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Approach to mechanical ventilation: a simplified approach for a pulmonologist

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Abstract

Mechanical ventilation is a critical intervention for patients with respiratory failure, providing essential support for oxygenation and ventilation while reducing the work of breathing. It operates through key breath-phase variables: triggering (breath initiation), targeting (flow or pressure delivery), and cycling (ending inspiration). Various ventilation modes, including invasive and non-invasive methods, are tailored to patient needs. Non-invasive ventilation and high-flow nasal cannula are first-line options in acute respiratory distress, whereas invasive mechanical ventilation is necessary for severe cases.

Optimal ventilatory strategies aim to prevent complications such as barotrauma, volutrauma, and dynamic hyperinflation by carefully adjusting parameters like tidal volume, respiratory rate, and positive end-expiratory pressure. One major challenge in mechanical ventilation is patient-ventilator dyssynchrony, where the patient's respiratory efforts do not align with the ventilator's cycles, leading to increased work of breathing and discomfort. Dyssynchrony can occur during the trigger, target, or cycle phases, requiring waveform analysis and ventilator adjustments to optimize synchrony.

Weaning from mechanical ventilation follows a structured process involving readiness assessment, spontaneous breathing trials, and extubation. Successful weaning depends on maintaining stable respiratory function, with close monitoring to prevent post-extubation failure. Identifying and managing ventilatory complications, optimizing patient comfort, and ensuring an individualized approach to ventilator management are key to improving patient outcomes. This review provides a comprehensive understanding of mechanical ventilation, its principles, common challenges, and weaning strategies to guide effective clinical decision-making.

Key words: mechanical ventilation, pulmonology, critical care, respiratory failure, ventilator management, invasive ventilation.

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Introduction

Mechanical ventilation (MV) is a medical intervention that employs a ventilator to support or completely take over the process of gas exchange in the lungs [1]. The primary objective of MV is to ensure adequate oxygenation and carbon dioxide elimination while reducing the burden on the respiratory muscles. The evolution of MV has been shaped by multiple disciplines, including anatomy, physiology, chemistry, and clinical medicine. One of the earliest recorded uses of positive-pressure ventilation dates back to 1543, when Andreas Vesalius successfully resuscitated an animal using this technique [2].

Significant progress in MV was made during the poliomyelitis outbreaks of the 1950s. A major breakthrough came when anesthesiologist Bjorn Ibsen introduced tracheostomy and positive-pressure ventilation for patients with bulbar poliomyelitis, resulting in a dramatic reduction in mortality rates from 87% to 40% within 24 hours [3]. The first functional mechanical ventilator was developed by Claus Bang and Carl-Gunnar Engström to overcome the limitations of manual ventilation [4]. Another landmark advancement in MV was the introduction of positive end-expiratory pressure (PEEP). In 1972, Siemens-Eléma launched the Servo 900A, the first ventilator to incorporate PEEP, marking a new era in ventilation technology [5,6]. Over the ensuing decades, ventilators have become increasingly sophisticated, featuring enhanced monitoring capabilities and alarm systems.

Despite significant advancements, well-defined criteria for initiating MV in acute respiratory failure remain elusive. The key objectives of MV include [7]: i) optimizing pulmonary gas exchange; ii) alleviating respiratory distress; iii) modifying pressure-volume relationships; iv) promoting lung and airway healing.

The primary indications for MV are as follows [8]: i) airway protection in patients with impaired consciousness due to conditions such as head trauma, stroke, drug overdose, or anesthesia; ii) hypercapnic respiratory failure caused by airway, chest wall, or neuromuscular disorders; iii) hypoxemic respiratory failure; iv) circulatory failure, where MV and sedation help decrease the oxygen demand of respiration.

This review aims to explore the fundamental principles of MV,

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its physiological interactions with the respiratory system, and the various ventilation modes available. Additionally, it examines the role of MV in managing specific conditions, including acute respiratory distress syndrome (ARDS) and chronic obstructive pulmonary disease (COPD).

Basics: physiology, mechanical breath-phase variables, mechanical ventilation-type of breath

Physiology

A thorough understanding of MV starts with the basic principles of respiratory physiology and mechanics involved in normal spontaneous breathing (SB). SB is the natural process in which air moves in and out of the lungs through the effort of the respiratory muscles. In contrast, positive pressure ventilation (PPV) delivers air into the lungs by generating positive pressure in the airway. This can be achieved through various methods, such as an endotracheal tube, tracheostomy tube, or non-invasive mask. Understanding these fundamental concepts is crucial for managing acute respiratory conditions, as ventilator-derived physiological data provide real-time assessments of pulmonary function on a breath-by-breath basis [9].

The lungs are situated within the chest cavity, which is enclosed by the chest wall (Figure 1). The pleural space, located between the lungs and the chest wall, plays a key role in respiratory mechanics. The lungs, made of elastic tissue, naturally tend to collapse inward, whereas the chest wall has a tendency to expand outward. The interaction of these opposing forces determines lung volume at the end of expiration. Due to this balance, the pressure within the pleural space, known as pleural pressure (Ppl), remains lower than atmospheric pressure at the end of expiration [10].

At equilibrium, the forces expanding the alveolar walls outward must counterbalance the forces pushing them inward. The outward force is represented by alveolar pressure (Palv), whereas the inward forces consist of Ppl and lung elastic recoil pressure (Pel). The difference between Palv and Ppl, known as transpulmonary pressure (Ptp), is equal in magnitude but opposite in direction to the Pel at a given lung volume [10] (Figure 1).

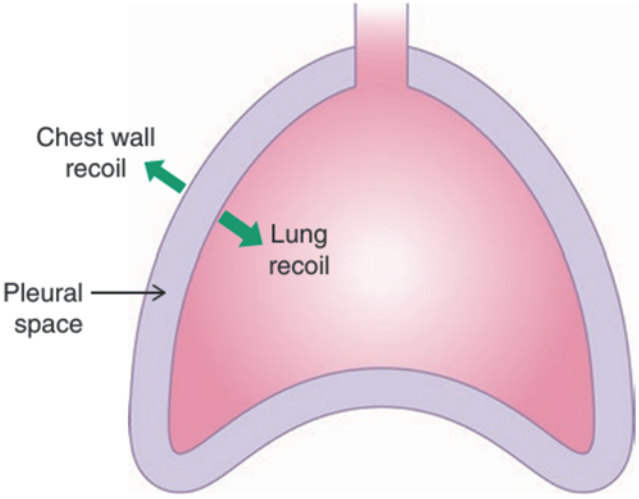


Figure 1. Balance between the inward recoiling of the lung and the outward recoiling of the chest wall.

For the lungs to inflate, Ptp must increase. This relationship is defined by: Ptp=Palv−Ppl. This increase can occur in two ways: by lowering Ppl during SB or by raising Palv using PPV.

Air movement in the respiratory system can be likened to an electrical circuit, following the principles of Ohm's law. In this comparison, the pressure difference between the proximal airway (Pair), measured at the mouth, and Palv functions like the voltage difference in an electrical circuit. Airflow (Q) represents the current, while airway resistance (R) is analogous to electrical resistance. The equation describing airflow in the respiratory system is: Q=(Pair−Palv)/R.

By convention, airflow is positive during inspiration (air entering the lungs) and negative during expiration (air leaving the lungs).

Mechanical ventilation and airflow dynamics

During MV, the ventilator increases airway pressure at the Pair during inspiration, creating a pressure gradient that moves air into the lungs. Expiration, however, remains a passive process, similar to natural breathing. A summary of the mechanisms involved in different breathing modes: i) SB – a drop in Palv below atmospheric pressure allows air to enter the lungs; ii) PPV – a rise in Pair pressure above atmospheric pressure forces air into the lungs.

Equation of motion in respiratory mechanics

The relationships between pressure, volume, and airflow throughout the breathing cycle are represented by the equation of motion for the respiratory system: Paw=P0+(R×flow)+(Vt×ERS)

Where Paw is the pressure at the airway opening, P0 is the initial Palv, R is the airway resistance, Vt is the tidal volume, and ERS is the elastance of the respiratory system. Each factor contributes to airway pressure regulation during MV.

Impact of airflow resistance and compliance

In conditions like COPD, insufficient expiratory time may prevent the lungs from fully returning to their resting volume before the next breath. This can result in intrinsic PEEP (auto-PEEP), where Palv remains elevated at the end of expiration. In passive patients, airway pressure measurements taken during an end-expiratory occlusion provide the total PEEP value.

ERS reflects the stiffness of the respiratory system and is the inverse of compliance (CRS): ERS=1/CRS (Figure 2).

R represents the pressure difference needed to generate airflow, with a significant portion of inspiratory resistance originating from the endotracheal tube. Understanding these principles is essential for optimizing ventilatory management in clinical settings (Figure 2).

During an end-inspiratory pause, the measured airway pressure is referred to as the plateau pressure (Pplat). Since airflow is absent at this moment, Pplat effectively represents Palv, eliminating the influence of airway resistance. Lung compliance can be assessed using the equation of motion during an inspiratory pause: CRS=Vt/Pplat−P0

Two straightforward tests (end-inspiratory and end-expiratory occlusion maneuvers) are valuable in diagnosing respiratory complications. These tests help identify increased R and elevated total PEEP, which are common in conditions like COPD and asthma. Additionally, they can detect high ERS, indicating reduced lung compliance, a hallmark of ARDS [8].

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Positive Pressure Ventilation	Spontaneous Ventilation
Ventilator increases proximal airway pressure ↓ Air flows into lungs ↓ Increase in alveolar pressure ↓ Increase in transpulmonary pressure ↓ Increase in lung volume	Inspiratory muscles contract ↓ Increase in intrathoracic volume ↓ Decrease in intrathoracic pressure ↓ Decrease in pleural pressure ↓ Increase in transpulmonary pressure ↓ Increase in lung volume ↓ Decrease in alveolar pressure ↓ Air flows into lungs until alveolar pressure equals atmospheric pressure

Figure 2. Algorithm showing the mechanism of positive pressure ventilation and spontaneous ventilation.

Mechanical ventilation phase variables

Breathing follows a repetitive cycle of inspiration and expiration. Each breath can be broken down into four phase variables that regulate different aspects of the respiratory cycle: i) trigger, which determines the initiation of inspiration; ii) target, which defines how flow is delivered during inspiration; iii) cycle, which specifies the mechanism that ends inspiration; iv) baseline, which maintains airway pressure during expiration. Different MV modes are classified based on these phase variables, as they dictate how each breath is delivered.

Trigger

The trigger variable sets the start of inspiration, which can be initiated either by the ventilator (ventilator-triggered) or by the patient (patient-triggered):

- ventilator-triggered breaths: initiated based on a pre-set time interval.
- patient-triggered breaths: activated by the patient's respiratory effort, detected through changes in pressure or flow.

Breaths can be of different types[10]: i) control breath, triggered by the ventilator using a time-based mechanism; ii) assist breath, initiated by the patient through pressure or flow variations; iii) assist-control (A/C) mode, a hybrid mode where breaths can be patient-triggered (assist) or ventilator-triggered (control).

Most ventilators display breath types using indicators such as "A" (assist) or "C" (control). These differences can also be observed in the pressure waveform: patient-triggered (assist) breaths show a negative pressure deflection before inspiration, while time-triggered (control) breaths lack this deflection.

If the patient's actual respiratory rate (RR) exceeds the ventilator's set rate, it indicates that the patient is actively initiating assist breaths.

Target

The target variable controls how flow is delivered during inspiration. The two most common target types are flow and pressure.

- Flow-targeted ventilation: flow is preset as the independent variable; airway pressure depends on airway resistance and Palv; waveforms often include constant flow or a decelerating ramp pattern; and changes in the pressure waveform may indicate patient inspiratory efforts.
- Pressure-targeted ventilation: the ventilator maintains a set airway pressure, adjusting flow as needed; and as Palv increases during inspiration, the ventilator decreases flow dynamically, producing a decelerating ramp pattern.

Cycle

The cycle variable determines when inspiration terminates. The most common cycle variables include: i) volume-cycled (inspiration ends when a pre-set tidal volume is delivered); ii) time-cycled (inspiration stops after a specific time interval); iii) flow-cycled (the breath ends when inspiratory flow decreases to a set threshold).

While pressure cycling is rarely the primary cycling mechanism, it functions as a safety feature in volume-cycled modes to prevent excessively high airway pressures.

In pressure-targeted ventilation, the flow waveform typically follows a decelerating ramp pattern, where flow is highest at the beginning of inspiration and gradually decreases as Palv rises. In flow-cycled ventilation, inspiration stops once inspiratory flow drops to a certain percentage of the peak inspiratory flow [10].

Baseline

The baseline variable refers to the Pair pressure during expi-

ration. In MV, this pressure is usually maintained above atmospheric pressure by the ventilator. This process is known as PEEP and helps prevent alveolar collapse during exhalation.

Mechanical ventilation: types of breaths

Breaths in MV are classified based on their initiation source. There are three primary types of breaths: i) mandatory breath, fully controlled by the ventilator and triggered at a preset RR; ii) assisted breath, a mix of spontaneous and mandatory breathing, where the patient initiates a breath, and the ventilator assists by delivering a set tidal volume and flow; iii) spontaneous breath, completely initiated by the patient. Even though the breath is self-initiated, the ventilator still provides support based on trigger sensitivity settings.

Different ventilation modes

MV modes are classified based on key phase variables: trigger, target, and cycle. The three primary modes include: i) volume-controlled ventilation (VCV); ii) pressure-controlled ventilation (PCV); iii) pressure support ventilation (PSV). Each mode determines how these variables interact to regulate breath delivery. Ventilator operation is managed by an electronic, pneumatic, or microprocessor-based controller, ensuring appropriate breath administration based on pre-programmed algorithms and feedback mechanisms [11,12]. Modern ventilators utilize piston/bellows systems, turbines, or high-pressure gas controllers to regulate airflow.

Volume-controlled ventilation

In VCV, breath initiation follows an A/C mechanism, meaning it can be either patient-triggered (by detecting pressure or flow changes) or ventilator-triggered (based on a preset time). The target variable is flow, meaning that both the flow rate and waveform pattern are predetermined. The most commonly used flow waveforms include constant flow and decelerating ramp flow. The cycle variable is volume, meaning the ventilator delivers a fixed tidal volume with each breath. Since the flow remains constant, inspiratory time is determined by the equation: inspiratory time=tidal volume/flow rate. As a result, neither patient effort nor lung mechanics affect the inspiratory duration.

Classification of volume-controlled ventilation

- Trigger: time- or patient-triggered
- Target: flow-targeted
- Cycle: volume-cycled
- Mode type: assist or controlled

Advantages and disadvantages of volume-controlled ventilation

Advantages: it ensures a consistent tidal volume, regardless of changes in lung mechanics, which is crucial for maintaining adequate ventilation [13].

Disadvantages: it generates higher airway pressures at the end of inspiration compared to PCV, potentially increasing the risk of ventilator-induced lung injury (VILI); and the constant inspiratory flow may not meet the demands of patients requiring higher flow rates, leading to discomfort. A decelerating flow pattern can improve patient tolerance [13].

Pressure-controlled ventilation

In PCV, the breath initiation mechanism is similar to VCV (A/C). However, the target variable is pressure, meaning the ventilator delivers a preset airway pressure while adjusting flow dynamically to maintain it. This results in a decelerating ramp flow waveform, where flow is highest at the start of inspiration and decreases gradually. The cycle variable is time, meaning inspiration continues until a predetermined inspiratory time is reached.

Classification of pressure-controlled ventilation

- Trigger: time- or patient-triggered
- Target: pressure-targeted
- Cycle: time-cycled
- Mode type: assist or controlled

Advantages and disadvantages of pressure-controlled ventilation

Advantages: it reduces the risk of alveolar overdistension and VILI by limiting peak Palv; and it provides better patient comfort compared to VCV due to a higher initial inspiratory flow and a longer inspiratory time [13].

Disadvantages: delivered alveolar volume may decrease if airway resistance increases or lung compliance decreases, potentially leading to inadequate ventilation [13].

Pressure support ventilation

In PSV, breaths are exclusively patient-triggered, meaning it functions in assist-only mode. Similar to PCV, the target variable is pressure, meaning the ventilator adjusts flow to reach and maintain a preset airway pressure. This mode also produces a decelerating ramp flow waveform. However, unlike PCV, the cycle variable is flow, meaning inspiration ends when inspiratory flow declines to a preset percentage of the peak inspiratory flow (*e.g.*, 25%).

Classification of pressure support ventilation

- Trigger: patient-triggered
- Target: pressure-targeted
- Cycle: flow-cycled
- Mode type: assist-only

End-inspiratory pressures in different modes

The interpretation of airway pressure at the end of inspiration differs between volume-cycled (VCV) and pressure-cycled (PCV) ventilation.

Volume-controlled ventilation: peak and plateau pressures

In VCV, airway pressure progressively increases as the preset tidal volume is delivered. The peak airway pressure (Ppeak) at the end of inflation represents the combined pressures needed to overcome both airway resistance (Pres) and lung/chest wall elastance (Pel) [13]: Ppeak=Pres+Pel

Pres results from airflow resistance within the airways and endotracheal tube, and Pel is determined by lung and chest wall compliance.

To measure Pel separately, an inflation-hold maneuver is per-

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formed by temporarily pausing airflow at the end of inspiration. The pressure recorded during this pause is the Pplat, which represents peak Palv at the end of inspiration [13]: Pplat =Palv (peak)

Thus, Pplat reflects the maximum Palv in the respiratory cycle, is obtained using the inspiratory pause maneuver, and increases with higher tidal volumes and lower lung compliance.

Pressure-controlled ventilation: end-inspiratory airway pressure

In PCV, the end-inspiratory airway pressure (Paw) should be equal to peak Palv at the end of inspiration, as there is no airflow at this stage [13,14]: Paw(end−insp)=Palv(peak) (Figure 3 and *Supplementary Tables 1 and 2*).

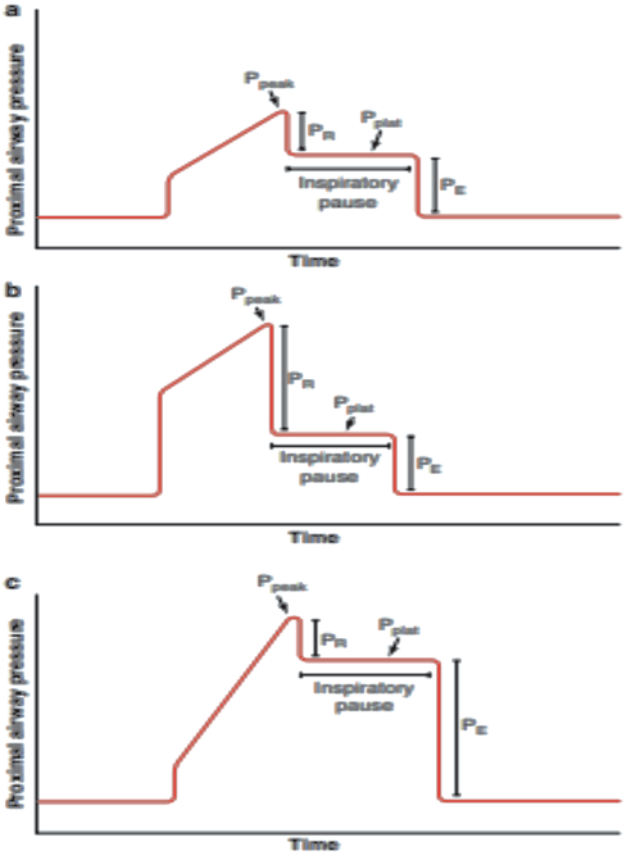


Figure 3. Pressure waveforms with inspiratory pause maneuver. a) normal peak and plateau pressure; b) elevated peak pressure but normal plateau pressure; the elevated peak pressure is due to elevation in the resistive component of proximal airway pressure; c) elevated peak pressure and elevated plateau pressure; the elevated peak pressure is due to elevation in the resistive component of proximal airway pressure. Ppeak, peak pressure; PR, resistive component of proximal airway pressure; Pplat, plateau pressure; PE, elastive component of proximal airway pressure.

Mechanical ventilation in acute respiratory distress syndrome and chronic obstructive pulmonary disease

Mechanical ventilation in acute respiratory distress syndrome

Pathophysiological basis of ventilation strategies in acute respiratory distress syndrome

ARDS is a life-threatening lung condition characterized by severe respiratory failure and high mortality rates. First identified over five decades ago, ARDS is distinct from acute hypoxemic respiratory failure due to the presence of bilateral pulmonary infiltrates on imaging, a key diagnostic criterion. It is an acute, widespread inflammatory condition that increases alveolar-capillary membrane permeability, lung weight, and reduces aerated lung volume. Clinically, ARDS presents with hypoxemia, bilateral radiographic opacities, reduced lung compliance, increased venous admixture, and elevated physiological dead space. The hallmark histopathological feature in early ARDS is diffuse alveolar damage.

The condition arises from direct lung injuries (*e.g.*, pneumonia, aspiration) or indirect systemic causes (*e.g.*, sepsis, pancreatitis, trauma), leading to non-hydrostatic pulmonary edema. Management primarily focuses on lung-protective ventilation (LPV) and supportive therapies to minimize further injury while optimizing oxygenation.

ARDS is diagnosed clinically using the Berlin definition (2012), which includes the following four criteria: i) timing – symptoms must develop within 1 week of a known insult or demonstrate worsening respiratory function within the past week; ii) chest imaging – bilateral pulmonary opacities on X-ray or computed tomography, not fully explained by effusions, lung collapse, or nodules; iii) origin of edema – respiratory failure must not be primarily due to cardiac dysfunction or fluid overload. If ARDS risk factors are absent, echocardiography or other tests should confirm the absence of hydrostatic pulmonary edema; iv) oxygenation impairment – defined by the partial pressure of oxygen (PaO_2)/fraction of inspired oxygen (Fio_2) ratio while receiving at least 5 cm H_2O of PEEP.

For resource-limited settings, the Kigali modification proposed by Riviello *et al*. adapts the Berlin definition by omitting PEEP as a requirement and using oxygen saturation/FiO_2≤315 to define hypoxia, ensuring ARDS is not underdiagnosed when arterial blood gas analysis is unavailable [15,16].

While treating the underlying cause remains a priority, MV plays a crucial role in supporting oxygenation and ventilation. However, improper ventilation strategies can contribute to VILI, a phenomenon where MV exacerbates lung damage. In ARDS, lung infiltrates predominantly affect dependent lung regions, reducing the functional lung volume and increasing the risk of overdistension in the remaining aerated lung areas, thereby predisposing to VILI [17].

There are three primary mechanisms of VILI: i) volutrauma – excessive alveolar expansion leading to structural damage at the alveolar-capillary barrier, triggering inflammation and pulmonary infiltration. This injury is volume-dependent rather than pressure-dependent [18]; ii) barotrauma – high airway pressures may rupture alveoli, resulting in air leaks, including complications like pneumothorax, pneumomediastinum, and subcutaneous emphysema. Keeping Pplat below 30 cm H_2O helps reduce this risk; iii)

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atelectrauma – repeated alveolar collapse and reopening during ventilation cause epithelial injury due to shear stress. Applying PEEP maintains alveolar stability and prevents atelectasis [18].

In addition to mechanical injury, ventilation can trigger an excessive inflammatory response (biotrauma), leading to the release of proinflammatory cytokines, which can result in systemic inflammatory response syndrome and multi-organ dysfunction [19].

Ventilator strategies in acute respiratory distress syndrome

The primary goal of MV in ARDS is to reduce respiratory muscle workload, ensure adequate gas exchange, and minimize VILI [20]. This is achieved through LPV, a strategy based on the findings of the ARMA trial [21]. Key principles of LPV include: i) ventilation mode – VCV is preferred to deliver a consistent tidal volume; ii) tidal volume – set at 6 mL/kg of predicted body weight, with RR adjustments to maintain a pH of 7.30-7.45 and/or appropriate $PaCO_2$ levels; iii) VILI prevention: maintain Pplat <30 cm H_2O to prevent barotrauma, limit tidal volume to 6 mL/kg to reduce volutrauma, and use at least 5 cm H_2O of PEEP to minimize atelectrauma.

Positive end-expiratory pressure and FiO_2 adjustment

PEEP prevents alveolar collapse at end-expiration, reduces atelectrauma, and improves oxygenation. Although the optimal PEEP and FiO_2 titration remains debated, guidelines suggest higher PEEP levels for moderate-to-severe ARDS. Studies indicate that higher PEEP, without lung recruitment maneuvers, provides the most benefit [22,23]. The recommended oxygenation target in LPV is PaO_2 between 55-80 mmHg [24].

Prone ventilation

Prone positioning enhances alveolar recruitment, improves oxygenation, and minimizes ventilation-induced lung injury by redistributing Ptp and reducing dorsal lung compression [25,26]. The PROSEVA trial demonstrated that severe ARDS patients (PaO_2/FiO_2<150 mmHg with FiO_2≥0.6 and PEEP≥5 cm H_2O) benefit significantly from prone positioning for at least 16 hours daily, ideally within 36 hours of ARDS diagnosis [27].

Neuromuscular blocking agents

Neuromuscular blocking agents (NMBAs) may be considered in severe ARDS cases requiring prone ventilation. Their advantages include reducing reverse triggering, pendelluft (regional air shifts within the lung), and patient-ventilator dyssynchrony [28]. A 2019 randomized controlled trial (RCT) found no significant mortality reduction at 90 days with NMBA use, though they may still be useful in select cases, such as extracorporeal membrane oxygenation (ECMO)-supported patients [29].

Extracorporeal carbon dioxide removal

For refractory ARDS, veno-venous ECMO is a potential option. A meta-analysis of two RCTs demonstrated a 90-day mortality benefit with ECMO in ARDS patients [30].

Corticosteroid use in acute respiratory distress syndrome

The role of steroids in non-COVID ARDS remains a subject of debate. Recent studies such as DEXA-ARDS and meta-analyses suggest a mortality benefit with systemic corticosteroids [31,32]. Early administration (within the first week of illness) and a treatment duration of over 7 days appear most effective. According to 2017 Society of Critical Care Medicine and European Society of Intensive Care Medicine guidelines, methylprednisolone (1 mg/kg) with a 13-day taper is recommended for ARDS patients with PaO_2/FiO_2<200, particularly when initiated early [33] (*Supplementary Table 3* and Figure 4).

Mechanical ventilation in chronic obstructive pulmonary disease exacerbation

Pathophysiology of ventilation strategies in chronic obstructive pulmonary disease

COPD encompasses chronic bronchitis and emphysema, both of which lead to persistent airflow restriction and respiratory distress. Acute exacerbations of COPD (AECOPD) can result in a sudden deterioration of lung function, manifesting as severe airway obstruction, gas trapping, dynamic hyperinflation (DH), and intrinsic PEEP (PEEPi). These physiological alterations often necessitate hospitalization and respiratory support.

Airflow limitation, gas trapping, and intrinsic positive end-expiratory pressure

COPD-related airway obstruction arises from increased resistance in both large and small airways due to factors such as bronchoconstriction, mucus overproduction, and structural damage to the airway walls, often triggered by epithelial irritation [34]. Expiratory flow limitation occurs as airway resistance impedes effective exhalation [35]. This issue intensifies with increased respiratory effort, leading to higher tidal volumes and RR [36]. The resultant air trapping causes Palv to remain elevated at the end of expiration, leading to the development of auto-PEEP or PEEPi [37].

Dynamic hyperinflation

DH occurs when air trapping leads to an increase in end-expiratory lung volume, particularly during heightened ventilatory demand, such as exercise or hyperpnea. The increased intrathoracic pressure associated with hyperinflation contributes to an elevated work of breathing, prolonged ventilator dependence, and difficulty in weaning from MV [38]. According to the "waterfall theory", increasing downstream pressure beyond a certain threshold does not improve expiratory flow unless external PEEP matches or exceeds the critical closing pressure of the airway. To mitigate hemodynamic instability and excessive hyperinflation, external PEEP should be maintained at approximately 75-85% of auto-PEEP [39].

Diagnosis of dynamic hyperinflation

DH can be identified using the following clinical indicators: i) delayed refilling of the manual ventilator bag; ii) absence of a plateau phase in capnography; iii) expiratory flow failing to return to zero in a flow-time waveform; iv) direct measurement of PEEPi.

Auto-PEEP can be measured in select ventilators through a 2-second expiratory pause, provided the patient is passive and not initiating breaths. It is calculated by subtracting external PEEP from total PEEP [40].

Ventilator strategies in acute exacerbation of chronic obstructive pulmonary disease

The primary aim of MV in AECOPD is to optimize gas exchange while preventing or minimizing DH. This is accom-

plished by extending expiratory time and reducing tidal volumes to mitigate air retention. Ensuring sufficient time for exhalation before the next breath reduces the likelihood of gas trapping, auto-PEEP, and further hyperinflation [10]. Instead of solely aiming for $PaCO_2$ normalization, ventilator settings should be tailored based on auto-PEEP levels and DH.

Goals of mechanical ventilation in AECOPD [41] are the following: i) stabilize oxygenation and ventilation; ii) reduce respiratory effort; iii) minimize DH.

Key determinants of dynamic hyperinflation and auto-positive end-expiratory pressure

DH and auto-PEEP are primarily influenced by three factors: i) minute ventilation; ii) inspiratory-to-expiratory (I:E) ratio; iii) expiratory time constant.

To optimize ventilation, strategies include maintaining a lower RR, reducing tidal volume, increasing the I:E ratio, and adjusting peak flow rates to prolong the expiratory phase and minimize hyperinflation.

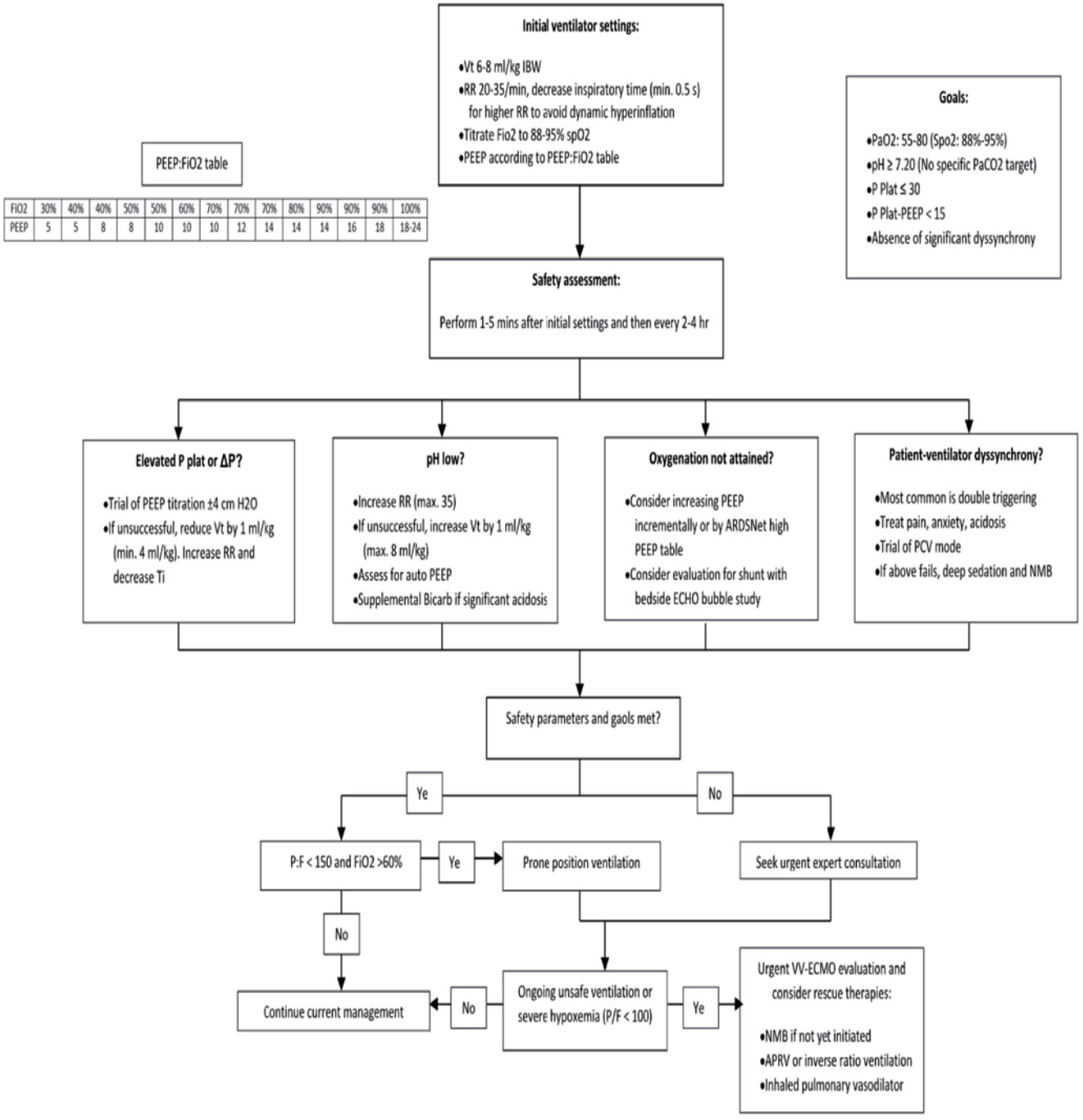


Figure 4. Algorithm for management of acute respiratory distress syndrome.

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Indications for invasive mechanical ventilation in acute exacerbation of chronic obstructive pulmonary disease [42]

MV is indicated if any of the following conditions are met: i) failure or intolerance of non-invasive ventilation (NIV); ii) respiratory or cardiac arrest; iii) significant aspiration; iv) altered mental status; v) inability to clear secretions; vi) heart rate <50 bpm; vii) hemodynamic instability unresponsive to fluid resuscitation; viii) severe ventricular arrhythmias; ix) life-threatening hypoxia.

Ventilation protocols for acute exacerbation of chronic obstructive pulmonary disease

For passive (sedated and non-triggering) patients:

- Mode: VCV for consistent tidal volume delivery
- Tidal volume: 5-6 mL/kg
- RR: 8-14 breaths/min
- PEEP: 5-8 cm H_2O
- I:E ratio: 1:3 to 1:6
- Flow waveform: square
- Pplat: ≤30 cm H_2O
- Target PaO_2: 55-60 mmHg
- Target pH: 7.2-7.4

External PEEP is typically avoided in passive patients as their breaths are time-triggered.

For SB patients [43]:

- Mode: pressure support, pressure control, or proportional assist ventilation
- Pressure support: adjusted to maintain a tidal volume of 8 mL/kg with minimal trigger pressure or flow; peak flow set to 80-100 L/min
- PEEP: start at 5 cm H_2O, adjusting in increments of 2 cm H_2O
- Monitoring: observe RR, work of breathing, and missed breaths in the flow-time scalar; settings should be modified to maintain an optimal RR and synchronize with the ventilator
- Pressure monitoring: regular assessment of peak inspiratory pressure and Pplat. If pressures increase, PEEP should be lowered. Expiratory sensitivity can be adjusted beyond the standard 25% if necessary

If a patient exhibits ventilator asynchrony, potential causes such as pain or fever should be investigated. If no underlying issue is found, mild sedation may be considered to improve comfort and synchronization (*Supplementary Table 4*).

Patient-ventilator dyssynchrony – monitoring ventilator waveforms

In patients who are deeply sedated, paralyzed, or have significant neurological impairment, the ventilator assumes full control of breathing. However, in many cases, MV is used to assist SB, reducing the patient's respiratory effort. Effective synchronization between the patient and ventilator is crucial for proper breath initiation (triggering), inspiratory flow delivery (targeting), and breath termination (cycling). When this coordination is disrupted, patient-ventilator dyssynchrony occurs, leading to patient discomfort and increased work of breathing. Dyssynchrony is categorized based on which phase of the ventilatory cycle is affected: trigger phase, target phase, or cycle phase [10].

Trigger-related dyssynchrony

Ineffective triggering

Ineffective triggering, also known as missed triggering, occurs when the patient's inspiratory effort fails to initiate a ventilator-assisted breath. For a breath to be triggered, the patient's inspiratory effort must exceed a preset pressure or flow threshold. In cases of severe respiratory muscle weakness, the patient may struggle to generate sufficient effort.

Common causes of ineffective triggering include: weak respiratory muscles, high trigger sensitivity threshold, continuous-flow nebulizer use with flow-triggered ventilators, and presence of PEEPi (auto-PEEP).

During AECOPD, incomplete lung emptying due to expiratory flow limitation leads to elevated Palv at the end of expiration. When Palv remains higher than the Pair pressure, auto-PEEP develops. To initiate a breath, the patient must generate enough negative pressure to lower Palv below Pair pressure.

If auto-PEEP contributes to ineffective triggering, applying external PEEP may help by increasing Pair pressure and reducing the inspiratory effort needed to trigger the ventilator. However, this intervention must be carefully adjusted to avoid worsening Palv. This principle follows the "waterfall effect", where external PEEP must be optimized to minimize air trapping while still improving breath initiation.

Extra triggering

Extra triggering occurs when the ventilator initiates breaths inappropriately, without actual inspiratory effort from the patient. This can manifest as either auto-triggering or double triggering.

Auto-triggering happens when external factors, such as circuit condensation, small air leaks, or cardiac oscillations, cause pressure or flow variations that the ventilator mistakenly interprets as patient-triggered efforts. Double triggering occurs when two consecutive breaths are delivered in rapid succession. This is often seen when the patient's inspiratory effort continues beyond the ventilator's preset inspiratory time, leading to an additional breath being triggered before exhalation is completed.

Target-related dyssynchrony

The target variable determines how inspiratory flow is delivered. If the ventilator fails to meet the patient's inspiratory demand, flow dyssynchrony may occur. This is characterized by a drop in Pair pressure and a distinctive "scooped-out" appearance on the pressure waveform.

Strategies to manage flow dyssynchrony include: i) increasing inspiratory flow to meet patient demand; ii) identifying and addressing underlying factors that increase respiratory effort (*e.g.*, agitation, pain); iii) switching to a pressure-targeted mode, such as PCV or PSV, which allows greater patient control over inspiratory flow.

Cycle-related dyssynchrony

The cycle variable determines when inspiration ends. The neural inspiratory time (neural TI) refers to the duration of inspiratory muscle contraction, while the ventilator inspiratory time (ventilator TI) is the duration set by the ventilator. When these two are misaligned, cycle dyssynchrony occurs [10].

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Premature cycling

Premature cycling occurs when the ventilator's inspiratory time (TI) is shorter than the patient's neural TI. This can lead to double triggering, where the patient's diaphragm remains contracted even after the ventilator cycles to expiration, causing a drop in Pair pressure and triggering an additional breath.

Management strategies include: i) extending the ventilator's inspiratory time; ii) in VCV, reducing the inspiratory flow rate or increasing tidal volume; iii) in PCV, increasing inspiratory time. Double triggering is identified by neural TI > ventilator TI.

Delayed cycling

Delayed cycling occurs when the ventilator prolongs inspiration beyond the patient's neural TI. This results in the ventilator continuing to deliver air while the patient is already attempting to exhale. In VCV, delayed cycling can be recognized by notching at the end of the pressure waveform. Delayed cycling is characterized by: neural TI < ventilator TI (*Supplementary Table 5*).

Weaning from mechanical ventilation

Weaning from MV is a critical step in the recovery process for critically ill patients in the intensive care unit. Prolonged dependence on ventilatory support increases the likelihood of complications such as ventilator-associated lung injury, infections, and neuromuscular weakness. The process of weaning is generally divided into four main phases: assessing readiness, conducting a SB trial (SBT), weaning, and performing extubation.

Assessing readiness for weaning

Evaluating a patient's readiness for weaning is essential to determine whether they can undergo an SBT. This assessment helps ensure that patients are appropriately selected for the next phase, minimizing the risks associated with premature or delayed weaning. Clinical criteria are used to assess readiness, as detailed in the corresponding table.

Spontaneous breathing trial

Once a patient meets the readiness criteria, they undergo an SBT to determine whether they can sustain SB without ventilatory assistance. The purpose of the SBT is to simulate post-extubation conditions and assess whether the patient has sufficient respiratory muscle strength and endurance to maintain unassisted breathing. The two primary methods used for conducting an SBT are the T-piece trial and low-level PSV. The trial duration typically ranges from 30 minutes to 2 hours.

Pressure support ventilation

Daily SBTs utilizing inspiratory pressure support are widely recommended due to their safety, effectiveness, and evidence from randomized studies. The specific mode of ventilatory support varies based on institutional protocols and clinical judgment. Typically, PSV is conducted with an inspiratory pressure support level of 5 to 8 cm H_2O, as recommended by the American College of Chest Physicians and the American Thoracic Society [44,45].

PSV has two key advantages that make it a reliable predictor of successful extubation [10]: i) triggering mechanism – PSV is fully patient-triggered (assist-triggered) without ventilator-driven control breaths; ii) compensation for airway resistance – the inspiratory pressure setting helps offset the increased work of breathing caused by the resistance of the endotracheal tube.

During a PSV-SBT, the standard parameters include: PEEP maintained at 5 cm H_2O, and FiO_2 kept at 0.4 or lower.

A successful trial is indicated by a high tidal volume with minimal inspiratory pressure, suggesting that the patient's respiratory muscles can sustain SB effectively.

T-piece trial

The T-piece trial is another method used to evaluate post-extubation respiratory function. In this approach, the patient is disconnected from the ventilator while the endotracheal tube remains in place.

Key characteristics of a T-piece trial include: i) the ventilator is removed, but the endotracheal tube remains in place; ii) oxygen is supplied through a T-piece without any positive pressure assistance; iii) the patient is monitored for indicators of respiratory distress, such as tachypnea, use of accessory muscles, hypoxemia, agitation or somnolence, and hemodynamic instability [10].

T-piece trials may be particularly useful in cases such as acute cardiogenic pulmonary edema, particularly if a patient does not tolerate an initial PSV-SBT. Similar to PSV trials, T-piece trials generally last between 30 minutes and 2 hours (Figure 5).

Conclusions

MV is a life-saving intervention used in critically ill patients with respiratory failure to ensure adequate oxygenation and ventilation, reducing the work of breathing and supporting gas exchange until the underlying cause is treated.

The process of MV is structured around key phases of a breath: the trigger phase (which initiates inspiration), the target phase (which delivers flow or pressure during inspiration), and the cycle phase (which ends the inspiratory period), all of which are critical to achieving synchrony between the patient and ventilator.

Various modes of MV are available, including invasive and non-invasive methods, tailored according to the patient's condition. NIV and high-flow nasal cannula are often first-line options in acute respiratory failure to avoid intubation, while invasive MV is reserved for more severe cases.

The choice of ventilatory strategy and mode depends on the patient's respiratory mechanics, focusing on protecting the lungs from further injury by using lung-protective strategies like low tidal volumes, optimal RR, and appropriate PEEP settings to improve oxygenation and avoid complications such as barotrauma and volutrauma.

During MV, conditions like gas trapping, DH, and PEEPi can develop due to incomplete exhalation or increased airway resistance, making careful adjustment of ventilatory settings essential to minimize patient discomfort and maintain adequate ventilation.

Patient-ventilator dyssynchrony is a common challenge where the patient's spontaneous efforts do not match the ventilator's cycles, resulting in increased work of breathing and discomfort; recognizing and correcting dyssynchrony requires careful analysis of ventilator waveforms and appropriate adjustment of settings.

Trigger dyssynchronies occur when the patient is unable to initiate a ventilator breath effectively (ineffective triggering) or when the ventilator delivers breaths inappropriately without patient effort

STEP 1: READINESS FOR WEANING

Clinical criteria used to determine readiness for trials of spontaneous breathing

Required criteria	
1.	The cause of the respiratory failure has improved
2.	PaO2/FiO2≥150 or SpO2≥90% on Fi02≥40% and PEEP≤5 cmH2O
3.	pH>7.25
4.	Hemodynamic stability (no or low dose vasopressor medications)
5.	Able to initiate an inspiratory effort
Additional criteria (optional criteria)	
1.	Hemoglobin ≥7 g/dL
2.	Core temperature ≤38 to 38.5°C
3.	Mental status awake and alert or easily arousable

↓

STEP 2: SPONTANEOUS BREATHING TRIAL (PSV/T-PIECE)

↓

STEP 3: Weaning Criteria and Protocol

Weaning Parameters	1. Alert, awake, cooperative 2. PaO2>60 mmHg, FiO2<50%, PEEP≤5 crn H2O, PaCO2 and pH acceptable 3. Spontaneous TV≥5 mL/kg, vital capacity >10 mL/kg, MV<10 L/min, MVV > double MV 4. Maximum negative inspiratory pressure >20-25 cm H2O 5. Static compliance >30 mL/cmH2O, stable vitals at 1 hour (<SBI) 6. Respiratory rate <30 bpm, RSBI <105
Endotracheal Tube	1. Largest tube possible 2. Consider supplemental PSV suction
Arterial Blood Gas	1. Avoid/treat metabolic alkalosis [PaO2 60-65 mmHg] 2. PaCO2 at or above baseline for patients with CO2 retention
Nutrition	Adequate, correct electrolyte imbalances
Secretions	Clear regularly, avoid dehydration
Neuromuscular Factor	1. Avoid neuromuscular depressing drugs 2. Avoid steroids
Obstruction	Appropriate bronchodilator
Wakefulness	Avoid over-sedation, preferably in morning

↓

STEP 4: EXTUBATION

Figure 5. Algorithm showing weaning protocol for a patient on mechanical ventilation. PaO2, partial pressure of oxygen; FiO2, fraction of inspired oxygen; SpO2, oxygen saturation; PEEP, positive end-expiratory pressure; PaCO2, partial pressure of carbondioxide; TV, tidal volume; MV, mechanical ventilation; MVV, maximum voluntary ventilation; SBI, Spontaneous Breathing Index; RSBI, Rapid Shallow Breathing Index; PSV, pressure support ventilation.

(auto-triggering), which may be due to circuit issues or improper sensitivity settings.

Target-related dyssynchronies arise when the flow or pressure delivered during inspiration does not meet the patient's demand, which can be addressed by adjusting inspiratory flow rates or switching to pressure-targeted modes to ensure better synchrony and comfort.

Cycle dyssynchronies are observed when there is a mismatch between the duration of inspiratory effort by the patient and the ventilator's set inspiratory time, leading to premature or delayed cycling, both of which can compromise effective ventilation and require fine-tuning of inspiratory time and flow termination criteria.

Weaning from MV involves a structured process that includes assessing readiness, conducting SBT to evaluate the patient's ability to breathe without assistance, and extubation when the patient demonstrates stable respiratory function, with continuous monitoring to prevent respiratory failure recurrence.

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Online supplementary material:

Supplementary Table 1. Three main ventilator modes and settings.

Supplementary Table 2. Basic modes of ventilation.

Supplementary Table 3. Management of ARDS based on ATS guidelines.

Supplementary Table 4. Indications and protocol of invasive mechanical ventilation for COPD/asthma exacerbation.

Supplementary Table 5. Patient-ventilator dyssynchrony – monitoring ventilator waveforms.

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