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REVIEW

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Safe titration of pressure-support ventilation: Balancing patient effort, drive, and ventilator assistance

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ABSTRACT:

Pressure-support ventilation (PSV) is commonly used for invasive weaning and assisted ventilation. However, bedside titration often relies on tidal volume and rate rather than direct indices of respiratory drive and inspiratory effort, risking over- or under-assistance with downstream diaphragm dysfunction, asynchrony, and lung stress. This narrative review synthesized a physiology-guided approach to safe PSV titration and proposed practical bedside guidance for intensivists adjusting trigger sensitivity, rise time, pressure-support level, and cycling to maintain synchrony while targeting a moderate, sustainable patient effort. Practical monitoring included airway pressure-based measures, including airway occlusion pressure at 100 ms (P0.1), end-expiratory occlusion pressure (Pocc), and the pressure muscle index (PMI), as well as esophageal manometry, diaphragm electrical activity (EAdi), and diaphragm ultrasound thickening fraction (TFdi). Integrating these tools promotes an "adequate assistance" zone in which tidal volume (VT) and respiratory rate remain stable across modest PS changes and effort is neither suppressed nor excessive. Proportional modes, such as neurally adjusted ventilatory assist (NAVA) and proportional assist ventilation plus (PAV+), can enhance interaction by scaling support to immediate effort, but physiology-based assessment remains central to individualized, lung- and diaphragm-protective ventilation during PSV.

Keywords: Diaphragm; Patient–ventilator interaction; Pressure-support ventilation; Proportional assist ventilation; Respiratory drive; Respiratory muscles; Lung and diaphragm protective ventilation

INTRODUCTION

Pressure-support ventilation (PSV) is a partial ventilatory support mode in which each breath is patient-triggered and flow-cycled, with the ventilator delivering a constant positive pressure above positive end-expiratory pressure (PEEP) to assist inspiration. Introduced in the 1980s as a weaning mode, PSV allows patients to maintain spontaneous breathing effort while unloading part of the inspiratory workload, thereby preventing respiratory muscle disuse atrophy compared with fully controlled ventilation modes [1,2].

Over the past decades, PSV has become the most commonly used partial support mode in intensive care, particularly during weaning and spontaneous-breathing trials [3]. Observational data also indicate that spontaneous-breathing modes, including PSV, are frequently applied even in the early stages of critical illness and acute respiratory distress syndrome (ARDS) [4]. Despite its prevalence, clinical practice for setting and titrating pressure support remains largely empirical. In most ICUs, clinicians adjust the level of support based on observed respiratory rate, tidal volume, or other vital signs rather than on direct physiological measurements of respiratory effort or drive [5,6].

Because the tidal volume during PSV ensues from the combined result of ventilator assistance and patient-generated effort, a fixed pressure support level may either over-assist or under-assist depending on the patient's drive, effort, and respiratory mechanics [6,7]. Both over-assistance and under-assistance have been associated with patient–ventilator asynchrony, ventilator-induced diaphragm dysfunction (VIDD), and patient self-inflicted lung injury (P-SILI) [8,9]. Yet, no universally accepted bedside framework exists to guide safe, physiology-based titration of PSV.

Prior work has established the importance of balancing lung- and diaphragm-protective goals during assisted ventilation and has summarized available tools to monitor respiratory drive and inspiratory effort [9,10]. However, this review outlines a physiology-guided approach to safely titrating pressure-support ventilation, emphasizing the interactions among respiratory drive, patient effort, and ventilator assistance. We summarize practical methods for assessing these variables at the bedside and describe how to recognize and avoid over- and under-assistance during PSV by integrating widely available bedside surrogates such as P0.1 (Airway occlusion pressure at 100 ms), Pocc, and PMI.

PHYSIOLOGY AND IMPORTANCE OF RESPIRATORY DRIVE AND EFFORT

During PSV, patient–ventilator interaction is largely governed by the patient's respiratory drive and muscular effort. Respiratory drive represents the neural output from the brainstem that initiates inspiration, while respiratory effort reflects the mechanical pressure generated by the respiratory muscles in response to that drive [9,11]. These two components are closely related, but not identical. Critically ill patients may exhibit strong neural drive but weak inspiratory effort due to muscle dysfunction, resulting in shallow tidal volumes despite visible respiratory distress [12].

Both insufficient and excessive drive can be harmful. High drive may arise from hypoxemia, hypercapnia, acidosis, pain, or anxiety, leading to excessive effort, large transpulmonary pressures, and potential patient self-inflicted lung injury (P-SILI) [13]. Conversely, low drive from sedation, alkalosis, or muscle weakness can lead to ineffective triggering and diaphragm disuse.

KEY MESSAGES:

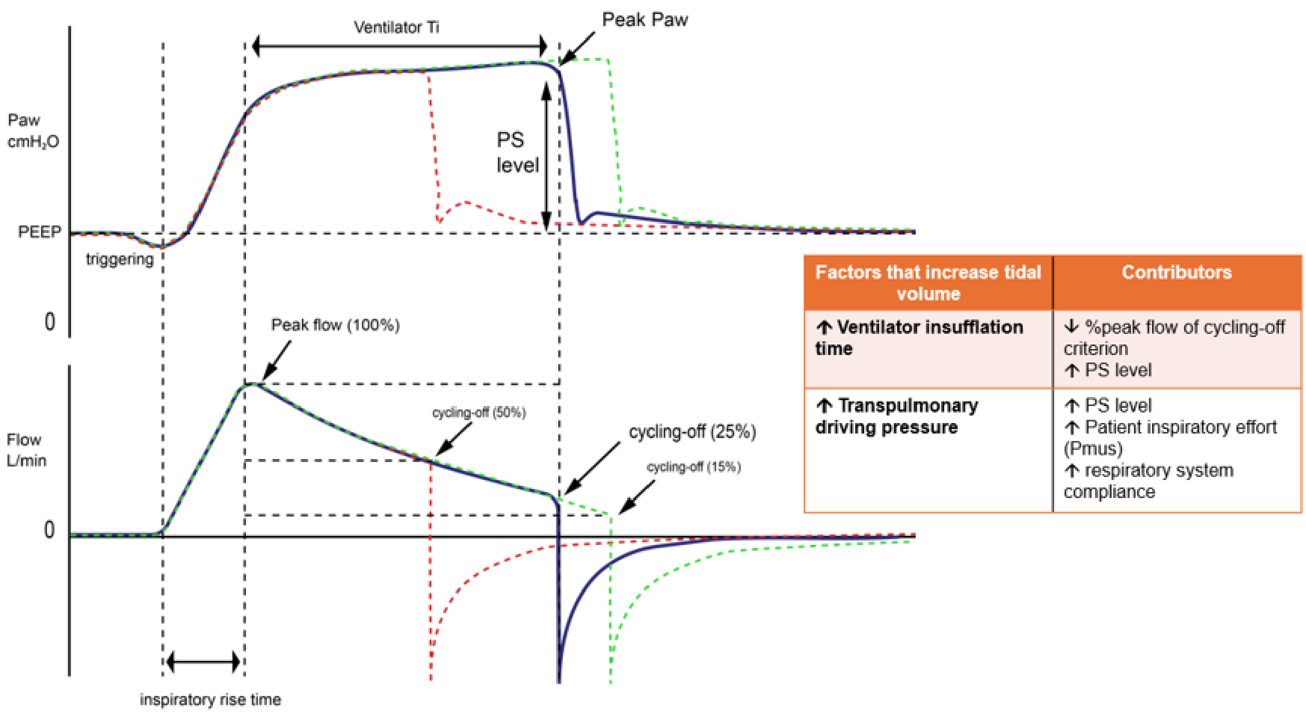
- Safe titration of pressure-support ventilation (PSV) remains challenging when based solely on basic physiological parameters such as respiratory rate, tidal volume, or heart rate.
- Both over-assistance (leading to diaphragm disuse and asynchrony) and under-assistance (causing fatigue or patient self-inflicted lung injury) can be harmful.
- Bedside assessment using airway pressure-based measures (P0.1, Pocc, PMI), esophageal manometry, and diaphragmatic measurements (ΔPes, EAdi, TFdi) allows physiology-guided individualization of ventilatory support, balancing respiratory drive, effort, and assistance.

Understanding the interplay between respiratory drive, inspiratory effort, and ventilator assistance is essential for safe PSV titration that maintains synchrony and achieves lung- and diaphragm-protective ventilation by preventing both under-assistance (excessive effort and lung stress) and over-assistance (diaphragm unloading and disuse) [6,7,14].

HOW TO SET PSV

In PSV, ventilator performance depends on the fine adjustment of several settings that determine how the machine senses, delivers, and terminates each assisted breath (Figure 1). Four key adjustable parameters govern how the ventilator interacts with the patient's inspiratory effort [5]:

- **Trigger sensitivity** – Determines how readily the patient's inspiratory effort initiates a ventilator breath; overly insensitive settings risk delayed triggering and asynchrony. It can be set as a flow-trigger (typically 1–5 L/min) or pressure-trigger (−1 to −2 cmH_2O) depending on the patient's condition.
- **Inspiratory rise time** – Controls how rapidly the ventilator reaches the target pressure after triggering; a shorter rise time improves comfort and synchrony but may cause overshoot or high flow in compliant lungs.
- **Pressure-support level (PS)** – Determines the magnitude of positive pressure above PEEP (positive end-expiratory pressure) delivered during inspiration, directly influencing VT (tidal volume) and unloading of respiratory muscles; in patients with normal compliance, higher PS generally yields higher VT.
- **Cycling-off criterion** – Defines the point at which the ventilator switches from inspiration to exhalation, usually when inspiratory flow decreases to a preset fraction of its peak (e.g., 25% of peak flow), thereby affecting inspiratory time (Ti) and patient–ventilator synchrony.



Factors that increase tidal volume	Contributors
↑ Ventilator insufflation time	↓ %peak flow of cycling-off criterion ↑ PS level
↑ Transpulmonary driving pressure	↑ PS level ↑ Patient inspiratory effort (Pmus) ↑ respiratory system compliance

Figure 1. Airway pressure and flow waveforms during PSV. Each assisted breath is patient-triggered and flow-cycled. Airway pressure (Paw) rises to the preset pressure-support level (PS) above positive end-expiratory pressure (PEEP) and remains constant until inspiratory flow decays to a predefined fraction of its peak (cycling-off), marking the transition to exhalation. Adjustments in PS level, cycling-off threshold, or patient effort alter inspiratory time (Ti) and VT. Lower cycling-off thresholds or higher PS levels prolong insufflation and increase VT, whereas higher cycling thresholds shorten Ti and reduce VT. The box summarizes major factors that increase tidal volume, including higher PS level, greater inspiratory effort (Pmus), longer insufflation time, and higher respiratory system compliance.

MONITORING INSPIRATORY DRIVE AND EFFORT DURING PSV

Monitoring respiratory drive and effort is essential for safe titration of PSV. Objective monitoring helps maintain synchrony, optimize unloading, and prevent both diaphragm disuse and excessive inspiratory workload.

A range of techniques can be used to evaluate respiratory drive and effort. These can be broadly categorized by the method used to assess respiratory drive or respiratory effort: Changes in airway pressure, esophageal manometry, or diaphragmatic measurement.

1. Assessment by changes in airway pressure
    - Airway occlusion pressure at 100 ms (P0.1)
    - End-expiratory occlusion pressure (Pocc)
    - Pressure Muscle Index (PMI)

    Figure 2 summarizes the typical waveform patterns observed during end-expiratory and end-inspiratory occlusion maneuvers used to assess respiratory drive (P0.1), inspiratory effort (Pocc), and the pressure muscle index (PMI) at the bedside.
2. Esophageal manometry
3. Diaphragmatic measurements
    - Diaphragm electrical activity (EAdi)
    - Diaphragm ultrasound

Assessment by changes in airway pressure

Airway occlusion pressure at 100 ms (P0.1)

The airway occlusion pressure at 100 milliseconds (P0.1) is an indirect measure of respiratory drive, meaning how strongly the brain signals the patient to breathe. It represents the drop in airway pressure that occurs during the first 100 milliseconds of an inspiratory effort, when the airway is briefly occluded at the end of expiration [5,15,16]

Because this time window is very short, the patient cannot consciously modify their effort, and lung mechanics (such as compliance or resistance) have minimal influence on the result. Therefore, P0.1 primarily reflects neural drive, rather than muscle strength or lung mechanics [15-17].

In PSV, P0.1 can help guide ventilatory support adjustments because it closely correlates with inspiratory effort and central respiratory drive. A P0.1 ≤ 1.0 cmH_2O suggests low respiratory drive or potential over-assistance, while a P0.1 ≥ 3.5–4.0 cmH_2O indicates high drive and possible under-assistance. These thresholds have demonstrated good diagnostic accuracy, detecting insufficient ventilatory support with approximately 92% sensitivity and 89% specificity [16,18].

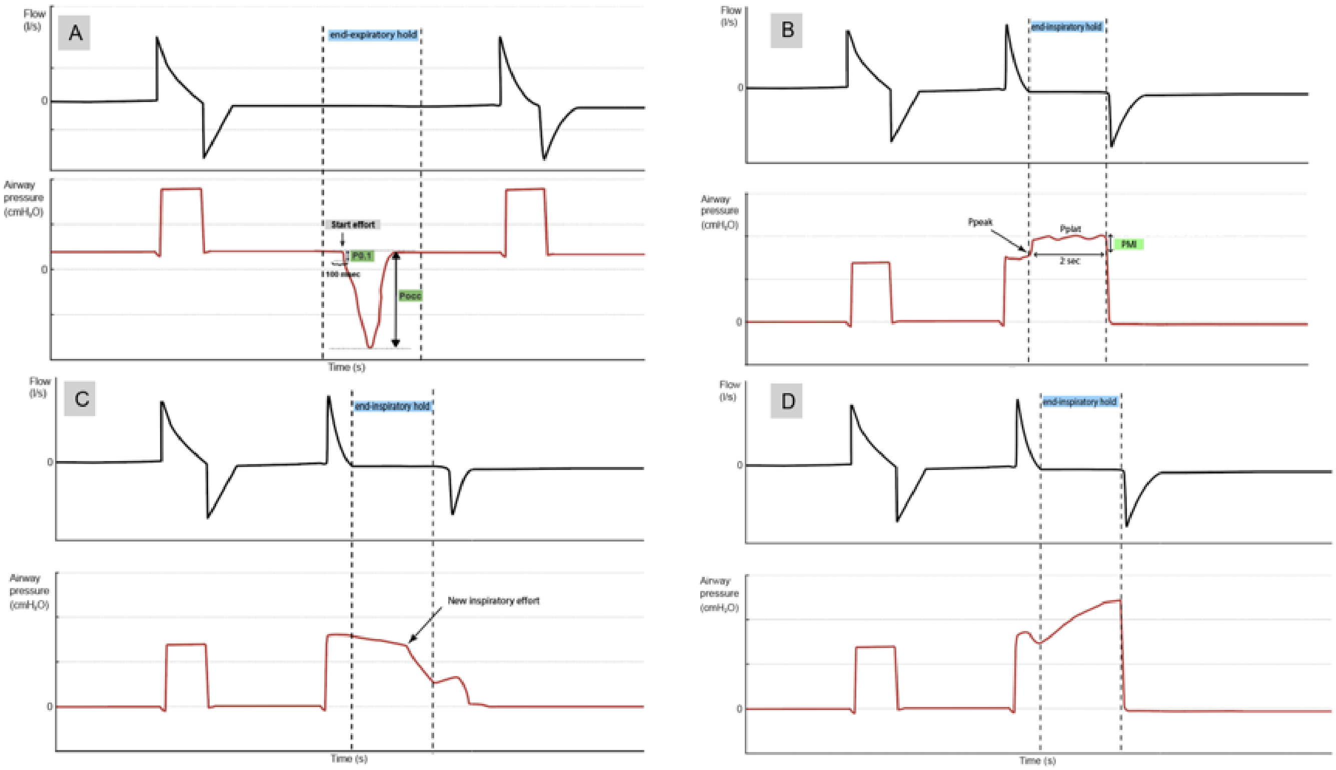


Figure 2. Representative airway pressure and flow waveforms during inspiratory and expiratory occlusion maneuvers used to assess respiratory drive and effort. Panels A–D illustrate pressure (Paw, red) and flow (black) waveforms during bedside occlusion maneuvers used to measure inspiratory drive and effort. (A) End-expiratory hold maneuver: P0.1 is the fall in Paw during the first 100 ms of an occluded inspiration, reflecting respiratory drive. Pocc is the maximum negative Paw during a full inspiratory effort against occlusion, representing inspiratory muscle pressure. (B) End-inspiratory hold maneuver: the Pressure Muscle Index (PMI) is defined as Pplat – Ppeak when a stable plateau (> 2 s) is reached, estimating the additional pressure contributed by patient effort. (C–D) Examples of unreadable plateaus caused by patient activity: (C) a new inspiratory effort during the hold, causing a pressure drop; (D) expiratory effort causing a pressure rise. Adapted from Bianchi et al. [26] and Kyogoku et al. [28]

Limitations:

P0.1 values can vary from breath to breath; therefore, it is best to average 3–4 consecutive measurements for accuracy [17].

Measurements also differ slightly between ventilator brands and software algorithms. Some ventilators perform a true end-expiratory occlusion for 100 ms to measure P0.1 directly, while others only estimate it during the trigger phase of a breath. In the latter case, because modern ventilators trigger within < 50 ms, the airway pressure drop may not be fully captured, leading to underestimation of P0.1, especially in patients with strong inspiratory efforts [16].

End-expiratory occlusion pressure (Pocc)

The end-expiratory occlusion pressure (Pocc) is an indirect indicator of inspiratory effort, reflecting the negative pressure generated by the respiratory muscles (Pmus) during an occluded inspiratory attempt. It is measured by performing a brief end-expiratory hold for a single spontaneous breath, during which the patient attempts to inhale against a closed airway. The resulting fall in airway pressure corresponds to the force of the inspiratory muscles [19].

Because of its simplicity, Pocc serves as a practical bedside screening tool for detecting excessive inspiratory effort and potential lung stress. The inspiratory muscle pressure (Pmus) can be estimated from Pocc using the following formula [19]:

$$\text{Predicted Pmus} = -0.75 \times \text{Pocc}$$

In a study of patients with acute hypoxemic respiratory failure transitioning to PSV, Pocc < -8.4 cmH_2O was proposed to indicate high inspiratory effort and possible under-assistance, while Pocc > -5.7 cmH_2O suggested low effort or over-assistance [20]. Moderate values (approximately -6 to -8 cmH_2O) may therefore represent an adequate balance between patient effort and ventilator support, corresponding to the desirable range of inspiratory workload during assisted ventilation.

In early-phase mechanically ventilated patients (within the first 36 hours), a more negative Pocc value indicates greater inspiratory effort, as the patient must generate a greater subatmospheric pressure to initiate inspiration. When the predicted Pmus is less than -13 to -15 cmH_2O (equivalent to Pocc more negative than -17 to -20 cmH_2O), this reflects excessive inspiratory muscle activity and inadequate ventilatory assistance, potentially increasing the risk of P-SILI [19]. A Pocc value less negative than -7 cmH_2O reflects low effort and insufficient diaphragmatic activity [21].

It is important to distinguish Pocc from maximal inspiratory pressure (MIP), also known as negative inspiratory force (NIF). MIP measures inspiratory muscle strength (via maximal effort), whereas Pocc measures inspiratory effort during a normal, spontaneous breath. While MIP or NIF can be assessed at the bedside using ventilator functions, these tests are intended to assess inspiratory muscle strength and typically require patient cooperation to generate a truly maximal inspiratory effort [22]. In contrast, during pressure-support ventilation, when no coached maximal maneuver is requested, the NIF function available on some ventilators can be used to perform a brief end-expiratory occlusion (EEO) and obtain Pocc. In this context, the recorded value should be interpreted as an airway occlusion pressure reflecting the patient's effort under the current pressure-support level, rather than a true MIP/NIF measurement.

Clinically, a low MIP (less negative than −30 cmH_2O) has been linked with a higher risk of extubation failure and long-term mortality in ventilated patients [23].

Limitations:

Like P0.1, Pocc may fluctuate between breaths; averaging 3–4 measurements improves accuracy [19].

Not all ventilators permit a manual end-expiratory hold during PSV; in these cases, clinicians may use the ventilator's built-in NIF/MIP maneuver as an alternative occlusion method to obtain Pocc.

Pocc can serve as a quick, noninvasive screening tool to detect excess Pmus, but the predicted absolute values of the parameter remain imprecise and are not a substitute for direct monitoring (e.g., esophageal pressure) [6,10,19].

Pressure muscle index (PMI)

The pressure muscle index (PMI) is another indirect measure of inspiratory effort. During PSV, a brief end-inspiratory occlusion (typically 2–3 seconds) allows airway pressure to equilibrate once inspiratory flow falls to zero. If inspiratory muscle activity ceases during the hold, airway pressure stabilizes at a plateau (Pplat) that reflects the relaxed elastic recoil pressure of the respiratory system. In assisted breaths, Pplat can be higher or lower than the pre-occlusion peak airway pressure (Ppeak), depending on the patient's contribution [24]. PMI is defined as the difference between the relaxed plateau airway pressure (Pplat) and the peak airway pressure produced by the ventilator, expressed as

$$\text{PMI} = \text{Pplat} - \text{Ppeak},$$

PMI reflects the additional pressure attributable to the patient's inspiratory effort at the time of the occlusion, thus reflecting the contribution of the inspiratory muscles in generating pressure beyond ventilator assistance [9,25]. PMI correlates with effort indices derived from esophageal pressure, such as the pressure–time product (PTP), and offers a practical, noninvasive bedside estimate of inspiratory effort [24].

A PMI less than 0 cmH_2O, where the plateau pressure (Pplat) is below the peak inspiratory pressure (Ppeak), indicates minimal inspiratory effort and suggests ventilatory over-assistance [7,20]. A PMI between 0 and 2 cmH_2O represents an appropriate or normal effort range, typically observed during the transition from controlled to assisted ventilation [20]. Higher PMI values may indicate stronger inspiratory efforts, although the exact threshold defining excessive effort remains uncertain [9].

PMI correlates with predicted Pmus derived from Pocc, but it represents a static measurement that excludes resistive pressure losses; therefore, the two indices are related but not interchangeable [26].

Limitations:

Not all ventilators allow inspiratory holds during assisted ventilation.

Achieving a stable plateau can be difficult in patients with strong drive, poor synchrony, or active expiratory effort.

An unreadable plateau (fluctuating or unstable pressure trace) may indicate excessive respiratory effort [26].

To improve accuracy, perform multiple inspiratory hold maneuvers and verify each measurement's reliability by examining both the flow-time and pressure-time waveforms. Table 1 summarizes the recommended reliability criteria, time to reach plateau < 800 ms, plateau duration > 2 s, and plateau variation < 0.6 cmH_2O/s, along with additional practical considerations for accurate interpretation [26,27].

Table 1. Proposed criteria for inspiratory holds reliability [26].

Reliability criteria (all of the following are required)
1. Time to reach Pplat < 800 ms 2. Pplat lasts > 2 s (to determine that a plateau has been reached and to unmask respiratory effort) 3. Pplat varies < 0.6 cmH_2O/s
Consideration
• Pplat may be greater, equal to, or smaller than Ppeak • Check the air leak before PMI measurement • Observe the flow-time waveform during occlusion to confirm zero flow

Esophageal manometry

Esophageal pressure (Pes) monitoring provides a direct, bedside method to quantify inspiratory effort and work of breathing during assisted ventilation [14]. Because Pes closely reflects pleural pressure, it allows estimation of the pressure generated by respiratory muscles and the mechanical load on the lungs. In PSV, where the patient actively contributes to each breath, Pes measurements can help identify both insufficient effort (suggesting over-assistance) and excessive effort (indicating under-assistance or risk of patient self-inflicted lung injury) [14].

A key variable derived from Pes is the esophageal pressure swing (ΔPes)—the difference between end-expiratory and end-inspiratory Pes values:

$$\Delta Pes = Pes\_end\text{-}expiratory - Pes\_nadir$$

ΔPes reflects the magnitude of inspiratory muscle contraction. Very small swings ($\Delta Pes < 3–5$ cmH_2O) indicate minimal effort or over-assistance, while large swings ($\Delta Pes > 14–18$ cmH_2O) suggest excessive effort and potentially harmful transpulmonary pressures [9]. Intermediate ΔPes values (approximately 5–10 cmH_2O) are, in the main, considered physiologically appropriate, balancing diaphragmatic activation with ventilator unloading [14].

For most clinical uses, tracking ΔPes trends provides actionable information to titrate pressure support levels, maintain synchrony, and achieve lung- and diaphragm-protective ventilation.

Limitations:

- Requires specialized equipment (esophageal balloon catheter and monitoring system).
- Needs precise balloon positioning and careful calibration to obtain reliable signals.
- Demands technical expertise for accurate interpretation of Pes and ΔPes waveforms.
- Artifacts caused by coughing, swallowing, or patient movement can distort readings.

Despite these challenges, esophageal manometry remains the reference gold standard for assessing inspiratory effort and guiding PSV titration at the bedside [29].

Diaphragmatic measurement

Diaphragm electrical activity (EAdi)

The electrical activity of the diaphragm (EAdi) can be monitored with an electrode-corporated nasogastric tube (EAdi catheter). While this device is primarily used in a ventilation mode called Neurally Adjusted Ventilatory Assist (NAVA), the EAdi catheter can be used as a monitoring tool on its own. EAdi tracks respiratory drive and typically declines as ventilatory assistance increases [30]. It correlates with Pmus and helps identify asynchronies that may be overlooked on flow and pressure waveforms alone [31].

Peak EAdi, which represents the maximum amplitude of the diaphragm's electrical activity during inspiration, was 10–20 μV in non-intubated healthy volunteers [32]. However, since EAdi can differ significantly between individuals, tracking changes over time within the same patient is generally more informative than relying on a single, fixed measurement. As a result, interpreting trends in EAdi offers greater reliability than applying absolute thresholds [9,32].

In PSV, EAdi-based optimization strategies have been shown to enhance patient–ventilator interaction [33].

Limitations:

Wide inter-patient variability without validated thresholds in the critically ill [34]. The EAdi tool is currently available commercially on only one ventilator model and is technically difficult to use for reliable EAdi monitoring.

Diaphragm ultrasound

Diaphragm thickening fraction (TFdi) reflects diaphragm contractile activity and effort and is feasible and reproducible in ventilated patients [35]. Diaphragm excursion cannot be used to quantitatively assess diaphragm contractile activity during positive mechanical ventilation because it is influenced by passive displacement of the diaphragm [36]. In post-surgical patients on assisted breathing, TFdi inversely correlated with PS level, but values showed substantial inter-individual variability even under standardized settings [36].

TFdi correlates poorly with transdiaphragmatic pressure (Pdi), the pressure difference between the abdomen and thorax that reflects the true diaphragmatic contractile effort, in mechanically ventilated patients. Therefore, absolute TFdi values should not be used as stand-alone markers of inspiratory effort during PSV. This limitation arises from the one-dimensional nature of ultrasound measurement within the diaphragm's zone of apposition as well as confounders such as hyperinflation, pleural effusions, abdominal hypertension, and obesity [37]. TFdi is better suited for monitoring within-patient trends, such as changes in diaphragmatic activity in response to adjustments in pressure-support levels, rather than for quantitative comparisons between patients.

Limitations:

Operator dependence, anatomic and mechanical confounding, and limited validity of absolute thresholds in critical illness.

ADEQUATE PSV ASSISTANCE

Achieving an optimal balance between patient effort and ventilatory support remains difficult, and definitive target values across these monitoring modalities are uncertain. Recent evidence supports aiming for an intermediate range of inspiratory effort, with avoidance of excessive driving transpulmonary pressures and large tidal volumes, a concept referred to as "adequate PSV assistance" [7].

Typically, increasing the level of PS raises VT and decreases patient effort [38]. Conversely, reducing PS does not always lead to a proportional decrease in VT, as some patients can compensate by increasing Pmus to maintain a target VT [7,39]. However, if a patient cannot augment Pmus due to muscle weakness or fatigue, VT falls, and PMI remains low.

Table 2. Overview of techniques for assessing inspiratory drive and effort at the bedside.

Technique	Parameter / Method	Clinical feasibility	Reference values (context)	Main advantages	Key limitations
Esophageal manometry	ΔPes (esophageal pressure swing)	Specialized	Critically ill adults on assisted ventilation [9]: • Low effort/over-assist: ΔPes < 3–5 cmH$_2$O • High effort/under-assist: ΔPes > 14–18 cmH$_2$O	Reference signal for inspiratory effort; allows trending; reflects transpulmonary pressure (PL)	Does not account for chest-wall load; requires esophageal balloon
Diaphragm electrical activity (EAdi)	Peak EAdi (µV)	Specialized	Proposed normal range on PSV: 5–15 µV [34]	Tracks neural drive; detects asynchrony; can guide pressure support titration	High inter-individual variability; limited device availability and setup complexity
Expiratory occlusion	P0.1	Routine	Adult ICU (PSV) studies: • Low drive: < 1.0 cmH$_2$O • High drive: > 3.5–4.0 cmH$_2$O	Rapid surrogate of respiratory drive; correlates with inspiratory effort	Inter-breath variability; ventilator algorithm differences; automated values may underestimate true P0.1
	Pocc	Routine	Transition to PSV [20]: • Low effort: > −5.7 cmH$_2$O • High effort: < −8.4 cmH$_2$OAcute respiratory failure[19,21] : • Low effort: > -7 cmH$_2$O • High effort: < −17 to −20 cmH$_2$O	Simple bedside screen for excessive effort; can estimate Pmus (≈ −¾ × Pocc)	Substantial inter-individual variability
Inspiratory occlusion	PMI	Routine	Transition to PSV: • Low effort < 0 cmH$_2$O • Normal 0–2 cmH$_2$O • High effort = no established cut-off [10]	Screens for low effort and over-assistance; informs about driving pressure and compliance	Some ventilators lack inspiratory-hold function; plateau difficult in strong effort; affected by expiratory muscles; multiple holds needed for reliability
Diaphragm ultrasound	TFdi	Specialized	No validated absolute threshold on PSV [36]	Useful for within-patient monitoring of support changes	Operator-dependent; inter-patient variability; poor correlation with Pdi; excludes extra-diaphragmatic effort

Abbreviations: ΔPes: esophageal pressure swing; PL: transpulmonary pressure; EAdi: electrical activity of the diaphragm; P0.1: airway occlusion pressure at 100 milliseconds; Pocc: end-expiratory occlusion pressure; Pmus: inspiratory muscle pressure; PMI: pressure muscle index; Pplat: plateau airway pressure; Ppeak: peak airway pressure; TFdi: diaphragm thickening fraction; Pdi: transdiaphragmatic pressure; PSV: pressure-support ventilation; ICU: intensive care unit.

Building on these observations, Docci et al.[7] described a nonlinear interaction among PS, Pmus, and VT, defining a range bounded by under-assistance (PSunder) and over-assistance (PSover), within which patients modulate Pmus to maintain VT (and thus ΔP) near a target level (VTtarget) (Figure 3).

Over-assistance

When PS exceeds PSover, patient effort declines to minimal levels, respiratory drive falls, inspiratory time shortens, and respiratory rate decreases. VT then depends mainly on PS and compliance, increasing nearly linearly with higher support.

Under-assistance

When PS drops below PSunder, the patient cannot sustain VT despite increased effort. VT decreases, respiratory rate rises, and accessory muscle recruitment becomes more intense. The PS threshold for under-assistance varies with compliance, resistance, metabolic demand, and respiratory muscle capacity.

Bedside recognition

- Adequate zone: VT remains stable across modest PS changes; PMI > 0 but < excessive; RR is steady.
- Over-assistance: PMI nearly absent, RR low, VT mainly PS-driven.
- Under-assistance: PMI rises, RR and P0.1 increase, accessory muscles are active, and CO$_2$ retention or diaphoresis may appear.

However, the precise “optimal” level of patient effort within the adequate zone remains to be defined.

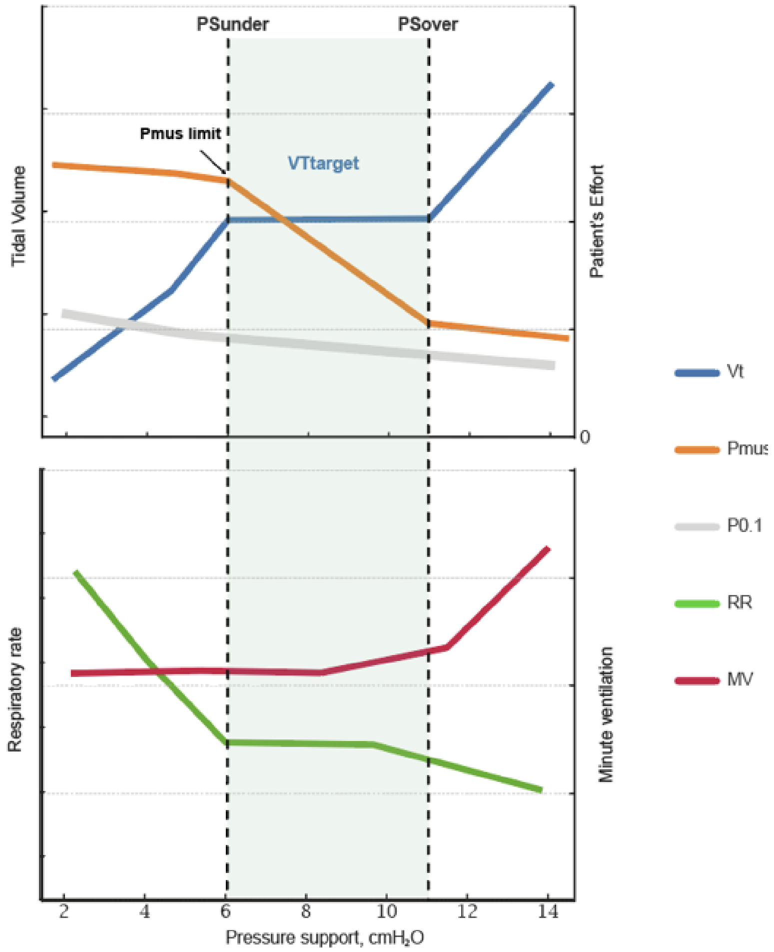
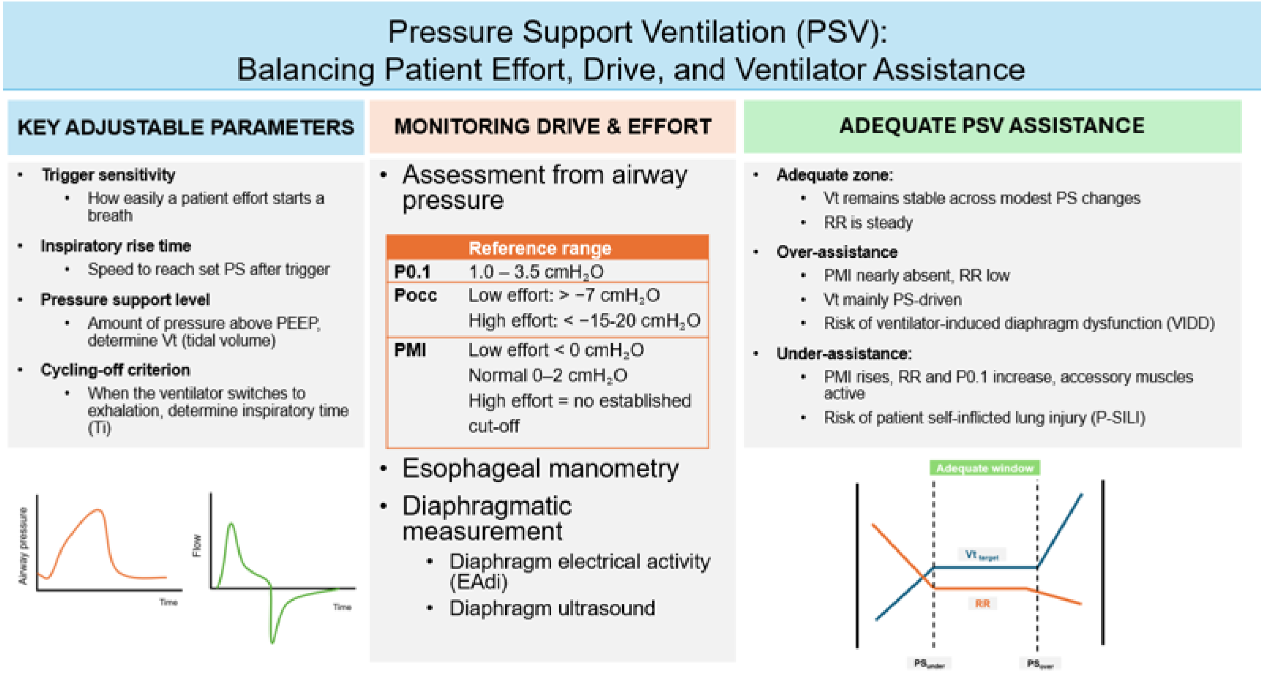


Figure 3. Relationship between pressure support level, patient effort, and tidal volume during PSV ("Adequate PSV assistance" concept). Tidal volume depends on pressure support, the patient's inspiratory effort (Pmus), and compliance. Three phases are identified: under-assistance, adequate assistance, and over-assistance. In the adequate zone (between PSunder and PSover), the patient adjusts Pmus to maintain VT near VTtarget. If the effort required to achieve VTtarget exceeds the sustainable Pmus limit, under-assistance occurs, accompanied by increases in P0.1 and respiratory rate to maintain minute ventilation. When PS = 0, VT reflects patient effort and compliance; at high PS > PSover, patient effort becomes minimal, and VT depends mainly on ventilator support and compliance. Adapted from Docci et al.[7]



Pressure Support Ventilation (PSV):
Balancing Patient Effort, Drive, and Ventilator Assistance

KEY ADJUSTABLE PARAMETERS

- **Trigger sensitivity**
  - How easily a patient effort starts a breath
- **Inspiratory rise time**
  - Speed to reach set PS after trigger
- **Pressure support level**
  - Amount of pressure above PEEP, determine Vt (tidal volume)
- **Cycling-off criterion**
  - When the ventilator switches to exhalation, determine inspiratory time (Ti)

MONITORING DRIVE & EFFORT

- Assessment from airway pressure

	Reference range
P0.1	1.0 – 3.5 cmH$_2$O
Pocc	Low effort: > −7 cmH$_2$O High effort: < −15-20 cmH$_2$O
PMI	Low effort < 0 cmH$_2$O Normal 0–2 cmH$_2$O High effort = no established cut-off

- Esophageal manometry
- Diaphragmatic measurement
  - Diaphragm electrical activity (EAdi)
  - Diaphragm ultrasound

ADEQUATE PSV ASSISTANCE

- **Adequate zone:**
  - Vt remains stable across modest PS changes
  - RR is steady
- **Over-assistance**
  - PMI nearly absent, RR low
  - Vt mainly PS-driven
  - Risk of ventilator-induced diaphragm dysfunction (VIDD)
- **Under-assistance:**
  - PMI rises, RR and P0.1 increase, accessory muscles active
  - Risk of patient self-inflicted lung injury (P-SILI)

Adapted from Docci et al., Critical Care 2024; 28:358 and Tonelli et al., Critical Care 2025; 29:339.

Figure 4. Graphical overview of key adjustable settings in pressure-support ventilation (trigger sensitivity, inspiratory rise time, pressure-support level, and cycling-off criterion), with representative pressure- and flow-time waveforms, methods to monitor respiratory drive and effort, and the concept of adequate PSV assistance.

CLINICAL APPLICATION

We propose a stepwise approach to PSV titration that integrates concepts of adequate PSV assistance and drive/effort monitoring. Figure 4 provides a graphical summary of the key settings and monitoring tools discussed in this review.

Step 1: Establish baseline PSV and optimize interaction

- Set FiO_2/PEEP to meet oxygenation goals. Initiate PS at a modest level to achieve an acceptable breathing pattern (VT, RR, minute ventilation), then optimize trigger sensitivity, rise time, and cycling-off.

Step 2: Screen for under-assistance and excessive effort

- Treat reversible causes of high drive and effort (pain, fever, acidosis, anxiety).
- Use P0.1 as a rapid screen and obtain Pocc when feasible.
  - If P0.1 > 3.5–4.0 cmH_2O and/or Pocc < −8.4 cmH_2O, this suggests high drive/effort; may consider incremental increase PS by 2–4 cmH_2O, re-check synchrony settings, and reassess.

Step 3: Avoid over-assistance and identify the "adequate PSV assistance" zone

- If findings suggest over-assistance, such as P0.1 < 1.0–1.5 cmH_2O, Pocc > −5.7 cmH_2O (minimally negative occlusion pressure), and/or PMI < 0 cmH_2O (if an interpretable end-inspiratory hold is feasible), decrease PS in small steps (≈ 2 cmH_2O) and reassess until the patient's respiratory status is within the acceptable assistance zone.

Step 4: Consider advanced measurements or alternative modes

- When airway measurements are not suitable or when precise quantification of inspiratory effort and lung stress is needed, consider an advanced option such as esophageal manometry, EAdi monitoring, and/or diaphragm ultrasound.

Consider proportional modes (e.g., NAVA or PAV+) if available and appropriate; they may improve matching of ventilator assistance to patient demand and enhance synchrony in selected patients.

PROPORTIONAL MODES OF VENTILATION: COMPARISON WITH NAVA AND PAV+

In PSV, each breath delivers a fixed pressure regardless of the patient's effort or ventilatory demand. Proportional modes, NAVA, and proportional assist ventilation with load-adjustable gain factors (PAV+) address these limitations by scaling assistance in real time to the patient's inspiratory effort, improving synchrony and adapting to changing load conditions [40,41]. However, limited data suggest improved clinical outcomes with proportional modes as compared to conventional modes; therefore, ongoing physiology-guided monitoring and titration remain essential [40-42]. Key operational and clinical distinctions among PSV, NAVA, and PAV+ are summarized in Table 3.

Table 3. Summary Comparison of PSV, NAVA, and PAV+

Aspect	PSV	NAVA	PAV+
Triggering	Flow or pressure	Neural (EAdi rise ≥0.5 μV)	Flow or pressure
Assistance mechanism	Fixed pressure, independent of effort	Paw = NAVA level × EAdi	Computed from equation of motion; ventilator provides % gain of total load
Adjustment to demand	Constant each breath	Scales with neural drive	Scales with mechanical load and patient effort
Cycling	Flow-based	Neural (≈70% of peak EAdi)	Flow-based
Synchrony vs PSV (Invasive ventilation)	Reference mode	↓ Asynchrony (AI > 10% RR 0.20, 95% CI 0.05–0.92) [42]	↓ Asynchrony (AI > 10% RR 0.06, 95% CI 0.01–0.46) [42]
Duration of MV vs PSV (Invasive ventilation)	Reference	↑ VFDs (+3.4 days, 95% CI 1.2–5.6) [45]	No difference in liberation time (RR 0.96, 95% CI 0.8–1.15) [44]
Advantages	Simple, familiar	Tracks neural drive; limits prolonged inspiration	Balances load between patient and ventilator
Limitations	Risk of over/under-assistance	Requires reliable EAdi; ineffective with absent drive	Risk of "runaway" or under-assist with leaks/low drive; complex setup

Abbreviations: PS: pressure support; Paw: airway pressure; EAdi: electrical activity of the diaphragm; Ti: inspiratory time; VT: tidal volume; AI: asynchrony index; WMD: weighted mean difference; MD: mean difference; RR: risk ratio; MV: mechanical ventilation; Pmus: inspiratory muscle pressure; PEEPi: intrinsic PEEP

Neurally adjusted ventilatory assist (NAVA)

NAVA aids proportional to the electrical activity of the diaphragm (EAdi), a surrogate of neural drive. The ventilator triggers, delivers, and cycles breaths according to the EAdi signal, with airway pressure determined by the NAVA level ($cmH_2O/\mu V$) [38,43].

Proportional assist ventilation (PAV+)

PAV+ estimates patient load using brief occlusion maneuvers to measure respiratory resistance and elastance, then delivers a clinician-set proportion of the total inspiratory pressure (gain factor, e.g., 75%) while the patient provides the rest [40,44].

CONCLUSION

Pressure-support ventilation remains a commonly used mode of invasive mechanical ventilation, particularly during the weaning phase. Physiology-guided, dynamic titration based on the patient's respiratory drive and effort is preferable to a fixed pressure-support level. Clinicians should aim to keep patients within an appropriate assistance range, avoid under-assistance, over-assistance, and patient–ventilator dyssynchrony. Numerous bedside monitoring tools beyond routine clinical and ventilatory parameters are available and should be integrated into care, as they may help optimize patient outcomes. Proportional modes may enhance synchrony, although evidence for faster liberation from mechanical ventilation is mixed. Future studies should prospectively validate titration targets across monitoring modalities and whether physiology-guided PSV titration enhances patient-centered outcomes.

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REFERENCES

1. Brochard LJ, Lellouche F. Chapter 8. Pressure-Support Ventilation. In: Tobin MJ, editor. Principles and Practice of Mechanical Ventilation, 3e. New York, NY: The McGraw-Hill Companies; 2013.
2. MacIntyre NR. Respiratory function during pressure support ventilation. Chest. 1986;89:677-83.
3. Esteban A, Ferguson ND, Meade MO, Frutos-Vivar F, Apezteguia C, Brochard L, et al. Evolution of mechanical ventilation in response to clinical research. Am J Respir Crit Care Med. 2008;177:170-7.
4. van Haren F, Pham T, Brochard L, Bellani G, Laffey J, Dres M, et al. Spontaneous breathing in early acute respiratory distress syndrome: Insights from the large observational study to UN derstand the global impact of severe acute respiratory failure study. Crit Care Med. 2019;47:229-38.
5. Telias I, Jonkman A, Rittayamai N. Assisted Ventilation: Pressure Support and Bilevel Ventilation Modes. In: Bellani G, editor. Mechanical Ventilation: From Pathophysiology to Clinical Evidence. 1st ed. Cham: Springer International Publishing; 2022. p 49-59.
6. Goligher EC, Jonkman AH, Dianti J, Vaporidi K, Beitler JR, Patel BK, et al. Clinical strategies for implementing lung and diaphragm-protective ventilation: avoiding insufficient and excessive effort. Intensive Care Med. 2020;46:2314-26.
7. Docci M, Foti G, Brochard L, Bellani G. Pressure support, patient effort and tidal volume: a conceptual model for a non linear interaction. Critical Care. 2024;28:358.
8. Vassilakopoulos T, Petrof BJ. Ventilator-induced diaphragmatic dysfunction. Am J Respir Crit Care Med. 2004;169:336-41.
9. Tonelli R, Protti A, Spinelli E, Grieco DL, Yoshida T, Jonkman AH, et al. Assessing inspiratory drive and effort in critically ill patients at the bedside. Critical Care. 2025;29.
10. van Oosten JP, Akoumianaki E, Jonkman AH. Monitoring respiratory muscles effort during mechanical ventilation. Current Opinion in Critical Care. 2025;31.
11. Theerawit P, Soipetkasem P. An importance of respiratory drive and effort during mechanical ventilation. Clinical Critical Care. 2023. http://dx.doi.org/10.54205/ccc.v31.259350.
12. Goligher EC, Dres M, Patel BK, Sahetya SK, Beitler JR, Telias I, et al. Lung- and diaphragm-protective ventilation. Am J Respir Crit Care Med. 2020;202:950-61.
13. Vaporidi K, Akoumianaki E, Telias I, Goligher EC, Brochard L, Georgopoulos D. Respiratory drive in critically ill patients. Pathophysiology and clinical implications. Am J Respir Crit Care Med. 2020;201:20-32.
14. Bertoni M, Spadaro S, Goligher EC. Monitoring patient respiratory effort during mechanical ventilation: Lung and diaphragm-protective ventilation. Crit Care. 2020;24:106.
15. Telias I, Junhasavasdikul D, Rittayamai N, Piquilloud L, Chen L, Ferguson ND, et al. Airway occlusion pressure as an estimate of respiratory drive and inspiratory effort during assisted ventilation. Am J Respir Crit Care Med. 2020;201:1086-98.
16. Telias I, Damiani F, Brochard L. The airway occlusion pressure (P(0.1)) to monitor respiratory drive during mechanical ventilation: increasing awareness of a not-so-new problem. Intensive Care Med. 2018;44:1532-5.
17. Consalvo S, Accoce M, Telias I. Monitoring and modulating respiratory drive in mechanically ventilated patients. Curr Opin Crit Care. 2025;31:30-7.
18. Rittayamai N, Beloncle F, Goligher EC, Chen L, Mancebo J, Richard J-CM, et al. Effect of inspiratory synchronization during pressure-controlled ventilation on lung distension and inspiratory effort. Annals of Intensive Care. 2017;7.
19. Bertoni M, Telias I, Urner M, Long M, Del Sorbo L, Fan E, et al. A novel non-invasive method to detect excessively high respiratory effort and dynamic transpulmonary driving pressure during mechanical ventilation. Critical Care. 2019;23.
20. Yang YL, Liu Y, Gao R, Song DJ, Zhou YM, Miao MY, et al. Use of airway pressure-based indices to detect high and low inspiratory effort during pressure support ventilation: a diagnostic accuracy study. Ann Intensive Care. 2023;13:111.
21. de Vries HJ, Tuinman PR, Jonkman AH, Liu L, Qiu H, Girbes ARJ, et al. Performance of noninvasive airway occlusion maneuvers to assess lung stress and diaphragm effort in mechanically ventilated critically ill patients. Anesthesiology. 2023;138:274-88.
22. Medrinal C, Prieur G, Combret Y, Quesada AR, Bonnevie T, Gravier FE, et al. Reliability of respiratory pressure measurements in ventilated and non-ventilated patients in ICU: an observational study. Ann Intensive Care. 2018;8:14.
23. Medrinal C, Prieur G, Frenoy É, Robledo Quesada A, Poncet A, Bonnevie T, et al. Respiratory weakness after mechanical ventilation is associated with one-year mortality - a prospective study. Crit Care. 2016;20:231.
24. Foti G, Cereda M, Banfi G, Pelosi P, Fumagalli R, Pesenti A. End-inspiratory airway occlusion: a method to assess the pressure developed by inspiratory muscles in patients with acute lung injury undergoing pressure support. Am J Respir Crit Care Med. 1997;156:1210-6.
25. Bellani G, Grassi A, Sosio S, Foti G. Plateau and driving pressure in the presence of spontaneous breathing. Intensive Care Medicine. 2019;45:97-8.
26. Bianchi I, Grassi A, Pham T, Telias I, Teggia Droghi M, Vieira F, et al. Reliability of plateau pressure during patient-triggered assisted ventilation. Analysis of a multicentre database. J Crit Care. 2022;68:96-103.
27. Soundoulounaki S, Akoumianaki E, Kondili E, Pediaditis E, Prinianakis G, Vaporidi K, et al. Airway pressure morphology and respiratory muscle activity during end-inspiratory occlusions in pressure support ventilation. Crit Care. 2020;24:467.
28. Kyogoku M, Shimatani T, Hotz JC, Newth CJL, Bellani G, Takeuchi M, et al. Direction and magnitude of change in plateau from peak pressure during inspiratory holds can identify the degree of spontaneous effort and elastic workload in ventilated patients. Crit Care Med. 2021;49:517-26.
29. Dianti J, Fard S, Wong J, Chan TCY, Del Sorbo L, Fan E, et al. Strategies for lung- and diaphragm-protective ventilation in acute hypoxemic respiratory failure: a physiological trial. Critical Care. 2022;26:259.
30. Beck J, Gottfried SB, NavalesI P, Skrobik Y, Comtois N, Rossini M, et al. Electrical activity of the diaphragm during pressure support ventilation in acute respiratory failure. American Journal of Respiratory and Critical Care Medicine. 2001;164:419-24.
31. Colombo D, Cammarota G, Alemani M, Carenzo L, Barra FL, Vaschetto R, et al. Efficacy of ventilator waveforms observation in detecting patient-ventilator asynchrony. Crit Care Med. 2011;39:2452-7.
32. Piquilloud L, Beloncle F, Richard J-CM, Mancebo J, Mercat A, Brochard L. Information conveyed by electrical diaphragmatic activity during unstressed, stressed and assisted spontaneous breathing: a physiological study. Annals of Intensive Care. 2019;9.
33. Beloncle F, Piquilloud L, Rittayamai N, Sinderby C, Roze H, Brochard L. A diaphragmatic electrical activity-based optimization strategy during pressure support ventilation improves synchronization but does not impact work of breathing. Crit Care. 2017;21:21.

34. Di Mussi R, Spadaro S, Volta CA, Bartolomeo N, Trerotoli P, Staffieri F, et al. Continuous assessment of neuro-ventilatory drive during 12 h of pressure support ventilation in critically ill patients. Crit Care. 2020;24:652.
35. Goligher EC, Laghi F, Detsky ME, Farias P, Murray A, Brace D, et al. Measuring diaphragm thickness with ultrasound in mechanically ventilated patients: feasibility, reproducibility and validity. Intensive Care Medicine. 2015;41:642-9.
36. Umbrello M, Formenti P, Longhi D, Galimberti A, Piva I, Pezzi A, et al. Diaphragm ultrasound as indicator of respiratory effort in critically ill patients undergoing assisted mechanical ventilation: a pilot clinical study. Critical Care. 2015;19.
37. Poulard T, Bachasson D, Fosse Q, Nierat MC, Hogrel JY, Demoule A, et al. Poor correlation between diaphragm thickening fraction and transdiaphragmatic pressure in mechanically ventilated patients and healthy subjects. Anesthesiology. 2022;136:162-75.
38. Carteaux G, Cordoba-Izquierdo A, Lyazidi A, Heunks L, Thille AW, Brochard L. Comparison between neurally adjusted ventilatory assist and pressure support ventilation levels in terms of respiratory effort. Crit Care Med. 2016;44:503-11.
39. Docci M, Rezoagli E, Teggia-Droghi M, Coppadoro A, Pozzi M, Grassi A, et al. Individual response in patient's effort and driving pressure to variations in assistance during pressure support ventilation. Ann Intensive Care. 2023;13:132.
40. Jonkman AH, Rauseo M, Carteaux G, Telias I, Sklar MC, Heunks L, et al. Proportional modes of ventilation: technology to assist physiology. Intensive Care Med. 2020;46:2301-13.
41. Schmidt M, Kindler F, Cecchini J, Poitou T, Morawiec E, Persichini R, et al. Neurally adjusted ventilatory assist and proportional assist ventilation both improve patient-ventilator interaction. Crit Care. 2015;19:56.
42. Kataoka J, Kuriyama A, Norisue Y, Fujitani S. Proportional modes versus pressure support ventilation: a systematic review and meta-analysis. Ann Intensive Care. 2018;8:123.
43. Patroniti N, Bellani G, Saccavino E, Zanella A, Grasselli G, Isgrò S, et al. Respiratory pattern during neurally adjusted ventilatory assist in acute respiratory failure patients. Intensive Care Medicine. 2012;38:230-9.
44. Bosma KJ, Burns KEA, Martin CM, Skrobik Y, Mancebo Cortes J, Mulligan S, et al. Proportional-assist ventilation for minimizing the duration of mechanical ventilation. N Engl J Med. 2025;393:1088-103.
45. Pinto CB, Leite D, Brandão M, Nedel W. Clinical outcomes in patients undergoing invasive mechanical ventilation using NAVA and other ventilation modes - A systematic review and meta-analysis. Journal of Critical Care. 2023;76:154287.