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Original Article

Ventilation mode effect on weaning outcome in COPD with type II respiratory failure

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Abstract: Objective: To compare adaptive support ventilation (ASV) with synchronized intermittent mandatory ventilation plus pressure support ventilation (SIMV+PSV) in patients with chronic obstructive pulmonary disease (COPD) and type II respiratory failure, and to identify predictors of weaning failure. Methods: This retrospective study classified patients into ASV and SIMV+PSV groups based on ventilation mode received. We collected blood gas values, respiratory mechanics (peak inspiratory pressure [Ppeak], plateau pressure [Pplat], mean airway pressure [Pmean]), pulmonary function indices, and clinical outcomes. Pulmonary function was not measured during invasive mechanical ventilation. Baseline values were obtained from stable pre-intubation records, and post-treatment values were assessed after successful extubation in awake, cooperative, and clinically stable patients. Firth-corrected logistic regression identified independent predictors of weaning failure. Results: Both groups showed similar improvements in blood gas values. ASV achieved greater reductions in Ppeak, Pplat, and Pmean, and larger improvements in forced expiratory volume in 1 second (FEV1), forced vital capacity (FVC), and peak expiratory flow rate (PEFR), which were assessed after successful extubation, compared to SIMV+PSV (all $P<0.001$). The ASV group had lower ventilator-associated pneumonia (VAP) incidence ($P=0.023$), reintubation rate ($P=0.042$), and weaning failure rate ($P=0.007$); in-hospital mortality did not differ. Higher Acute Physiology and Chronic Health Evaluation II (APACHE II) score, pre-treatment arterial carbon dioxide tension ($PaCO_2$), pre-treatment Pplat, and SIMV+PSV mode independently increased weaning failure risk, whereas higher pre-treatment pH and FEV_1 were protective (all $P<0.05$). Conclusion: ASV reduced airway pressures, improved post-extubation pulmonary function, and was associated with fewer VAP events, reintubations, and weaning failures than SIMV+PSV in COPD patients with type II respiratory failure.

Keywords: Chronic obstructive pulmonary disease, adaptive support ventilation, synchronized intermittent mandatory ventilation, pressure support ventilation, weaning failure, firth correction

Introduction

Chronic obstructive pulmonary disease (COPD) imposes a growing global burden, with both incidence and mortality still rising [1, 2]. COPD is common in China. More than 13% of adults 40 years or older are affected, and the rate is rising [3]. During acute exacerbations, many patients develop type II hypercapnic respiratory failure with hypoventilation and hypoxemia. Some require intubation, and in-hospital mortality is high [2, 4]. Mechanical ventilation restores gas exchange and relieves fatigued respiratory muscles. If support is prolonged or poorly set, it can cause ventilator-induced lung injury or ventilator-associated pneumonia and make weaning harder [5]. In hypercapnic acute exacerbations of COPD, noninvasive ventilation reduces the need for intubation and lowers mortality. Even so, many patients still require invasive support [6]. For these patients, careful choice and titration of invasive ventilator modes is key. Whether ASV offers advantages over SIMV+PSV is unclear. We test this question in the present study.

Weaning is often the hardest step in COPD. Success rests on mechanics and gas exchange.

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Reserve matters as well. Stiff lungs and high airway resistance, together with tired respiratory muscles, make separation from the ventilator difficult. Bedside risk tools help. In acute exacerbation of COPD (AECOPD), diaphragm ultrasound predicts weaning. NT-proBNP and RSBI add to signal; longer disease duration does too [7, 8]. Many units still use synchronized intermittent mandatory ventilation plus pressure support ventilation (SIMV+PSV). This is feasible but prone to asynchrony and frequent re-titration. Adaptive support ventilation (ASV) uses a closed loop that adjusts tidal volume (VT) and rate to measured mechanics and the patient's spontaneous effort [9]. In mixed intensive care unit (ICU) cohorts, ASV lowered mechanical power and respiratory rate [10]. Another study showed lower peak airway pressure with better oxygenation than SIMV [11]. A meta-analysis reported shorter ventilation time and lower peak airway pressure [12]. Whether these gains make weaning more reliable in COPD is unclear. We test that question here.

ASV may help by improving synchrony and automating titration. We compared ASV with SIMV+PSV in COPD with type II respiratory failure and assessed blood gases, respiratory mechanics, post-extubation pulmonary function, and key clinical outcomes.

Materials and methods

Study population and sample size calculation

This retrospective observational study included 255 patients with COPD and type II respiratory failure admitted to Lanzhou Petrochemical General Hospital and Zhangye Second People's Hospital between July 2021 and July 2024. Medical records were retrospectively reviewed, and patients were classified into SIMV+PSV or ASV groups based on the ventilation mode they received as part of routine clinical care. The choice of ventilation mode was made by the attending physician according to clinical judgment and ventilator availability, without prospective randomization. All patients received invasive ventilation plus standard therapy, including antimicrobials, expectorants, bronchodilators, and nutritional support. For sample size justification, we referenced El-Behairy et al. [13], who compared PAV with PSV in acute COPD exacerbations and observed a 5 mmHg mean $PaCO_2$ difference (SD 6 mmHg). Although the ventilation modes differed from our study, this reference was selected because it targets the same population and uses the same primary physiologic endpoint ($PaCO_2$ change), and no prior study has directly compared ASV to SIMV+PSV in COPD patients. Using α=0.05 and 80% power, approximately 30 patients per group would detect such a difference. Our actual sample (n=255) substantially exceeded this minimum requirement. We also considered model stability from Iqbal et al. [14], a multicenter cohort (n=214; 161 successes, 53 failures) with 5-6 predictors, yielding an events-per-variable (EPV) of 8.8; despite EPV<10, Firth correction produced stable estimates. This suggests robust estimation is feasible as EPV approaches 10. In our cohort, we observed 52 weaning failures and 203 non-events, with 5 planned predictors, giving EPV=10.4. Thus, the sample (n=255) is adequate for the primary outcome and supports Firth-corrected multivariable logistic regression.

Inclusion and exclusion criteria

Inclusion criteria were: (1) Guideline-defined COPD with type II respiratory failure (PaO_2<60 mmHg and $PaCO_2$>50 mmHg) [15]; (2) Need for invasive mechanical ventilation (IMV); (3) Age ≥18 years; and (4) Complete clinical data with a documented ventilator mode. Exclusion criteria were: (1) Severe cardiac, hepatic, or renal failure; (2) Pneumothorax, sizable pleural effusion, major chest wall deformity, or significant interstitial lung disease; (3) Sedative or analgesic use that precluded valid assessment of spontaneous breathing; (4) IMV<24 hours or missing key data; and (5) GOLD stage I or IV. Patients with GOLD stage I were excluded as they rarely develop type II respiratory failure requiring invasive ventilation. Patients with GOLD stage IV were excluded due to frequent end-stage complications (cor pulmonale, severe malnutrition, respiratory muscle weakness) that may confound the comparison between ventilation modes. The study protocol was approved by the Medical Ethics Committee of Zhangye Second People's Hospital.

Treatment protocol

After admission, all patients received guideline-based care, including antimicrobials, bronchodilators, expectorants, oxygen therapy, and nutritional support. Patients underwent orotra-

cheal intubation and were started on volume assist/control (V-A/C). After patients showed stable hemodynamics and consistent spontaneous effort, we switched to the assigned mode. Control was SIMV+PSV. Initial settings were RR 12-18 breaths/min, VT 7-10 mL/kg IBW, I:E 1:2, FiO_2 0.50, PS 10-20 cmH_2O, and positive end-expiratory pressure (PEEP) titrated per unit protocol. Individual parameter titration was guided by the following criteria: (1) achieving target tidal volume of 6-8 mL/kg IBW with adequate chest rise; (2) maintaining SpO_2 ≥92% and PaO_2 ≥60 mmHg; (3) gradual reduction of $PaCO_2$ toward normal range; (4) ensuring patient comfort without excessive work of breathing; and (5) maintaining peak airway pressure <35 cmH_2O. Parameters were reassessed and adjusted every 4-6 hours based on arterial blood gas results and clinical status.

ASV was the treatment mode. We entered IBW (calculated as: male = 50 + 0.91 × [height in cm - 152.4]; female = 45.5 + 0.91 × [height in cm - 152.4]) and set target minute ventilation to 100% of the predicted value (predicted minute ventilation = IBW × 0.1 L/min/kg). The ventilator automatically optimized the combination of tidal volume and respiratory rate based on measured respiratory system mechanics to achieve the target minute ventilation. FiO_2 was 0.50. PEEP started at 5-8 cmH_2O. We adjusted settings to support spontaneous VT and ventilatory efficiency while avoiding higher airway pressures, based on clinical status, arterial blood gases, Ppeak and Pplat, patient-ventilator synchrony, and comfort.

Patients were considered eligible for transition from invasive ventilation to NIV when the following criteria were met: (1) Resolution or significant improvement of the precipitating cause of respiratory failure; (2) Hemodynamic stability without vasopressor support; (3) Adequate oxygenation (PaO_2/FiO_2>150 mmHg with FiO_2 ≤0.4 and PEEP ≤8 cmH_2O); (4) Respiratory rate <30 breaths/min without signs of respiratory distress; (5) Adequate cough reflex and manageable secretions; (6) Glasgow Coma Scale ≥13; and (7) Successful completion of a spontaneous breathing trial (SBT) for 30-120 minutes with stable respiratory rate (<35 breaths/min), heart rate, blood pressure, and SpO_2 (≥92%).

Clinical data collection

Baseline data included sex, age, height, weight, smoking history, and body mass index (BMI). We recorded Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage, COPD duration, comorbidities, and glucocorticoid use. Comorbidities included diabetes and hypertension; we also noted coronary heart disease. Symptoms were scored with the COPD Assessment Test (CAT) and overall severity with the Acute Physiology and Chronic Health Evaluation II (APACHE II). We measured peak inspiratory pressure (Ppeak) and plateau pressure (Pplat). We also recorded mean airway pressure (Pmean). Arterial blood gases included PaO_2, $PaCO_2$, PaO_2/FiO_2, and pH. These variables were obtained at baseline and after treatment. Pulmonary function indices included forced expiratory volume in 1 second (FEV1), forced vital capacity (FVC), and peak expiratory flow rate (PEFR). Because standard spirometry cannot be validly performed during invasive mechanical ventilation, pulmonary function was not assessed while patients were intubated. Pre-treatment pulmonary function values were obtained from the most recent stable pulmonary function records before intubation or from measurements performed before intubation during the current hospitalization, when available. "Pre-treatment" blood gas and respiratory mechanics measurements were obtained within ≤6 hours before switching to the study ventilation mode (ASV or SIMV+PSV) during a stable period. "Post-treatment" blood gas and respiratory mechanics measurements were uniformly performed after 72 hours of continuous ventilation mode (with a time window of ± 6-12 hours allowed), when ventilation parameters and FiO_2 remained stable for ≥30 minutes. In contrast, posttreatment pulmonary function was assessed only after successful extubation, when patients were awake, cooperative, and clinically stable, using the following criteria: respiratory rate <30 breaths/min, SpO_2 ≥92%, hemodynamic stability, adequate cough, and ability to follow instructions. Thus, post-treatment pulmonary function values were not tied to the 72-hour invasive ventilation window but were obtained after liberation from invasive mechanical ventilation under standardized stable conditions. All measurements were performed when patients were at rest, in consistent position, with unchanged FiO_2, and without additional sedation, with quality control and data recording completed by the same team.

Measurement methods and functional scoring

Blood gas values were measured using the PL2200 portable blood gas analyzer (Prang Medical, Shenzhen, China). This determined arterial blood gas values including PaO_2, $PaCO_2$, and pH. Respiratory mechanics parameters were automatically captured using the BIPAP S/T20 non-invasive ventilator (Philips Respironics, USA), including Ppeak, Pplat, and Pmean. Pulmonary function testing used the BBR-FT-1 pulmonary function tester (Bored, Switzerland), operated by the same technician to measure FEV1, FVC, and PEFR. Pulmonary function testing was performed only after successful extubation, not during invasive mechanical ventilation. All tests were conducted with a standard mouthpiece when patients were awake, cooperative, and clinically stable. At least three acceptable maneuvers were obtained for each patient, and the best value was recorded in accordance with standard operating procedures and ATS/ERS-recommended spirometry quality criteria. We used two functional scoring systems: the CAT [16] and APACHE-II [17]. The CAT has 8 items, each worth 0-5 points (total 0-40). Higher scores mean worse symptom burden. The APACHE-II incorporates 12 physiologic values plus age and chronic health status. Scores range from 0-71, with higher values indicating more severe illness and worse prognosis. Two trained assessors scored all patients independently within 24 hours of admission. We averaged their results.

Definition of clinical outcome indicators

We tracked four outcomes: ICU mortality, VAP, reintubation, and weaning failure. ICU mortality was death from any cause during the ICU stay. VAP was pneumonia occurring >48 h after intubation and initiation of mechanical ventilation (MV), requiring a new or worsening pulmonary infiltrate on imaging plus at least one clinical criterion (fever, purulent secretions, or abnormal white blood cell count). Reintubation was reinsertion of the endotracheal tube within 48-72 h after planned extubation, typically for respiratory failure or airway obstruction. Weaning failure was the inability to sustain spontaneous breathing within 48-72 h after extubation or after a spontaneous breathing trial (SBT); events included reintubation, death, or resumption of MV.

Study outcomes

Our primary outcome was weaning failure, meaning patients needed reintubation or mechanical ventilation restarted within 48 hours of initial weaning. Secondary outcomes were: (1) VAP incidence; (2) Reintubation rate; (3) Inhospital mortality; (4) Improvements in respiratory mechanics and post-extubation pulmonary function (ΔPpeak, ΔPplat, ΔPmean, ΔFEV1, ΔFVC, ΔPEFR); and (5) Blood gas and oxygenation changes (ΔPaO_2, $\Delta PaCO_2$, $\Delta PaO_2/FiO_2$, ΔpH).

Statistical analysis

All analyses were performed in R 4.5.1 and SPSS 26.0. Continuous variables were assessed for normality using the Shapiro-Wilk test. Variables with $P>0.05$ were considered approximately normally distributed and summarized as mean ± SD; otherwise, they were presented as median (Q1, Q3). Between-group comparisons used independent t tests for normally distributed variables or Mann-Whitney U tests for non-normally distributed variables. Categorical variables were presented as n (%) and compared by χ^2 or Fisher's exact tests. To identify independent predictors of weaning failure, we first ran univariate logistic regressions (screening threshold $P<0.05$) and then entered significant variables into a multivariable model. Six variables were retained: APACHE-II score (overall disease severity), pre-treatment pH and $PaCO_2$ (respiratory acidosis severity), Pplat (respiratory mechanics), baseline FEV1 derived from pre-intubation pulmonary function records, and treatment modality (ASV vs. SIMV+PSV). With 52 events and 6 predictors (EPV≈8.67), we applied Firth-corrected logistic regression because: (1) EPV<10 indicates elevated risk of overfitting and separation; (2) Imbalanced event distribution between treatment groups (36 vs. 16 failures) created potential sparse data bias; and (3) Firth correction provides more reliable estimates in such circumstances. Effect sizes are reported as odds ratios (OR) with 95% confidence intervals (CI). Multicollinearity was assessed using the variance inflation factor (VIF), with VIF<5 indicating

Table 1. Comparison of general characteristics and baseline features between two ventilation modalities

Factor	Total	ASV (n=121)	SIMV+PSV (n=134)	Statistic	*P*-value
Sex				0.32	0.571
Male	148 (58.04%)	68 (56.20%)	80 (59.70%)		
Female	107 (41.96%)	53 (43.80%)	54 (40.30%)		
Smoking history				0.81	0.667
Current	164 (64.31%)	80 (66.12%)	84 (62.69%)		
Former	59 (23.14%)	25 (20.66%)	34 (25.37%)		
Never	32 (12.55%)	16 (13.22%)	16 (11.94%)		
GOLD classification				0.109	0.741
III	228 (89.41%)	109 (90.08%)	119 (88.81%)		
II	27 (10.59%)	12 (9.92%)	15 (11.19%)		
Diabetes mellitus				1.415	0.234
Yes	55 (21.57%)	30 (24.79%)	25 (18.66%)		
No	200 (78.43%)	91 (75.21%)	109 (81.34%)		
Hypertension				0.835	0.361
Yes	67 (26.27%)	35 (28.93%)	32 (23.88%)		
No	188 (73.73%)	86 (71.07%)	102 (76.12%)		
Coronary artery disease				0.981	0.322
Yes	42 (16.47%)	17 (14.05%)	25 (18.66%)		
No	213 (83.53%)	104 (85.95%)	109 (81.34%)		
Glucocorticoid use				0.257	0.612
Yes	220 (86.27%)	103 (85.12%)	117 (87.31%)		
No	35 (13.73%)	18 (14.88%)	17 (12.69%)		
Age (years)	70.03 ± 4.47	70.26 ± 4.72	69.81 ± 4.23	-0.805	0.422
Height (cm)	158.12 ± 4.80	158.39 ± 4.57	157.87 ± 5.00	-0.859	0.391
Weight (kg)	54.47 ± 4.30	54.48 ± 4.30	54.46 ± 4.31	-0.029	0.977
BMI (kg/m^2)	20.80 [19.33, 22.34]	20.66 [19.46, 22.23]	20.98 [19.11, 22.38]	0.334	0.738
COPD duration (years)	10.00 [8.00, 12.00]	10.00 [8.00, 12.00]	10.00 [8.00, 12.00]	0.302	0.763
CAT score	26.71 ± 4.36	26.53 ± 4.61	26.87 ± 4.13	0.629	0.53
APACHE-II score	18.05 ± 4.14	17.93 ± 3.93	18.17 ± 4.34	0.473	0.637

Note: ASV, Adaptive Support Ventilation; SIMV, Synchronized Intermittent Mandatory Ventilation; PSV, Pressure Support Ventilation; COPD, Chronic Obstructive Pulmonary Disease; GOLD, Global Initiative for Chronic Obstructive Lung Disease; BMI, Body Mass Index; CAT, COPD Assessment Test; APACHE-II, Acute Physiology and Chronic Health Evaluation II.

acceptable collinearity. All tests were two-sided with α=0.05.

Results

Comparison of baseline characteristics between two ventilation modes

No significant differences were observed between the ASV and SIMV+PSV groups by sex (P=0.571), smoking history (P=0.667), GOLD stage (P=0.741), diabetes (P=0.234), hypertension (P=0.361), coronary heart disease (P=0.322), glucocorticoid use (P=0.612), age (P=0.422), height (P=0.391), weight (P=0.977), BMI (P=0.738), COPD duration (P=0.763), CAT score (P=0.530), or APACHE-II score (P=0.637) (**Table 1**).

Effects of two ventilation modes on blood gas and oxygenation values

After treatment, both groups showed significant improvement in PaO_2, $PaCO_2$, and PaO_2/FiO_2 compared to baseline (all P<0.001), characterized by increased PaO_2 and PaO_2/FiO_2 and decreased $PaCO_2$. pH showed no significant within-group changes in either group (P>0.05). No significant between-group differences were found in pre-treatment, post-treatment, or

Table 2. Comparison of arterial blood gas and oxygenation indices before and after treatment between two ventilation modalities

Group	Pre-treatment pH value	Post-treatment pH value	Statistic/*P*-value	Change (Δ)
SIMV+PSV group (n=134)	7.12 (7.07, 7.17)	7.35 (7.3, 7.39)	-10.044/<0.001	0.22 (0.21, 0.23)
ASV group (n=121)	7.12 (7.07, 7.19)	7.35 (7.3, 7.4)	-9.546/<0.001	0.23 (0.21, 0.24)
Statistic/*P*-value	-0.353/0.725	-0.471/0.638		-0.519/0.603
	Pre-treatment PaO_2	Post-treatment PaO_2	Statistic/*P*-value	Change (Δ)
SIMV+PSV group (n=134)	49.818 ± 4.821	83.541 ± 5.56	-54.658/<0.001	33.723 ± 7.142
ASV group (n=121)	49.845 ± 4.474	83.222 ± 5.86	-47.83/<0.001	33.377 ± 7.676
Statistic/*P*-value	-0.048/0.962	0.445/0.657		0.372/0.71
	Pre-treatment $PaCO_2$	Post-treatment $PaCO_2$	Statistic/*P*-value	Change (Δ)
SIMV+PSV group (n=134)	79.968 ± 4.298	40.798 ± 5.089	65.8/<0.001	-39.17 ± 6.891
ASV group (n=121)	79.982 ± 3.931	40.912 ± 5.015	69.591/<0.001	-39.07 ± 6.176
Statistic/*P*-value	-0.027/0.978	-0.181/0.856		-0.123/0.902
	Pre-treatment PaO_2.FiO_2	Post-treatment PaO_2.FiO_2	Statistic/*P*-value	Change (Δ)
SIMV+PSV group (n=134)	162.116 ± 31.37	346.737 ± 48.072	-37.558/<0.001	184.621 ± 56.903
ASV group (n=121)	161.14 ± 31.148	351.025 ± 49.177	-34.859/<0.001	189.885 ± 59.92
Statistic/*P*-value	0.249/0.804	-0.703/0.483		-0.717/0.474

Note: ASV, Adaptive Support Ventilation; SIMV, Synchronized Intermittent Mandatory Ventilation; PSV, Pressure Support Ventilation; PaO_2, Partial pressure of arterial oxygen; $PaCO_2$, Partial pressure of arterial carbon dioxide; FiO_2, Fraction of inspired oxygen; pH, Potential of hydrogen; Δ, Change.

Table 3. Comparison of respiratory mechanics pressure indices before and after treatment between two ventilation modalities

Group	Pre-treatment Ppeak	Post-treatment Ppeak	Statistic/*P*-value	Change (Δ)
SIMV+PSV group (n=134)	30.355 ± 6.25	26.982 ± 4.072	5.543/<0.001	-3.374 ± 7.045
ASV group (n=121)	30.194 ± 6.574	22.921 ± 3.66	10.339/<0.001	-7.273 ± 7.738
Statistic/*P*-value	0.201/0.841	8.387/<0.001		4.192/<0.001
	Pre-treatment Pplat	Post-treatment Pplat	Statistic/*P*-value	Change (Δ)
SIMV+PSV group (n=134)	25.307 ± 2.926	21.926 ± 3.054	10.02/<0.001	-3.47 (-6.545, -0.605)
ASV group (n=121)	25.118 ± 2.901	18.822 ± 2.929	18.219/<0.001	-6.296 ± 3.801
Statistic/*P*-value	0.519/0.604	8.281/<0.001		5.417/<0.001
	Pre-treatment Pmean	Post-treatment Pmean	Statistic/*P*-value	Change (Δ)
SIMV+PSV group (n=134)	11.819 ± 1.992	9.976 ± 1.099	9.885/<0.001	-1.843 ± 2.158
ASV group (n=121)	12.093 ± 1.955	9.119 ± 1.027	13.862/<0.001	-2.974 ± 2.36
Statistic/*P*-value	-1.109/0.268	6.432/<0.001		3.979/<0.001

Note: ASV, Adaptive Support Ventilation; SIMV+PSV, Synchronized Intermittent Mandatory Ventilation plus Pressure Support Ventilation; Ppeak, Peak inspiratory pressure; Pplat, Plateau pressure; Pmean, Mean airway pressure; Δ, Change.

change values (Δ) for any of these values (all P>0.05) (**Table 2**).

Effects of two ventilation modes on respiratory mechanics pressure values

Pre-treatment Ppeak, Pplat, and Pmean showed no significant between-group differences (P>0.05). After treatment, both groups demonstrated significant reductions in Ppeak (P<0.001), Pplat (P<0.001), and Pmean (P< 0.001) compared to baseline. Between-group comparisons revealed that the ASV group had lower post-treatment Ppeak (P<0.001), Pplat (P<0.001), and Pmean (P<0.001) than the SIMV+PSV group. Corresponding change values (Δ) indicated greater reductions in the ASV group for Ppeak (P<0.001), Pplat (P<0.001), and Pmean (P<0.001) (**Table 3**).

Effects of two ventilation modes on post-extubation pulmonary function values

Pre-treatment pulmonary function values obtained from pre-intubation records, including FEV1, FVC, and PEFR, showed no significant

Table 4. Comparison of pre-intubation baseline and post-extubation pulmonary function indices between two ventilation modalities

Group	Pre-treatment FEV_1	Post-treatment FEV_1	Statistic/*P*-value	Change (Δ)
SIMV+PSV group (n=134)	56.855 (55.133, 58.573)	76.398 ± 5.409	-10.044/<0.001	19.407 ± 5.642
ASV group (n=121)	56.86 (55.13, 59.07)	83.882 ± 4.694	-9.546/<0.001	26.891 ± 5.151
Statistic/*P*-value	-0.281/0.779	-11.826/<0.001		-11.072/<0.001
	Pre-treatment FVC	Post-treatment FVC	Statistic/*P*-value	Change (Δ)
SIMV+PSV group (n=134)	1.6 (1.54, 1.68)	1.91 (1.792, 2.027)	-9.914/<0.001	0.288 ± 0.171
ASV group (n=121)	1.61 (1.53, 1.68)	2.098 ± 0.1	-9.546/<0.001	0.489 ± 0.129
Statistic/*P*-value	0.482/0.63	-9.965/<0.001		-10.628/<0.001
	Pre-treatment PEFR	Post-treatment PEFR	Statistic/*P*-value	Change (Δ)
SIMV+PSV group (n=134)	2.407 ± 0.304	3.774 ± 0.405	-31.626/<0.001	1.367 ± 0.5
ASV group (n=121)	2.421 ± 0.305	4.441 ± 0.355	-50.455/<0.001	2.02 ± 0.44
Statistic/*P*-value	-0.378/0.706	-14.04/<0.001		-11.084/<0.001

Note: ASV, Adaptive Support Ventilation; SIMV, Synchronized Intermittent Mandatory Ventilation; PSV, Pressure Support Ventilation; FEV_1, Forced Expiratory Volume in 1 second; FVC, Forced Vital Capacity; PEFR, Peak Expiratory Flow Rate; Δ, Change.

Table 5. Comparison of mortality, VAP, reintubation, and weaning failure between two ventilation modalities

Group	Mortality	VAP	Reintubation	Weaning Failure
SIMV+PSV group (n=134)	16 (11.94%)	27 (20.15%)	31 (23.13%)	36 (26.87%)
ASV group (n=121)	10 (8.26%)	12 (9.92%)	16 (13.22%)	16 (13.22%)
Statistic/*P*-value	0.938/0.333	5.138/0.023	4.155/0.042	7.290/0.007

Note: ASV, Adaptive Support Ventilation; SIMV+PSV, Synchronized Intermittent Mandatory Ventilation plus Pressure Support Ventilation; VAP, Ventilator-Associated Pneumonia.

between-group differences (P>0.05). Within-group comparisons showed that both groups had significant improvements in post-extubation FEV1 (P<0.001), FVC (P<0.001), and PEFR (P<0.001) compared with pre-intubation baseline values. Between-group comparisons demonstrated that post-treatment FEV_1 (P< 0.001), FVC (P<0.001), and PEFR (P<0.001) were superior in the ASV group compared with the SIMV+PSV group, with greater improvement magnitudes in corresponding change values ΔFEV_1 (P<0.001), ΔFVC (P<0.001), and ΔPEFR (P<0.001) (**Table 4**).

Comparison of prognosis and weaning failure-related outcomes between two ventilation modes

Mortality rates showed no significant difference between the two groups (P>0.05), suggesting no obvious difference in overall mortality outcome between ASV and SIMV+PSV ventilation modes. However, VAP incidence was significantly lower in the ASV group than in the SIMV+PSV group (P=0.023). Reintubation rate was also markedly lower in the ASV group compared to the SIMV+PSV group (P=0.042). Weaning failure rate was likewise significantly lower in the ASV group than in the SIMV+PSV group (P=0.007). Results indicated that ASV mode demonstrates statistical advantages in reducing VAP incidence, decreasing reintubation, and improving weaning success rates, though mortality rates showed no significant difference from the SIMV+PSV mode (**Table 5**).

Comparison of demographics and clinical characteristics between successful and failed weaning patients

To explore factors influencing weaning failure in patients with COPD and respiratory failure, clinical characteristics were compared between successful and failed weaning patients using univariate analysis (**Table 6**). Results showed that weaning success rates differed significantly between treatment strategies (P=0.007), with the ASV mode achieving higher weaning success than the SIMV+PSV mode. APACHE-II score (P<0.001), pre-treatment pH (P<0.001),

Table 6. Comparison of general characteristics and clinical features between weaning success and weaning failure patients

Factor	Total	Weaning Failure (n=52)	Weaning Success (n=203)	Statistic	*P*-value
Treatment regimen				7.290	0.007
SIMV+PSV group	134 (52.55%)	36 (69.23%)	98 (48.28%)		
ASV group	121 (47.45%)	16 (30.77%)	105 (51.72%)		
Sex				0.067	0.796
Male	148 (58.04%)	31 (59.62%)	117 (57.64%)		
Female	107 (41.96%)	21 (40.38%)	86 (42.36%)		
Smoking history				0.158	0.924
Current	164 (64.31%)	33 (63.46%)	131 (64.53%)		
Former	59 (23.14%)	13 (25.00%)	46 (22.66%)		
Never	32 (12.55%)	6 (11.54%)	26 (12.81%)		
GOLD classification				0.579	0.447
III	228 (89.41%)	48 (92.31%)	180 (88.67%)		
II	27 (10.59%)	4 (7.69%)	23 (11.33%)		
Diabetes mellitus				0.701	0.402
Yes	55 (21.57%)	9 (17.31%)	46 (22.66%)		
No	200 (78.43%)	43 (82.69%)	157 (77.34%)		
Hypertension				0.014	0.905
Yes	67 (26.27%)	14 (26.92%)	53 (26.11%)		
No	188 (73.73%)	38 (73.08%)	150 (73.89%)		
Coronary artery disease				0.430	0.512
Yes	42 (16.47%)	7 (13.46%)	35 (17.24%)		
No	213 (83.53%)	45 (86.54%)	168 (82.76%)		
Glucocorticoid use				0.004	0.951
Yes	220 (86.27%)	45 (86.54%)	175 (86.21%)		
No	35 (13.73%)	7 (13.46%)	28 (13.79%)		
Age (years)	70.00 [67.00, 73.00]	69.00 [66.75, 72.00]	70.00 [67.00, 73.50]	1.294	0.196
Height (cm)	158.12 ± 4.80	158.33 ± 4.68	158.06 ± 4.84	-0.360	0.719
Weight (kg)	54.20 [51.40, 57.15]	55.25 [52.48, 57.65]	54.00 [51.25, 56.85]	1.741	0.082
BMI (kg/m^2)	20.80 [19.33, 22.34]	21.06 [19.54, 22.32]	20.72 [19.30, 22.34]	0.797	0.426
COPD duration (years)	10.00 [8.00, 12.00]	9.50 [8.00, 11.00]	10.00 [7.00, 12.00]	0.430	0.667
CAT score	26.71 ± 4.36	26.29 ± 4.46	26.82 ± 4.33	0.781	0.435
APACHE-II score	18.00 [15.00, 21.00]	23.00 [21.00, 24.00]	17.00 [15.00, 19.00]	6.832	<0.001
Pre-treatment pH	7.12 [7.07, 7.18]	7.05 [7.02, 7.08]	7.14 [7.09, 7.20]	-7.774	<0.001
Pre-treatment PaO_2	49.90 [47.06, 52.38]	50.52 [47.55, 52.52]	49.85 [46.78, 52.34]	0.443	0.658
Pre-treatment $PaCO_2$	80.89 [77.24, 83.90]	85.59 [81.78, 86.72]	79.74 [76.78, 82.69]	6.247	<0.001
Pre-treatment PaO_2/FiO_2	160.09 ± 31.60	162.18 ± 29.65	159.55 ± 32.12	-0.533	0.594
Pre-treatment Ppeak	30.28 ± 6.39	30.77 ± 7.37	30.15 ± 6.13	-0.616	0.538
Pre-treatment Pplat	25.22 ± 2.91	28.08 ± 2.44	24.48 ± 2.55	-9.171	<0.001
Pre-treatment Pmean	11.95 ± 1.98	12.41 ± 1.91	11.83 ± 1.98	-1.880	0.061
Pre-intubation FEV_1	56.86 [55.13, 58.78]	58.88 [56.87, 60.48]	56.52 [54.97, 58.26]	5.228	<0.001
Pre-intubation FVC (L)	1.60 [1.54, 1.68]	1.65 [1.55, 1.73]	1.60 [1.54, 1.67]	2.118	0.034
Pre-intubation PEFR (L/s)	2.41 ± 0.30	2.40 ± 0.27	2.42 ± 0.31	0.454	0.650

Note: APACHE-II, Acute Physiology and Chronic Health Evaluation II; $PaCO_2$, Partial pressure of arterial carbon dioxide; Pplat, Plateau pressure; FEV_1, Forced Expiratory Volume in 1 second; FVC, Forced Vital Capacity; ASV, Adaptive Support Ventilation; SIMV+PSV, Synchronized Intermittent Mandatory Ventilation plus Pressure Support Ventilation.

pre-treatment $PaCO_2$ (P<0.001), pre-treatment Pplat (P<0.001), pre-treatment FEV_1 (P<0.001), and pre-treatment FVC (P=0.034) all showed significant between-group differences, sug-

Table 7. Variance inflation factor (VIF) test results for each variable

Variable	Assignment content	VIF	Interpretation
APACHE II score	Original data	1.160	Low multicollinearity
Pre-treatment pH	Original data	1.262	Low multicollinearity
Pre-treatment $PaCO_2$	Original data	1.216	Low multicollinearity
Pre-treatment Pplat	Original data	1.269	Low multicollinearity
Baseline pre-treatment FEV_1	Original data	1.130	Low multicollinearity
Treatment plan	SIMV+PSV group = 1, ASV group = 0	1.158	Low multicollinearity

Note: VIF, Variance Inflation Factor; APACHE-II, Acute Physiology and Chronic Health Evaluation II; $PaCO_2$, Partial pressure of arterial carbon dioxide; Pplat, Plateau pressure; FEV_1, Forced Expiratory Volume in 1 second; ASV, Adaptive Support Ventilation; SIMV+PSV, Synchronized Intermittent Mandatory Ventilation plus Pressure Support Ventilation.

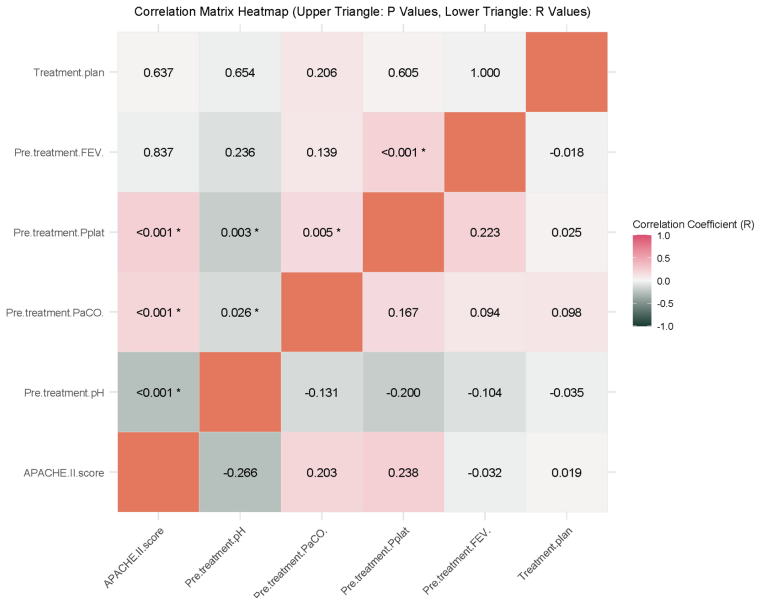

Figure 1. Correlation matrix heatmap for each variable (Upper Triangle: *P*-values; Lower Triangle: R-values). Note: R, Correlation Coefficient; P, *P*-value; APACHE-II, Acute Physiology and Chronic Health Evaluation II; $PaCO_2$, Partial pressure of arterial carbon dioxide; Pplat, Plateau pressure; FEV_1, Forced Expiratory Volume in 1 second; ASV, Adaptive Support Ventilation; SIMV+PSV, Synchronized Intermittent Mandatory Ventilation plus Pressure Support Ventilation.

gesting these indicators may be associated with weaning outcome. Variables including sex, smoking history, GOLD stage, diabetes, hypertension, coronary heart disease, glucocorticoid use, age, height, weight, BMI, COPD duration, CAT score, PaO_2, PaO_2/FiO_2, Ppeak, Pmean, and PEFR showed no statistical significance (P>0.05) (**Table 6**).

Multicollinearity testing and correlation analysis of variables

To verify whether multicollinearity and linear correlation existed among independent variables included in multivariable logistic regression analysis, VIF testing and correlation analysis were performed on major variables (**Table 7** and **Figure 1**). VIF results showed that all variables had VIF values below 2, indicating low multicollinearity among independent variables with no significant impact on regression model stability. Correlation analysis revealed that APACHE-II score showed significant negative correlation with pre-treatment pH (P<0.001), and pre-treatment $PaCO_2$ also showed significant negative correlation with pH (P=0.026). Pre-treatment Pplat demonstrated statistically significant correlations with both $PaCO_2$ and FEV_1 (P=0.003, P=0.005). Other variable correlations did not reach significance (P>0.05). Overall, linear correlations among major influencing factors were weak, data structure was reasonable, and the dataset was suitable for subsequent multivariable logistic regression analysis (**Table 7** and **Figure 1**).

Risk factors for weaning failure: univariate and multivariable results from Firth-corrected logistic regression

To further explore independent risk factors for weaning failure in patients with COPD and respiratory failure, a Firth-corrected logistic regression modeling was employed to mitigate bias from small sample size and data se-

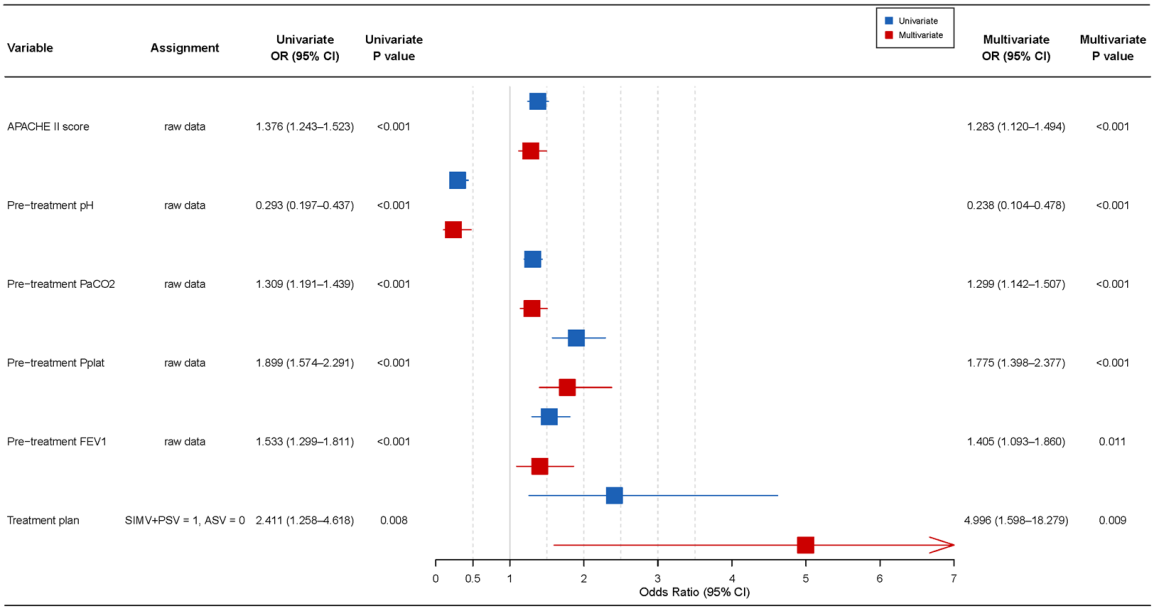

Figure 2. Forest plot of risk factors for weaning failure (Firth-Corrected Logistic Regression). Note: APACHE-II, Acute Physiology and Chronic Health Evaluation II; $PaCO_2$, Partial pressure of arterial carbon dioxide; Pplat, Plateau pressure; FEV_1, Forced Expiratory Volume in 1 second; ASV, Adaptive Support Ventilation; SIMV+PSV, Synchronized Intermittent Mandatory Ventilation plus Pressure Support Ventilation; OR, Odds Ratio; CI, Confidence Interval.

Table 8. Interaction analysis results between treatment regimen and major risk factors

Term	OR	LCL	UCL	*p*.value
Treatment:APACHE-II score	1.007	0.704	1.405	0.967
Treatment:Pre-treatment pH	0.853	0.583	1.179	0.361
Treatment:Pre-treatment $PaCO_2$ (mmHg)	0.980	0.457	1.886	0.954
Treatment:Pre-treatment Pplat	0.798	0.414	1.449	0.469
Treatment:Pre-treatment FEV_1 (%)	0.222	0.006	3.976	0.346

Note: APACHE-II, Acute Physiology and Chronic Health Evaluation II; $PaCO_2$, Partial pressure of arterial carbon dioxide; Pplat, Plateau pressure; FEV_1, Forced Expiratory Volume in 1 second; ASV, Adaptive Support Ventilation; SIMV+PSV, Synchronized Intermittent Mandatory Ventilation plus Pressure Support Ventilation; OR, Odds Ratio; CI, Confidence Interval.

paration. EPV testing showed 52 events, 203 non-events, and 6 candidate independent variables, yielding EPV=8.67<10, indicating some overfitting risk. Therefore, Firth correction was used for robust estimation. Univariate analysis showed that APACHE-II score (P<0.001), pre-treatment pH (P<0.001), pre-treatment $PaCO_2$ (P<0.001), pre-treatment Pplat (P<0.001), pre-treatment FEV_1 (P<0.001), and treatment strategy (P=0.008) were all significantly associated with weaning failure. Multivariable Firth-corrected logistic regression analysis revealed that APACHE-II score (OR=1.255, 95% CI: 1.104-1.448, P<0.001), pre-treatment $PaCO_2$ (OR=1.267, 95% CI: 1.124-1.455, P<0.001), pre-treatment Pplat (OR=1.682, 95% CI: 1.345-2.210, P<0.001), and treatment strategy (SIMV+PSV group = 1, ASV group = 0) (OR=4.265, 95% CI: 1.459-14.347, P=0.007) were independent risk factors for weaning failure. Pre-treatment pH (OR=0.270, 95% CI: 0.124-0.522, P<0.001) and pre-treatment FEV_1 (OR=1.363, 95% CI: 1.074-1.773, P=0.010) showed protective effects. The model demonstrated excellent fit, with ROC analysis yielding AUC=0.961, indicating high predictive performance (**Figure 2**).

Interaction analysis between treatment strategy and major risk factors

We assessed whether treatment effects on weaning failure varied by patient risk using Firth-corrected logistic regression with interac-

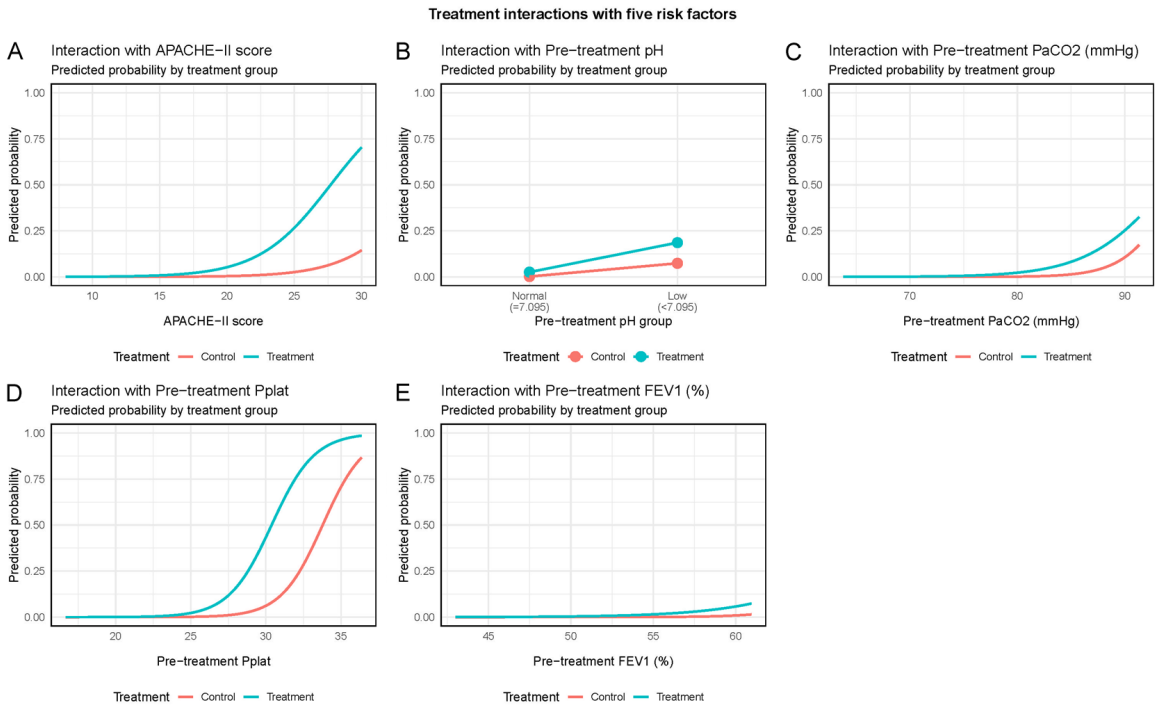

Figure 3. Interaction-predicted probability curves between treatment regimen and five major risk factors. This figure illustrates the predictive effects of interactions between treatment regimen (ASV versus SIMV+PSV) and APACHE-II score (A), pre-treatment pH (B), pre-treatment $PaCO_2$ (C), pre-treatment Pplat (D), and pre-treatment FEV_1 (E) on the probability of weaning failure. APACHE-II, Acute Physiology and Chronic Health Evaluation II; $PaCO_2$, Partial pressure of arterial carbon dioxide; Pplat, Plateau pressure; FEV_1, Forced Expiratory Volume in 1 second; ASV, Adaptive Support Ventilation; SIMV+PSV, Synchronized Intermittent Mandatory Ventilation plus Pressure Support Ventilation.

tion terms between treatment (ASV vs. SIMV+PSV) and five prespecified variables (APACHE II, pre-treatment pH, $PaCO_2$, Pplat, FEV_1; **Table 8**; **Figure 3**). None of the interactions reached statistical significance: treatment×APACHE II (P=0.347), treatment×pH (P=0.590), treatment×$PaCO_2$ (P=0.571), treatment×Pplat (P=0.139), and treatment×FEV_1 (P=0.735). These results did not provide evidence that the treatment effect differs across levels of these risk factors. Marginal predicted probability curves (**Figure 3**) suggested a consistent pattern: at higher risk levels (higher APACHE II, $PaCO_2$, or Pplat), ASV was associated with a lower probability of weaning failure than SIMV+PSV, with the between-group difference narrowing at lower risk. *Post-hoc* power analysis indicated that with 52 events and 5 interaction terms, the study had approximately 35-45% power to detect moderate interaction effects (OR≈2.0) at α=0.05.

Discussion

We compared ASV to SIMV+PSV in COPD with type II respiratory failure. ASV was associated with greater improvements in respiratory mechanics and post-extubation pulmonary function, and with lower rates of weaning failure, reintubation, and VAP. In Firth-corrected logistic regression, higher APACHE II, higher pre-treatment $PaCO_2$, and higher Pplat independently predicted weaning failure, whereas higher pre-treatment pH and FEV_1 were protective. Interaction analyses between treatment and APACHE II, pH, $PaCO_2$, Pplat, or FEV_1 did not reach significance, suggesting no detected heterogeneity of the treatment effect across these risk levels; these trends should be interpreted cautiously.

ASV's defining feature is closed-loop control: the ventilator adjusts VT and respiratory rate in real time to measured compliance, airway resistance, and spontaneous effort [9]. Prior studies report advantages relevant to lung protection and synchrony, including lower mechanical power versus standard pressure control ventilation in mixed ICU cohorts [10] and lower Ppeak with improved oxygenation compared with SIMV [11]. In AECOPD, ASV performed

comparably to NIV for failure and major outcomes while improving patient-ventilator synchrony [18]. In our cohort, ASV lowered Ppeak and Pplat; Pmean also fell. Reductions were larger than with SIMV+PSV. This pattern points to more efficient ventilation at lower pressures and a possible drop in VILI risk. SIMV+PSV needs manual titration. Fixed settings can lag shifts in mechanics and effort, driving asynchrony and extra work of breathing. Gas exchange may slip and pressures can swing. In ARDS, ASV shows similar pressure benefits [11]. COPD is different, so extrapolation must be done with caution. Even so, the pressure profile we observed favors weaning.

PaO_2 rose and $PaCO_2$ fell in both groups. Between-group changes were not statistically significant, so either mode achieved adequate gas exchange. ASV showed a slightly larger $PaCO_2$ drop. A closed loop that adds support when CO_2 drifts up may explain this, though sedation or secretion load could also play a role. pH recovered similarly in both groups, so acid-base status tracked overall ventilatory efficiency rather than a mode effect. In hypercapnic COPD, NIV corrects blood gases efficiently [19]. The setting differs from invasive ventilation. Still, the broader point holds. Responsiveness to changing ventilatory demand helps clear CO_2.

Post-extubation pulmonary function favored ASV. FEV1 and FVC increased more in the ASV group, and PEFR showed a similar trend. Because spirometry was performed only after successful extubation in awake, cooperative, and clinically stable patients, these findings should be interpreted as differences in functional recovery after liberation from invasive mechanical ventilation rather than measurements obtained during invasive ventilation itself. Lower airway pressure exposure and better patient-ventilator synchrony may have contributed to reduced dynamic hyperinflation and improved respiratory recovery, although this interpretation remains inferential. In ARDS, ASV adapts VT to lung mechanics and can lower strain [20], but COPD differs in pathophysiology, so caution is needed when extrapolating these findings. Better synchrony may also reduce respiratory muscle load and support recovery of spontaneous breathing capacity, as suggested by prior studies of weaning physiology in AECOPD [7]. Clinical events were consistent with these physiologic findings. We observed lower VAP incidence, fewer reintubations, and fewer weaning failures in the ASV group, whereas mortality was similar between groups. ASV provided effective support at lower airway pressures. Stronger mechanistic links will require standardized weaning protocols and prespecified assessment times. Prior work reported shorter ventilation time and lower peak airway pressures with ASV versus SIMV [12], and lower pressures during weaning with INTELLIVENT-ASV [21]. Potential explanations include reduced exposure to high pressures and improved synchrony, which may facilitate earlier liberation from the ventilator and may be associated with lower reintubation and VAP risk. ASV's stepwise reduction in support as spontaneous breathing improves may also smooth transition off the ventilator. Reports of higher weaning success and shorter ICU stays with ASV [22] are consistent with our findings, although direct causal inference requires randomized evaluation and standardized weaning protocols.

The lower VAP incidence observed with ASV in our cohort may be explained by several plausible mechanisms, although these were not directly measured in this study. First, ASV's closed-loop algorithm continuously optimizes patient-ventilator synchrony by adjusting tidal volume and respiratory rate based on real-time respiratory mechanics, thereby reducing ineffective triggering and minimizing the risk of microaspiration associated with asynchrony. Second, the significantly lower airway pressures achieved with ASV in our cohort - reflected by greater reductions in Ppeak, Pplat, and Pmean - may help preserve mucociliary clearance and reduce ventilator-induced lung injury, both of which contribute to host defense against pulmonary infection. Notably, our multivariable analysis identified lower Pplat as independently associated with weaning success, suggesting that ASV's pressure-reducing effect may facilitate earlier liberation from mechanical ventilation and thereby shorten ventilator exposure time. Third, improved synchrony reduces the requirement for deep sedation, facilitating earlier spontaneous breathing efforts and potentially shortening the duration of mechanical ventilation - a well-established risk

factor for VAP. The lower reintubation rate observed in the ASV group further supports this pathway, as reintubation prolongs ventilator exposure and substantially increases VAP risk. Finally, ASV's automated weaning function enables a smoother transition from full support to spontaneous breathing, further reducing ventilator exposure time. The superior pulmonary function improvements observed with ASV may reflect reduced dynamic hyperinflation and improved ventilation-perfusion matching, which could accelerate respiratory recovery and facilitate earlier weaning. Collectively, these findings suggest that ASV's pressure-reducing and synchrony-optimizing properties may create a beneficial cascade: lower airway pressures contribute to faster respiratory recovery, earlier weaning eligibility, shorter ventilator duration, and consequently reduced VAP risk. Our findings are consistent with previous meta-analyses reporting shorter ventilation duration and lower airway pressures with ASV compared to conventional modes [12]. However, some studies have not demonstrated significant differences in clinical outcome between ASV and other modes. These discrepancies may be explained by several factors: (1) Differences in patient populations - our study focused specifically on COPD patients with type II respiratory failure, whereas many previous studies included heterogeneous ICU populations with varying underlying pathophysiology; (2) Variations in disease severity and baseline respiratory mechanics, which may influence the degree of benefit from closed-loop ventilation; (3) Differences in conventional ventilator settings and weaning protocols used in control groups; and (4) Heterogeneity in VAP diagnostic criteria across studies. The relatively homogeneous COPD population in our study may have allowed the benefits of ASV to be more apparent.

Predictors of weaning failure in this cohort were higher APACHE II, higher pre-treatment $PaCO_2$, and higher Pplat, while higher pre-treatment pH and FEV_1 were protective. APACHE II reflects global physiologic derangement; sicker patients are less likely to sustain stable cardiorespiratory function after extubation, consistent with reports in hypercapnic COPD and NIV cohorts [23, 24]. Elevated $PaCO_2$ signals inadequate alveolar ventilation and limited respiratory muscle reserve; a higher Pplat indicates reduced compliance and greater resistive load, both unfavorable for weaning. Conversely, preserved FEV_1 marks better ventilatory capacity and endurance, and higher pH reflects less severe respiratory acidosis. Prior studies similarly link illness severity and ventilatory burden with weaning outcome in COPD with acute respiratory failure [2, 25].

We also tested whether treatment effects varied by risk profile. In Firth-corrected models, interactions between treatment (ASV vs. SIMV+PSV) and APACHE II, pH, $PaCO_2$, Pplat, or FEV_1 were not statistically significant, indicating no detected heterogeneity of the treatment effect across these factors. Marginal predicted probabilities suggested a consistent pattern: among higher-risk profiles (higher APACHE II, $PaCO_2$, or Pplat), ASV showed a lower probability of weaning failure than SIMV+PSV, with the difference narrowing at lower risk. Given limited power for interaction testing and multiple comparisons, we interpret these trends as exploratory. Findings that APACHE II predicts adverse outcomes in elderly COPD with type II failure [8] align with the observed direction of ASV's benefit in higher-risk patients, but confirmation requires prospective studies.

Clinically, our findings support ASV as a reasonable option for COPD with type II respiratory failure. ASV was associated with lower airway pressures and fewer weaning complications, and may be particularly useful when V/Q mismatch and respiratory muscle fatigue are prominent, given its closed-loop adjustments and improved synchrony. Automation reduces repeated re-titration. It lightens workload and allows more tailored support. In ARDS, ASV used with recruitment maneuvers improved mechanics and oxygenation [26]. COPD is different, but this points to room to refine ASV strategies in COPD. We used Firth-corrected logistic regression. With few events, it gave stable estimates and suits small samples. NIV already lowers intubation and mortality in hypercapnic AECOPD [19]. ASV fits within invasive ventilation, not as a substitute for NIV. This study had several limitations. First, as a single-center retrospective study, patients were not randomly assigned to ventilation modes; treatment allocation was based on physician discretion and equipment availability, which may have introduced selection bias. Although baseline characteristics were comparable between

groups and multivariable analysis was performed to adjust for confounders, residual confounding cannot be excluded. Additionally, the lack of allocation concealment and blinding inherent to the retrospective design may have introduced subjective bias in clinical decision-making. Second, despite providing standardized initial parameter ranges, individual titration of ventilator settings (e.g., tidal volume, pressure support, PEEP) was performed according to patient response and clinician judgment, which may have introduced variability in treatment delivery between patients and may have influenced outcome. Third, although pulmonary function testing was performed only after successful extubation rather than during invasive mechanical ventilation, spirometry in recently extubated ICU patients still presents inherent challenges. Measurements require patient cooperation, adequate recovery, and standardized technique, which may affect the accuracy and reliability of FEV1, FVC, and PEFR values. Fourth, we did not fully capture several possible confounding factors, including sedative and analgesic exposure, nutritional status, duration of COPD exacerbation before admission, and prior exacerbation frequency, all of which may influence weaning outcome. Fifth, we lacked long-term follow-up data, so sustained pulmonary recovery, readmission rates, and survival beyond hospitalization remain uncertain. Thus, multicenter prospective randomized trials with standardized weaning protocols are needed to confirm effect sizes and test whether these findings generalize to other populations. Future research should also investigate potential interactions between pharmacologic agents (e.g., sevoflurane vs. propofol) and different ventilation modes (e.g., ASV vs. SIMV+PSV) to optimize individualized ventilatory strategies [27].

Conclusion

ASV was associated with greater improvements in respiratory mechanics and post-extubation pulmonary function than SIMV+PSV in COPD with type II respiratory failure, and with lower rates of weaning failure, reintubation, and VAP. In multivariable analysis, higher APACHE II, higher pre-treatment $PaCO_2$, and higher Pplat independently predicted weaning failure, whereas higher pre-treatment pH and FEV_1 were protective. Interaction tests between treatment and key risk factors did not detect significant affect modification; however, post-hoc power analysis revealed limited statistical power (approximately 35-45%) to detect moderate interaction effects. Therefore, while no heterogeneity was detected, these findings should be considered exploratory, and larger studies are needed to adequately evaluate effect modification.

Disclosure of conflict of interest

None.

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