduled for elective laparoscopic colectomy or robot-assisted laparoscopic prostatectomy were screened for inclusion and contacted by primary investigators a day before the surgery to obtain written informed consent.

The inclusion criteria were age 20 yr or greater and American Society of Anesthesiologists (ASA; Schaumburg, Illinois) Physical Status I to III. The exclusion criteria were a body mass index 30 kg/m^2 or greater, pregnancy, underlying lung disease, moderate or severe obstruction observed on pulmonary function test, previous lung surgery, pneumothorax, pulmonary tuberculosis, pleural effusion, expectation of difficult intubation, and patient's refusal. The dropout criteria included the withdrawal of consent, change of surgical plan to open surgery, intraoperative blood loss greater than 400 ml, or unstable hemodynamics, which is defined as vital signs not maintained within the target range (20% of the baseline values) despite administration of fluid and vasopressors.

Randomization

Randomization was performed using a computer-generated random numbers table with a fixed block size of four and a 1:1 ratio. Allocation was sequentially numbered and sealed in opaque envelopes by the corresponding author. The attending anesthesiologists opened the envelopes 10 min before commencing the emergence procedure.

Blinding Method

This was a randomized, controlled, patient- and evaluator-blinded trial. The patients, surgeons, sonographers, and staff of the postanesthesia care unit (PACU) were blinded to patient

group allocation. Attending anesthesiologists were not blinded to the group allocation. However, they were not aware of the aim of the study. Attending anesthesiologists were not involved in lung ultrasound examination or data analysis.

Anesthesia and Monitoring

A chest x-ray was performed 1 day before operation to exclude preexisting lung pathologies including atelectasis. No patient received sedating premedication. The induction and maintenance of anesthesia were standardized and identical for all patients. After standard monitoring, IV propofol (2.0 to 2.5 mg/kg) and rocuronium (1.0 mg/kg) were administered for induction, and maintenance was achieved using 1.0 to 2.0 minimum alveolar concentration of sevoflurane and IV remifentanil (0.05 to 0.2 $\mu g \cdot kg^{-1} \cdot min^{-1}$). Rocuronium was continuously infused at a rate of 0.3 to 0.8 $mg \cdot kg^{-1} \cdot h^{-1}$ under train-of-four monitoring (target train-of-four count, 1/4) and stopped approximately 40 min before the end of surgery. An additional bolus was allowed if necessary.

Preoxygenation was performed for 2 min (O_2 4 l/min) in the supine position. After the loss of spontaneous breathing, the patients were bag mask–ventilated with a fraction of inspired oxygen (F_{IO_2}) of 0.8. Endotracheal intubation was performed 4 min after the start of preoxygenation. After intubation, an arterial catheter was placed in the radial artery for blood gas sampling and invasive blood pressure monitoring.

Mechanical ventilation was maintained using an anesthesia machine (Carestation 650; Datex-Ohmeda, Inc.; USA) in a volume-controlled mode. The ventilatory settings in both groups were F_{IO_2}, 0.4; tidal volume (V_T), 8 ml/kg of predicted body weight; inspiratory to expiratory ratio, 1:2; and PEEP, 5 cm H_2O. The respiratory rate (RR) was set to 12 breaths/min and further adjusted to maintain end-tidal carbon dioxide pressure between 33 and 45 mmHg. The recruitment maneuver was not used.

The patients were placed in the lithotomy with Trendelenburg positioning during surgery (approximately 30 degrees head-down position in both laparoscopic colectomy and robot-assisted laparoscopic prostatectomy). Pneumoperitoneum was established with carbon dioxide, and intra-abdominal pressure in all patients was maintained between 12 and 15 mmHg during abdominal insufflation.

Blood pressure was maintained within 20% of the baseline values. Phenylephrine, ephedrine, or nicardipine was administered as required to maintain mean arterial blood pressure within this range. If the heart rate was less than 40/min, IV atropine 0.5 mg was administered. Lactated Ringer's solution was used as maintenance fluid and infused at a rate of 4 to 6 $ml \cdot kg^{-1} \cdot h^{-1}$. If intraoperative bleeding occurred, crystalloid solution was administered to replace blood loss. IV hydromorphone 0.01 mg/kg and paracetamol 1 g were administered 5 min before the end of surgery, and IV patient-controlled analgesia was applied to all patients (bolus dose, fentanyl 10 μg; basal infusion dose, fentanyl 10 μg/h). In the PACU, patients who complained of moderate pain greater than 4, measured using a numeric rating scale (0 = no pain, 10 = worst pain), received rescue opioids (IV hydromorphone 0.01 mg/kg) until the numeric rating scale was 4 or lower.

Study Protocol

The duration of anesthesia emergence was the duration from the end of surgery to extubation. The designated ventilatory support method was maintained during emergence in both groups. At the end of surgery, sevoflurane was ceased, and the attending anesthesiologist began the recovery protocol; the pressure support group received pressure support ventilation. The initial pressure support ventilation setting was a driving pressure of 5 cm H_2O, PEEP of 5 cm H_2O, and safety backup ventilation of 12 breaths/min (safety backup ventilation setting, V_T, 8 ml/kg of predicted body weight; and PEEP, 5 cm H_2O). The flow trigger and end of breath were set at 2 l/min and 30% of peak flow, respectively. Support amount and safety backup ventilation were adjusted according to the patient's response to meet the target V_T of 7 to 8 ml/kg and RR of 10 to16 breaths/min and decreased gradually as the patient restored his or her own V_T and RR. Ventilatory support was stopped when the patient showed adequate V_T (greater than 6 ml/kg) and RR (10 breaths/min or greater) without ventilatory support. However, PEEP (5 cm H_2O) was maintained until extubation in the pressure support group. In the control group, the emergence process was led by the discretion of the attending anesthesiologist. The basic strategy was to allow the patient to breathe spontaneously and only help respiration if necessary, with intermittent manual assistance. Both groups received fresh gas flow at 4 l/min and F_{IO_2} of 0.4 during emergence from anesthesia. In patients who developed oxygen saturation measured by pulse oximetry (S_{PO_2}) less than 90% after extubation, rescue mask ventilation was applied with F_{IO_2} of 1.0.

A train-of-four of peripheral nerve stimulator was monitored using the ulnar nerve throughout recovery, and neuromuscular blockade was reversed with 0.2 mg/kg pyridostigmine and 0.008 mg/kg glycopyrrolate IV when the train-of-four counts were 3 or greater or sugammadex 2 to 4 mg/kg IV when the train-of-four counts were 2 or less. In both groups, extubation was performed when the patient met the following criteria: obeys commands such as eye-opening or hand-grip, V_T greater than 250 ml, end-tidal carbon dioxide less than 45 mmHg, RR 10 to 20 breaths/min, and a train-of-four ratio greater than 0.9. After extubation, all patients were transferred to the PACU without oxygen supplementation.

Lung Ultrasonography and Scoring System

All patients were evaluated using lung ultrasonography 30 min after their PACU arrival. Lung ultrasonography was

performed using a Vivid S70N (GE Vingmed Ultrasound AS; Norway) with an 11-MHz linear transducer and real-time B-mode. Inspection of each lung was performed at 12 lung sections (each hemithorax was divided into six sections), determined by parasternal, anterior, and posterior axillary lines (vertically) and by nipple and diaphragm lines (horizontally) as landmarks in the supine position, similar to previous studies.[19,20] The 12 lung sections were scanned sequentially from right to left, cranial to caudal, and anterior to posterior. Posterior fields were examined while an assistant held the patient in the 45-degree lateral position. The examination was carried out longitudinally on the acoustic windows of the intercostal spaces, and the ultrasound probe was applied perpendicular to the pleura, with a conventional gel between the transducer and skin. The following signs were assessed: the lung "sliding" sign, A-lines, B-lines, lung pulse, and air bronchograms. Postoperative atelectasis was scored between 0 and 4, according to the degree of de-aeration: score 0: normal lung, or one or two well-spaced vertical lines per intercostal space; score 1: three or more well-spaced vertical lines per intercostal space (B-lines) or juxtapleural consolidation with normal pleural line; score 2: loss of A-line with multiple juxtapleural consolidations and irregular pleural lines; score 3: loss of lung sliding and appearance of lung pulse; score 4: consolidation exceeding 1 cm × 2 cm with or without air bronchogram (fig. 1; Supplemental Digital Content 1, http://links.lww.com/ALN/C690).[21] Vertical lines originating from the consolidation were not scored as 1. Transient loss of A-line by B-lines was not scored as 2. The atelectasis score was calculated by adding up the scores of the 12 sections, and a higher score indicated a more severe loss of aeration. We defined anesthesia-induced atelectasis to be clinically significant if more than three sections (approximately 25% of total lung surface) showed any sign of atelectasis (atelectasis score 1 or greater). Ultrasonography was performed by two anesthesiologists (P. Tanatporn and H. Yeo) with more than 1 yr of experience, and they had performed more than 100 cases. They were blinded to the group assignment. All measurements were conducted during deep spontaneous respiration. All clips were stored and interpreted by the consensus read of the two sonographers.

Arterial Blood Gases and Oxygenation

Arterial blood gas analysis was performed immediately after arrival at the PACU without oxygen supplementation. In both groups, patients were encouraged to breathe deeply without oxygen during the PACU and ward stay. Oxygen *via* nasal prong at 3 l/min was provided to patients when Spo_2 could not be maintained at 92% or greater.

Study Outcomes and Measurements

The primary outcome was the incidence of postoperative atelectasis diagnosed by lung ultrasonography at PACU. The secondary endpoints were Pao_2 at PACU and incidence of Spo_2 less than 92% during 48 h postoperatively.

We recorded the patients who needed supplemental oxygen or mechanical ventilatory support during 48 h postoperatively. The patient's vital signs, temperature, and respiratory symptoms (cough, sputum, and sore throat) were evaluated every 8 h during 48 h postoperatively. Pulmonary complications, adverse cardiac events, postoperative acute kidney injury, delirium until discharge, and postoperative transfusion were also recorded. The definitions of each complication are presented in Supplemental Digital Content 2 (http://links.lww.com/ALN/C691).

Statistical Analysis

The incidence of postoperative atelectasis was assumed to be 53% based on previous studies.[10,22] Our hypothesis was that pressure support ventilation reduces the incidence of postoperative atelectasis by 30%. Thus, we expected an incidence of postoperative atelectasis of 37% in the pressure support group. With a significance level of 0.05 (two-tailed) and a power of 80%, 88 study subjects were required. Considering a dropout rate of 15%, we included 100 patients in this study. Continuous variables are presented as the mean ± SD or median (interquartile range). Categorical variables are described as counts (%). The normal distribution of data was evaluated using the Shapiro–Wilk test. CIs for nonnormally distributed variables were calculated using the Hodges–Lehmann estimator.

The primary outcome (incidence of atelectasis) was analyzed using the chi-square test. The secondary outcomes (Pao_2, event of Spo_2 less than 92%) were evaluated using an independent *t* test and chi-square test. Effect sizes were also evaluated by computing risk ratio with 95% CIs for binary outcomes and calculating Cohen's *d* with pooled SD with 95% CIs for continuous outcomes. As a *post hoc* sensitivity analysis, we used a logistic regression model with a simultaneous entering of age, body mass index, cardiovascular disease, ASA Physical Status, and operation duration. All *P* values were two-sided, and $P < 0.05$ was considered significant. All data were analyzed using MedCalc 14.12.0 (MedCalc Software Ltd., Belgium).

Results

Enrollment ceased when the target sample size was obtained. In total, 108 patients were assessed for eligibility between April 2020 and September 2020. Eight patients were excluded due to their refusal (n = 7) or cancelation of surgery (n = 1). The remaining 100 patients were randomized and received their allocated treatment. However, three patients dropped out. One in the control group dropped out due to a technical problem with the ultrasound machine. Two in the pressure support group dropped out due to postoperative pneumothorax and open conversion. (Postoperative pneumothorax in the pressure

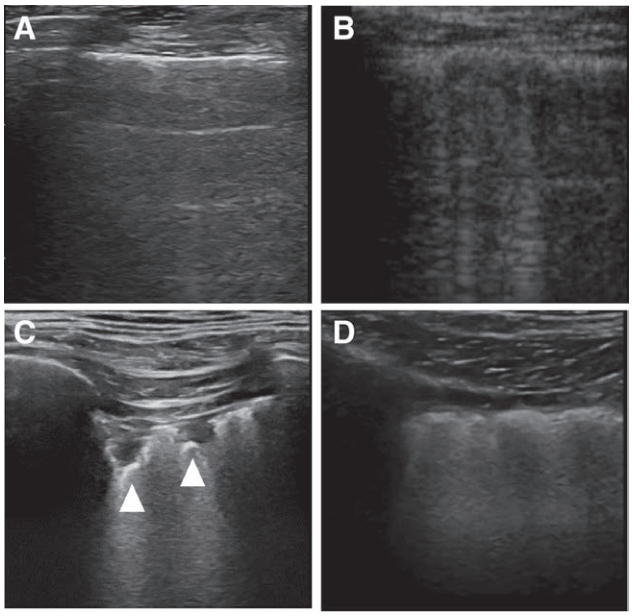

Fig. 1. Lung sonographic signs associated with atelectasis. (*A*) Score 0: normal lung. Pleura is thin and A-lines are apparent. One or two well-spaced lines per intercostal space are allowed. (*B*) Score 1: more than three well-spaced vertical lines per intercostal space (B-lines). (*C*) Score 1: juxtapleural consolidation (*arrows*) with normal pleural line. Juxtapleural consolidation is caused by a loss of lung aeration. It commonly arises from the pleural line. (*D*) Score 2: loss of A-line with multiple juxtapleural consolidations and irregular pleural lines are seen. Score 3 (loss of lung sliding and appearance of lung pulse; Supplemental Digital Content 1, http://links.lww.com/ALN/C690) and score 4 (large consolidation, no occurrence in our study) are not presented here.

support group was developed from peritoneal carbon dioxide insufflation during surgery. The peritoneal gas traveled to the mediastinum and then pleura through the esophageal hiatus. The amount of pleural air was minimal, but the ultrasonographic diagnosis was impaired.) Finally, 49 and 48 patients in the control and pressure support groups, respectively, were analyzed (fig. 2). There were no missing data for these 97 patients for subsequent analyses. There were no adverse events related to study protocol, including immediate postextubation respiratory failure, in both groups.

The baseline characteristics of the participants, operative, and ventilatory data between the two groups are presented in table 1. The mean duration of emergence (from the end of surgery to extubation) was 8 ± 3 min and 9 ± 4 min in the control and pressure support groups, respectively. We defined anesthesia-induced atelectasis to be clinically significant if more than three sections (approximately 25% of total lung surface) showed any sign of atelectasis. Based on this definition, the incidence of postoperative atelectasis diagnosed by lung ultrasonography was 28 of 49 (57%) and 16 of 48 (33%) in the control and pressure support groups, respectively (risk ratio, 0.58; 95% CI, 0.35 to 0.91; $P = 0.024$). The area of atelectasis is shown in figure 3 and Supplemental Digital Content 3 (http://links.lww.com/ALN/C692). Atelectasis was most common in the dependent area in both lungs, and the left lower lobe showed the highest incidence.

Various phenotypes of atelectasis are shown in table 2. The most common atelectasis findings were loss of A-lines with multiple juxtapleural consolidations and irregular pleural lines (score 2, n = 64), followed by multiple B-lines (score 1, n = 51), juxtapleural consolidation with normal pleural line (score 1, n = 19), and loss of sliding and appearance of lung pulse (score 3, n = 2). No patient showed score 4 consolidation. The sum of score was 5 (2 to 8) and 3 (1 to 6) in the control and pressure support groups, respectively (median [interquartile range]; $P = 0.093$).

Spo_2 at extubation was 100 (100 to 100) *vs.* 100 (100 to 100) in the control and pressure support groups, respectively (median [interquartile range]; $P = 0.715$). The duration of PACU stay was 65 (56 to 79) min *vs.* 68 (60 to 75) min in the control and pressure support groups, respectively (median [interquartile range]; $P = 0.318$).

The percentage of patients who showed Spo_2 less than 92% during the PACU stay was 26% and 23% ($P = 0.680$) in the control and pressure support groups, respectively. Pao_2 at PACU was higher in the pressure support group (83 ± 13 mmHg *vs.* 92 ± 26 mmHg; $P = 0.034$).

After transfer to the ward, the incidence of Spo_2 less than 92% during the postoperative 48 h was 22% and 19% in the control and pressure support groups, respectively

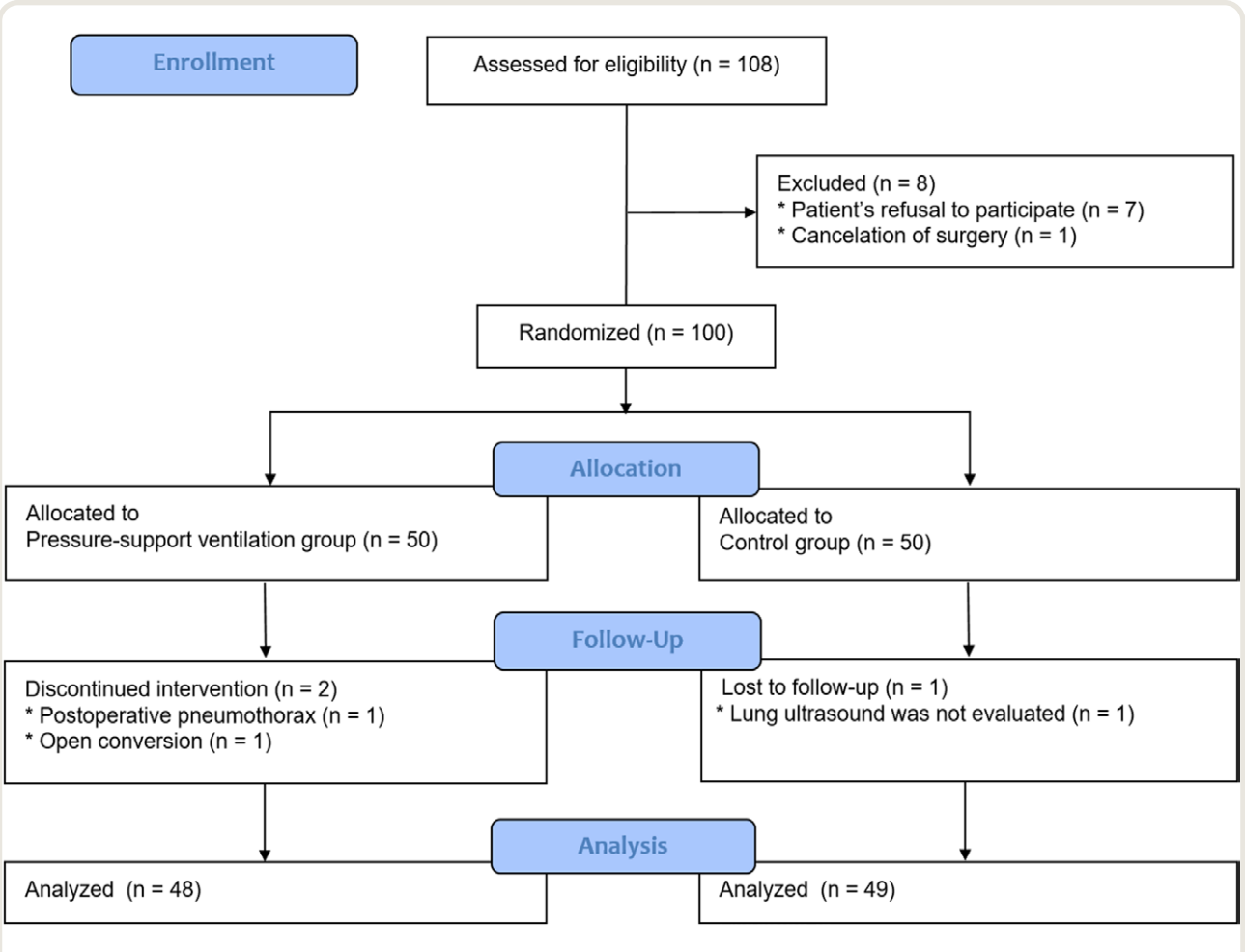

Fig. 2. Consolidated Standards of Reporting Trials flow diagram of the study.

(P = 0.653; table 3). Twenty patients (22% *vs.* 19%, control group *vs.* pressure support group; P = 0.653) received supplemental oxygen therapy to maintain Spo_2 of 92% or greater. However, none received ventilatory support during the postoperative 48 h. Other major postoperative complications did not differ between the groups (Supplemental Digital Content 2, http://links.lww.com/ALN/C691).

The beneficial effect of pressure support ventilation on postoperative atelectasis diagnosed by lung ultrasonography was not changed after *post hoc* sensitivity analysis using variables that may influence postoperative atelectasis (age, body mass index, cardiovascular disease, ASA Physical Status, and operation duration; table 4).

Discussion

In the current study, the pressure support group showed a lower incidence of postoperative atelectasis and higher oxygenation compared to the control group in laparoscopic colectomy and robot-assisted laparoscopic prostatectomy.

Pressure support ventilation is widely utilized in ventilator weaning of patients in the ICU,[23–26] and the latest American Thoracic Society (New York, New York) guideline recommends pressure support ventilation for successful weaning.[27] However, few studies evaluated it as a ventilatory mode of emergence from anesthesia.

Due to the lack of reports in surgical patients, anesthesiologists may be concerned that some patients who breathe comfortably with pressure support ventilation could develop respiratory failure immediately after extubation, or others may argue that we need to watch our patients' spontaneous breathing to predict the patients' physiologic conditions after extubation.[25] Pellegrini *et al.*[28] demonstrated that high continuous positive airway pressure reduced respiratory drive and the contractile activity of the diaphragm in patients in the ICU. In our study, pressure support ventilation was not associated with postextubation hypoxia or extubation failure; rather, it contributed to the lower incidence of postoperative atelectasis and higher oxygenation. Pressure support ventilation reduced the risk

Table 1. Baseline Characteristics, Operative Data, and Ventilatory Data of Participants

Variables	Control (n = 49)	Pressure Support (n = 48)
Age, yr	64 ± 9	62 ± 10
Sex, male	38 (78)	31 (65)
Body mass index, kg/m^2	25 ± 3	24 ± 3
ASA Physical Status ≥ III	5 (10)	4 (8)
Smoking*	2 (4)	2 (4)
Comorbid condition		
Hypertension	23 (47)	17 (35)
Diabetes mellitus	11 (22)	10 (21)
Cardiovascular diseases†	4 (8)	3 (6)
Difficult intubation‡	6 (12)	3 (6)
Duration of surgery, min	157 ± 40	172 ± 54
Type of surgery		
Laparoscopic colectomy	22 (45)	29 (60)
Robot-assisted laparoscopic prostatectomy	27 (55)	19 (40)
Intraoperative fluid infusion, ml/min	4.5 ± 1.2	4.9 ± 2.1
Estimated blood loss, ml	118 ± 92	134 ± 111
Mean arterial pressure, mmHg	85 ± 8	88 ± 12
Heart rate, beats/min	66 ± 11	67 ± 10
Peak airway pressure,§ cm H_2O	25 [23–27]	24 [22–26]
Plateau airway pressure,§ cm H_2O	20 [18–22]	19 [17–21]
Driving pressure,§ cm H_2O	15 [13–17]	14 [12–16]
Tidal volume per predicted body weight,§ ml/kg	6 [6–7]	7 [6–8]
Respiratory rate,§ breaths/min	13 [12–14]	13 [12–14]
Static compliance,§ ml/cm H_2O	29 [25–34]	35 [31–42]
End-tidal carbon dioxide pressure,§ mmHg	36 ± 2	37 ± 2
Intraoperative Pao_2,§ mmHg	255 ± 113	222 ± 98
Use of sugammadex before extubation	22 (45)	16 (33)
Event of Spo_2 < 92% during operation	3 (6)	2 (4)
Duration of emergence, min	8 ± 3	9 ± 4
Opioid consumption during the PACU stay, fentanyl equivalent, μg	20 [0–35]	20 [0–30]

Data are presented as n (%), mean ± SD, or median [interquartile range].

*Smoking included current smokers and ex-smokers within 1 month. †Cardiovascular diseases included angina pectoris and myocardial infarction. ‡Difficult intubation included more than two attempts of intubation with direct laryngoscope or cases were needed video-assisted intubation devices. §Respiratory values were measured during 30-degree Trendelenburg position and abdominal insufflation.

ASA, American Society of Anesthesiologists; PACU, postanesthesia care unit, Spo_2, oxygen saturation measured by pulse oximetry.

of postoperative atelectasis diagnosed by current method ultrasonography regardless of patient's age, body mass index, cardiovascular disease, ASA Physical Status, or operation duration (table 4). There is no evidence that a short duration of pressure support ventilation would have a significant impact on respiratory muscle dysfunction.

The possible mechanisms for how pressure support ventilation shows a lower incidence of postoperative atelectasis are as follows. First, in inspiratory pressure support, driving pressure helps lung expansion during inspiration with reduced work of breathing by as much as 30 to 40%.[25,29,30] Second, PEEP increases the end-expiratory lung volume and counteracts airway closure with a dominant effect in the dependent lung region, which is sufficient to prevent or reverse atelectasis in healthy patients undergoing surgery.[31]

To date, the use of low FIO_2 has been the most commonly suggested technique to decrease atelectasis during recovery from anesthesia.[6,7,32–36] An FIO_2 of 0.3 to 0.4 before extubation resulted in reduced incidence of postoperative atelectasis compared to an FIO_2 of 1.0,[6] and the same was observed in patients with chronic obstructive pulmonary disease (COPD)[35] and obese patients undergoing laparoscopic surgery.[34] However, some studies were unable to show the protective effect of low FIO_2 against atelectasis.[33] In the current study, the incidence of postoperative atelectasis was as high as 57% in laparoscopic surgery, even though low FIO_2 (0.4) was administered. Pressure support ventilation reduced the incidence of atelectasis by 42% in these patients. Our finding suggests that pressure support ventilation is another armamentarium against postoperative atelectasis.

Most of the previous studies which compared ventilatory techniques used computed tomography to diagnose immediate postoperative atelectasis.[6,7,33,37] In most studies, a single-sliced transverse scan was performed approximately 5 mm above the right dome of diaphragm 15 to 60 min postoperatively.[7] The approximate measurement time was similar to that in our study, but a single cut scan may not reflect the lesions in other lung areas. Lung ultrasonography is a fast, simple, noninvasive, and radiation-free technique in which the entire lung area and dynamic changes during respiration can be examined.[38–40] The quantitative association between lung ultrasonography scores of aeration and the volumetric data of atelectasis observed on thoracic computed tomography showed that lung ultrasonography had reliable performance in the diagnosis of postoperative atelectasis, with a sensitivity of 88%, a specificity of 92%, and a diagnostic accuracy of 91%.[41] A recent meta-analysis demonstrated that lung ultrasonography had a higher diagnostic ability compared to chest x-ray film for lung consolidation/collapse (lung ultrasonography: sensitivity of 92% and specificity of 92%; x-ray film: sensitivity of 53% and specificity of 78%).[42]

However, the atelectasis scoring system using ultrasonography has not yet been standardized.[20,21,43,44] Originally, lung ultrasound score was developed for ICU patients to assess the severity of pulmonary disease, including B-lines 3 or greater (score 1), multiple coalescent B-lines (score 2), and consolidation (score 3).[45] Several studies have adopted the same system to assess postoperative atelectasis.[46] B-lines are hyperechoic lines produced by the interaction between alveolar air and interstitial fluid and can be seen in histologically normal lungs which are deflated to a critical level of density (greater than 0.45 g/ml).[43] However, coalescent B-lines, which are common in pulmonary edema and acute respiratory distress syndrome, were not observed in our study, and they were probably not in previous studies either, according to lung ultrasound photos presented.[43,44] We found that B-lines in postoperative atelectasis were mostly pseudo B-lines. They were not long enough to reach 8 to

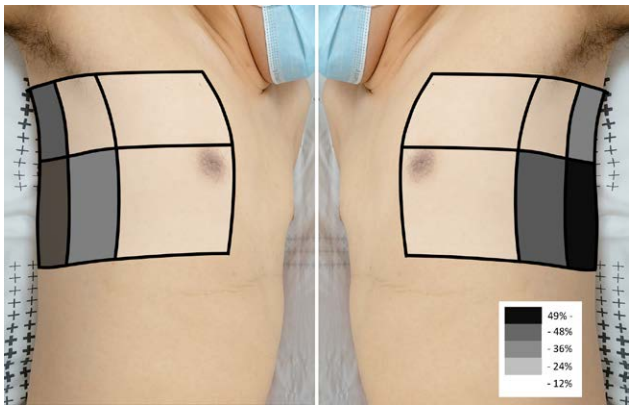

Fig. 3. The regional distribution of atelectasis. *Darker colors* indicate higher incidence. Most atelectasis occurred in the dependent area. The left lower lobe showed the highest incidence.

Table 2. Postoperative Atelectasis Outcomes in the Postanesthesia Care Unit

Variables	Control (n = 49)	Pressure Support (n = 48)	Effect Estimate (95% CI)	*P* Value
Atelectasis diagnosed by lung ultrasonography	28 (57)	16 (33)	0.58 (0.35 to 0.91)*	0.024
Atelectasis score	5 [2 to 8]	3 [1 to 6]	0.35 (–0.06 to 0.72)†	0.093
Major findings of atelectasis				
B-lines ≥ 3	25 (51)	26 (54)	1.06 (0.72 to 1.57)*	0.756
Juxtapleural consolidation with normal pleural line	12 (25)	7 (15)	0.60 (0.24 to 1.35)*	0.228
Loss of A-line with multiple juxtapleural consolidations and irregular pleural lines	35 (71)	29 (60)	0.85 (0.62 to 1.13)*	0.257
Loss of lung sliding and appearance of lung pulse	0 (0)	2 (4)	Not reported‡	0.149
Tissue-like change with or without airbronchogram	0	0		

Data are presented as n (%) or median [interquartile range].

*Effect estimate is risk ratio (two-sided 95% CI) by Wald likelihood ratio approximation test and chi-square hypothesis tests. †Effect estimate is calculated by Cohen's *d* with pooled SD. ‡Not reported because there were no patients in the control group.

Table 3. Secondary and Other Outcomes of Participants

Variables	Control (n = 49)	Pressure Support (n = 48)	*P* Value
Pa_{O_2} measured in the PACU, mmHg	83 ± 13	92 ± 26	0.034
Events of Sp_{O_2} < 92% during the PACU stay	13 (26)	11(23)	0.680
After discharge to ward			
Events of Sp_{O_2} < 92% 48 h postoperatively	11 (22)	9 (19)	0.653
Patients who needed supplemental oxygen 48 h postoperatively	11 (22)	9 (19)	0.653
Patients who needed mechanical ventilation support	0	0	
Fever (≥ 37.5ºC) 48 h postoperatively	6 (12)	9 (19)	0.376
Postoperative hospital stay, day	7 [6–8]	7 [6–8]	0.515

Data are presented as mean ± SD, n (%), or median [interquartile range].

PACU, postanesthesia care unit; Sp_{O_2}, oxygen saturation measured by pulse oximetry.

10 cm in length and usually occur below the subpleural consolidation, not below the pleura.[44] Acosta *et al.*,[20] who first examined the capability of lung ultrasound to diagnose postoperative atelectasis, and others[41] also reported findings similar to ours. Therefore, Monastesse *et al.*[21] proposed modified lung ultrasound scores that emphasize subpleural consolidation for postoperative atelectasis. Other researchers combined B-lines (score 0 to 3) and subpleural

Table 4. *Post Hoc* Sensitivity Analysis Using Multiple Logistic Regression

Variable	Odds Ratio	95% CI	*P* Value
Sensitivity analysis using multiple logistic regression model			
Pressure support ventilation	0.381	0.159–0.91	0.030
Age, per yr	1.03	0.98–1.08	0.232
Body mass index ≥ 25 kg/m^2	0.70	0.274–1.79	0.459
Cardiovascular diseases*	2.16	0.171–27.4	0.552
ASA Physical Status ≥ III	1.28	0.132–12.3	0.833
Duration of surgery, per min	1.00	0.99–1.00	0.504

Multiple logistic regression with a simultaneous entering of variables associated with postoperative atelectasis was conducted for *post hoc* sensitivity analysis.

*Cardiovascular diseases included angina pectoris and myocardial infarction.

ASA, American Society of Anesthesiologists.

consolidation (score 0 to 3)[1,19] in the assessment of postoperative atelectasis. Our scoring system is similar to that in the proposal by Monastesse *et al.*,[21] but we replaced "coalescent B-lines" with "loss of A-line with multiple juxtapleural consolidations and irregular pleural lines" (score 2, the most common finding), and added loss of lung sliding and appearance of lung pulse, which indicate noninflating lungs (score 3).[20,44] Of note, A-lines, the reverberation artifacts of the pleura, were lost when the lung parenchyma became inhomogeneous with de-aeration.[44] We did not observe large consolidation (score 4) in anesthesia-induced atelectasis, which is in line with several pediatric cases.[19,20]

This study has some limitations. First, lung ultrasonography depends on the sonographer's skill, and requires patient cooperation. Both greatly influence the diagnostic accuracy of lung ultrasonography. Second, the median lung ultrasound score (5 and 3) and the incidence of hypoxia (22% and 19%) during 48 h postoperatively were not different between the two groups. This may be because these outcomes were not powered to see differences, but it may be because the atelectasis occurring postoperatively is low-grade, and the antiatelectasis effect of pressure support ventilation is transient. However, we regard that small bits of improvement collectively contribute to a better outcome. Therefore, lower incidence of immediate postoperative atelectasis with the use of pressure support ventilation will have an important role in a multimodal approach. Third, atelectasis was diagnosed by consensus reading of two sonographers. It is known that inter- or intrarater variability exists. Thus, independent diagnosis by two sonographers and a statistical test for the degree of the agreement would be a more reliable assessment than consensus reading of two sonographers.[20] Fourth, in this study, low F_{IO_2} (0.4) was maintained during emergence, and patients did not receive oxygen at PACU to avoid absorption atelectasis in both groups. Usually, higher F_{IO_2} is used to prevent hypoxemia in these periods.[35] Therefore, the baseline incidence of atelectasis and outcomes may be different when higher F_{IO_2} is used. Fifth, there were nine patients with unexpected difficult intubation. Attending anesthesiologists proceeded with the protocol because mask ventilation was adequate and intubation was successful with video-laryngoscope in these patients. However, using low F_{IO_2} can be risky in patients with the previous difficult intubation. Finally, this study was performed in patients with a relatively low risk of postoperative atelectasis. Thus, the effect of pressure support ventilation is not known in patients with COPD, obesity, or other significant comorbidities.

In conclusion, pressure support ventilation during emergence from general anesthesia showed a lower incidence of postoperative atelectasis compared to the patient's spontaneous respiration with intermittent manual assistance in laparoscopic colectomy and robot-assisted laparoscopic prostatectomy. Because this result was derived from the low-risk patients of postoperative atelectasis, subsequent validation studies for high-risk patients such as obesity and COPD are required.

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Competing Interests

The authors declare no competing interests.

Reproducible Science

Full protocol available at: hyunjooahn@skku.edu. Raw data available at: hyunjooahn@skku.edu.

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