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Towards optimal Patient-Ventilator Interaction: The interacting signals

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Abstract

Background

Patient-ventilator interaction (PVI) emerges from the continuous interaction between physiological signals generated by the patient and signals generated, measured, or displayed by the mechanical ventilator. Traditionally, PVI has been described predominantly through recognizable forms of patient-ventilator asynchrony. Although this approach remains clinically useful, the visible waveform abnormalities represent the final expression of interactions occurring between multiple neural, muscular, mechanical and ventilator signals.

Methods

This conceptual narrative review builds on contemporary PVI taxonomies by organizing bedside analysis around the relationships between selected patient and ventilator signals. Patient-derived physiological signals may reflect neural respiratory activity, respiratory muscle activation, and respiratory muscle pressure, whereas ventilator-derived signals include mechanical breath onset, assistance delivery, cycling-off, and ventilator-measured airway pressure, flow and volume.

Rather than introducing new PVI categories, the framework uses four sequential relational questions: whether expected patient and ventilator signals correspond; whether their inspiratory onsets are appropriately aligned; whether their inspiratory offsets are appropriately aligned; and whether the magnitude of ventilator-delivered assistance is appropriate for the intended physiological target. Loss of expected correspondence produces abnormalities such as failed triggering and unintended (false) triggering. Abnormal temporal relationships between corresponding inspiratory onsets produce early or late triggering, whereas abnormal offset relationships produce early or late cycling. Appropriate timing, however, does not guarantee appropriate assistance: under-assistance and over-assistance represent abnormalities in the magnitude of ventilator contribution relative to patient respiratory demand and the intended unloading goal.

The same signal-identification approach is applied across the expiratory phase, where active expiratory muscle contraction, expiratory-flow deformation, persistence of inspiratory activity after cycling-off, and expiratory muscle relaxation may alter waveform appearance and influence subsequent triggering. Directionality is treated as an interpretive modifier when temporal sequence alone is insufficient to establish the underlying mechanism. Reverse triggering illustrates this distinction, demonstrating that the interaction may proceed from the ventilator to the patient rather than from the patient to the ventilator.

Conclusions

A stepwise bedside algorithm is proposed to translate this signal-based framework into practical waveform interpretation. Understanding which signals are interacting, how they correspond, how their timing and magnitude relate, and how interactions propagate across respiratory phases provides a physiological path toward more precise interpretation of PVI and ultimately towards optimal patient-ventilator interaction.

Keywords: Patient-Ventilator Interaction; Patient-Ventilator Asynchrony; Respiratory Signals; Mechanical Ventilation.

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Introduction

During assisted mechanical ventilation, respiration is generated neither by the patient nor by the ventilator alone. Each breath emerges from the interaction between the patient's respiratory drive, respiratory muscle activity, respiratory system mechanics, and the mechanical ventilator settings. The patient generates physiological signals beginning with respiratory drive and neural respiratory output, followed by respiratory muscle activation and pressure generation. These processes ultimately modify pressure, flow, and volume within the respiratory system. In parallel, the ventilator detects triggering variables, processes those signals, initiates mechanical inspiration, delivers pressure and flow, and terminates inspiration according to its cycling-off criteria. The mechanically assisted breath is therefore the product of interacting signals.

Patient-ventilator interaction has traditionally been evaluated by identifying discrete forms of patient-ventilator asynchrony, including failed triggering, auto-triggering, late triggering, early cycling, late cycling, and double triggering. This terminology has provided an essential language for clinical practice and research. More recent PVI taxonomies have further emphasized the importance of standardized definitions and explicit identification of patient and ventilator signals. [1-6]

Relationship to existing taxonomies

The framework presented in this review is not intended to replace, compete with, or redefine contemporary taxonomies of patient-ventilator interaction. Mireles-Cabodevila et al. have proposed a comprehensive taxonomy organized around explicit patient and ventilator signal identification and standardized waveform interpretation. [1,2]

The present review adopts the same foundational principles but reorganizes the resulting abnormalities into four explicit relational domains: correspondence, onset alignment, offset alignment, and magnitude alignment, and extends this relational framework systematically into the expiratory phase and into the question of interaction directionality (patient to ventilator versus ventilator to patient). The intended contribution is therefore one of conceptual reorganization and extension, particularly expiratory-phase signal interactions which are comparatively underrepresented in prior taxonomic work. Accordingly, failed triggering, late triggering, early cycling, over-assistance, active expiration, and reverse triggering are not proposed as new phenomena. Rather, the intended contribution is analytical and translational rather than taxonomic, providing a signal-centered framework that connects physiological measurements and ventilator waveforms with established PVI phenotypes, while emphasizing reference-signal dependence, cross-phase propagation, and the possibility that similar waveform abnormalities may arise from different physiological mechanisms.

What this framework adds

The framework makes four organizational contributions. First, established PVI abnormalities are approached through explicit relationships between signals rather than phenotype alone. Second, timing interpretation is made explicitly reference-signal dependent, separating measured intervals from judgment that an interval is abnormal. Third, the framework follows abnormalities across inspiratory-expiratory boundary allowing an abnormality originating in one phase to explain signals observed in another. Fourth, directionality is used as an interpretive modifier when the order of events does not by itself establish whether the patient or ventilator initiated the interaction.

Signals, waveforms and reference points

Before describing the two interacting systems, a few terms require precise definition because they recur throughout this review. A signal is a physiological or mechanical variable that changes over time (e.g., diaphragm electrical activity, esophageal pressure, airway flow). A waveform is the graphical representation of that signal (e.g., the flow-time tracing displayed on a ventilator screen). A reference point is an identifiable event on the signal, such as inspiratory onset or inspiratory offset. An interval is the measured temporal difference between corresponding reference points (e.g., trigger delay). Correspondence refers to the presence of an expected paired event, i.e., whether an intended patient inspiratory signal is associated with an appropriately paired ventilator inspiratory signal, independent of timing or magnitude of that pairing, which are assessed separately.
These distinctions matter because clinical statements about PVI often conflate a signal with its waveform representation, or a measured interval. This review separates measurements from interpretation throughout.

Patient-Ventilator interaction: Two systems, multiple signals

The physiological and mechanical components of patient-ventilator interaction can be conceptualized as two parallel, dynamically coupled pathways (**Figure** 1). The patient pathway begins with central respiratory drive, which generates neural respiratory output and subsequent activation of the respiratory muscles. This activation produces respiratory muscle pressure, resulting in measurable changes in airway pressure, flow and volume.

In parallel, the ventilator pathway begins with acquisition of a trigger input, followed by detection and processing of the triggering signals. Once the predefined triggering criterion is reached, the ventilator initiates mechanical inspiration and delivers pressure and flow, resulting in tidal volume delivery. Inspiration continues until the cycling-off criterion is reached, after which the ventilator transitions to the expiratory phase (Figure 1).

The signals measured at different points along these pathways do not represent the same physiological event. Neural activation precedes muscle force generation; muscle force generation precedes or modifies changes in airway pressure and flow; ventilator recognition of a triggering variable occurs before full development of mechanical pressurization. Thus, PVI cannot be interpreted adequately without specifying which signals are being compared.

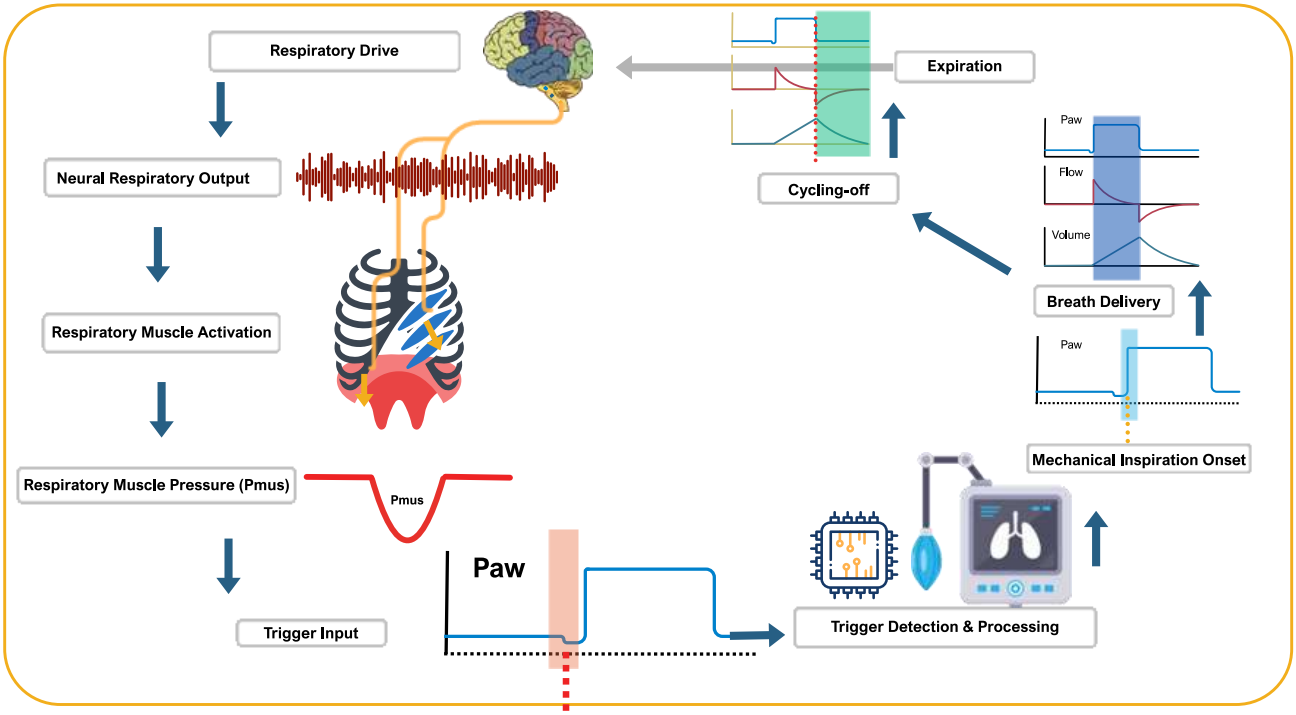

Figure 1: Two systems, multiple signals.

Respiratory drive, activation, effort, and capacity

Several related physiological concepts are important when interpreting patient signals. Respiratory drive refers to the neural output of the respiratory center. Respiratory muscle activation describes recruitment of the respiratory muscles in response to neural command. Respiratory effort refers to the mechanical pressure generated by those muscles. Respiratory muscle capacity determines how much pressure can be generated for a given level of neural activation.

These relationships are important during magnitude assessment because high respiratory drive does not invariably translate into proportionally high respiratory muscle pressure, particularly in patients with neuromuscular dysfunction or severe respiratory muscle weakness. Bedside signals therefore interrogate different levels of this pathway. EAdi primarily reflects neural activation; P0.1 provides a practical estimate of respiratory drive; and esophageal pressure and derived muscular pressure (Pmus) provide mechanical estimates of effort. [7,8]

The reference signal

Every assessment of alignment requires a reference. A reference signal is a physiological or mechanical signal used as the baseline for measuring, comparing, and evaluating the timing and characteristics of patient-ventilator interaction.

The choice of reference signal is fundamental because different signals identify different physiological events. For example, the onset of diaphragm electrical activity, the onset of esophageal-pressure deflection, and the first detectable perturbation in airway flow may occur at different times. Consequently, a statement such as "trigger delay was 150 milliseconds" is incomplete unless the patient reference signal

used to establish inspiratory onset is specified. The reference signal is therefore not simply a technical detail; it determines how the interaction is measured and interpreted.

Patient signals

A patient signal is a patient-derived physiological signal used to identify respiratory muscle activity, including its onset, offset, and, where applicable, magnitude.

Neural respiratory activity

Electrical activity of the diaphragm (EAdi) provides information relatively close to neural activation of the diaphragm. Its onset can identify neural inspiratory onset, while termination of activity provides information regarding neural inspiratory termination. Because electrical activation occurs upstream from mechanical pressure generation, EAdi may identify respiratory activity before substantial mechanical consequences become evident in airway pressure and flow waveforms. EAdi is therefore particularly useful when the principal question concerns neural timing.

Respiratory muscle pressure

Respiratory muscle contraction generates pressure. Esophageal pressure provides an estimate of pleural pressure changes, whereas respiratory muscle pressure can be derived by considering the pressure required to move the passive respiratory system. Mechanical pressure signals provide information not only regarding inspiratory timing but also regarding the magnitude of respiratory effort. A patient may exhibit neural respiratory activation while generating little mechanical pressure because of respiratory muscle weakness; conversely, substantial pressure may be generated when respiratory system loading is high. Neural activation and respiratory effort should therefore not be treated as interchangeable concepts. [3,7,8]

Patient activity inferred from airway pressure and flow

In routine clinical practice, direct measurements of neural activity or respiratory muscle pressure are frequently unavailable. Patient activity is then inferred from alterations in airway pressure or flow. These signals can provide valuable information regarding the approximate onset and offset of respiratory muscle activity. [4,9] Changes in ventilator-measured airway pressure or flow that indicate patient respiratory activity should be considered as indirect indicators of patient activity rather than direct patient-derived physiological signals.

Their appearance depends on magnitude of patient effort, respiratory system mechanics, intrinsic PEEP, ventilator mode and settings.

Ventilator signals

A ventilator signal is a time-varying signal generated, measured, calculated, or displayed by the ventilator and used to characterize ventilator-delivered assistance.

The ventilator waveform is the graphical representation of such a signal over time. Pressure is the signal; the pressure-time curve displayed on the screen is its waveform. Similarly, flow is the signal, and the flow-time tracing is its waveform. PVI assessment therefore concerns the relationship between physiological and ventilator signals, while waveforms are one method through which those relationships are visualized.

Four relational questions for bedside interpretation

Once the relevant patient and ventilator signals, and reference points have been identified, bedside interpretation can proceed through four relational questions (Table 1). These questions are not proposed as a new category of PVI; they provide a structured sequence for analyzing established abnormalities.

Signal correspondence: Do the expected patient and ventilator signals correspond?

Inspiratory-onset alignment: What is the temporal relationship between patient inspiratory-signal onset and ventilator inspiratory-signal onset?

Inspiratory-offset alignment: What is the temporal relationship between patient inspiratory-signal offset and ventilator inspiratory-signal offset?

Magnitude alignment: Is ventilator-delivered assistance appropriate relative to patient respiratory demand and the intended unloading target?

These relationships describe different components of PVI and should not be conflated. A breath may show appropriate correspondence and timing while still exhibiting an inappropriate magnitude relationship. Conversely, an abnormal timing relationship may occur despite an otherwise appropriate amount of assistance.

Table 1: Operational definitions for the interacting-signals framework.

Concept	Operational definition / question	Interpretation
Signal correspondence	Are the expected paired patient and ventilator events both present?	Presence or loss of expected correspondence.
Onset interval	What is the measured time difference between defined patient and ventilator inspiratory-onset reference points?	A measurement; not intrinsically abnormal.
Onset mismatch	Is the onset interval physiologically inappropriate for the reference signal and clinical context?	Interpreted as early or late triggering after mechanism is established.
Offset interval	What is the measured time difference between defined patient and ventilator inspiratory-offset reference points?	A measurement; not intrinsically abnormal.
Offset mismatch	Is the offset relationship physiologically inappropriate for the reference signal and clinical context?	Interpreted as early or late cycling.
Magnitude alignment	Does ventilator assistance produce a patient-ventilator workload distribution compatible with the intended physiological target?	Integrated interpretation of drive, effort, delivered ventilation, load, muscle capacity, and intended unloading.
Directionality	Which event initiated the coupled sequence when temporal order is atypical?	Patient-to-ventilator or ventilator-to-patient coupling; used as an interpretive modifier.
Cross-phase propagation	Does an abnormal relationship originating in one respiratory phase alter signals in the subsequent phase?	Mechanistic link across the inspiratory-expiratory boundary.

Signal correspondence

Before evaluating timing, one must establish whether the signals expected to interact are in fact both present. During conventional patient-triggered assisted ventilation, the expected relationship is one patient inspiratory signal followed by one corresponding ventilator inspiratory signal.

Correspondence must be established before timing can be assessed

Patient Signal Present, Ventilator Signal Absent

If a patient inspiratory signal occurs but fails to initiate the expected ventilator inspiratory signal, the abnormality is failed triggering. This is conceptualized here primarily as an abnormality of signal correspondence rather than as an extreme trigger delay, because there is no corresponding ventilator event from which a finite timing interval can be measured (Figure 2).

Ventilator Signal Present, Intended Patient Signal Absent

When a ventilator inspiratory signal occurs without a corresponding intended patient inspiratory signal and is initiated by an artifactual or non-respiratory trigger, the interaction represents unintended (false) triggering. This should be distinguished from an intentionally delivered mandatory breath (Figure 2).

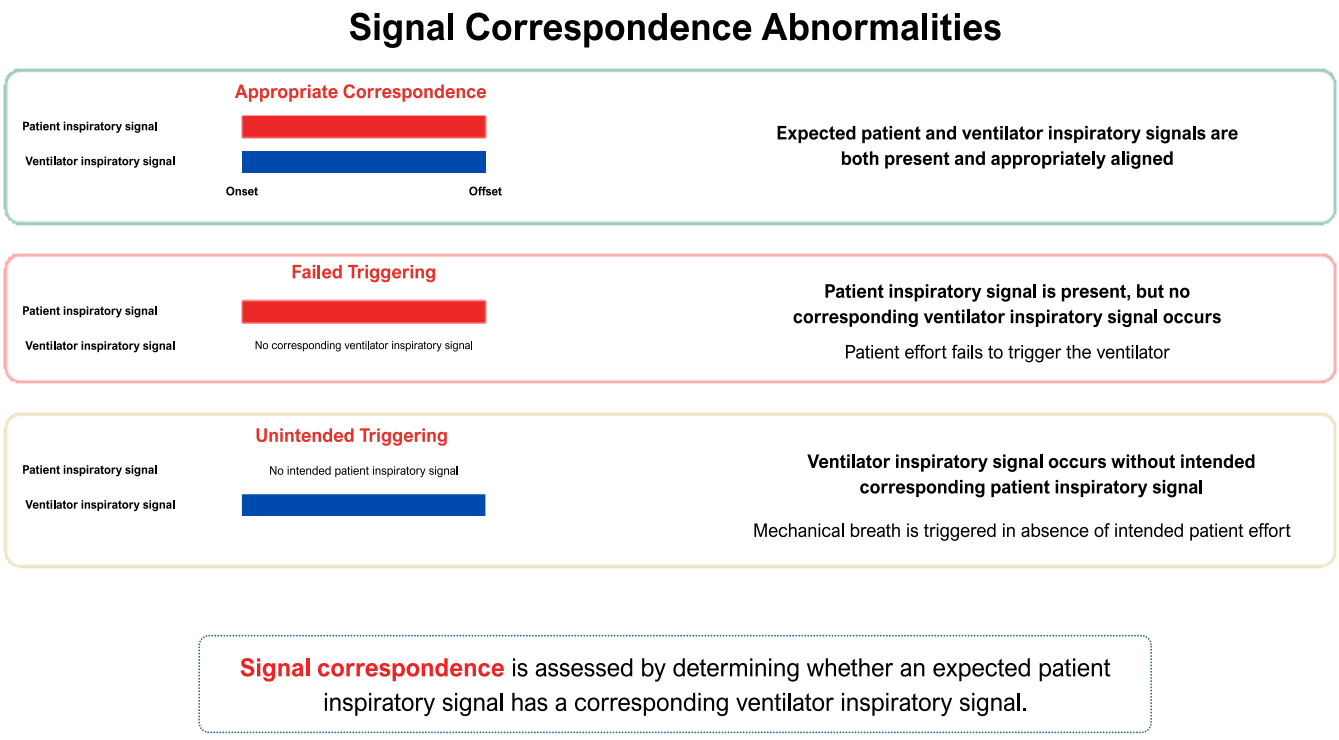

Figure 2: Signal correspondence abnormalities.

Signal correspondence precedes timing analysis

Timing should be evaluated only after the existence and correspondence of the signals being compared have been established. Identify the relevant signals and reference points; establish whether expected signals correspond; measure their timing when correspondence exists; and interpret timing and magnitude in the physiological context. This sequencing prevents a missing signal from being incorrectly described as an extreme timing delay.

Inspiratory-onset alignment

When a patient inspiratory signal has a corresponding ventilator inspiratory signal, their onset can be compared. This relationship forms the physiological basis of triggering. The trigger reference point compares ventilator signal onset with patient inspiratory signal onset. The interval between these events represents the trigger delay. Importantly, trigger delay is a measurement; it is not necessarily an abnormality.

Optimal triggering

Optimal triggering requires both appropriate signal correspondence and appropriate timing alignment. The objective is not necessarily simultaneous activation. Physiological signal transmission, respiratory muscle activation, ventilator sensing, signal processing, and mechanical response all introduce delay. Optimal triggering should therefore describe an appropriate temporal relationship rather than absolute simultaneity (Figure 3). As an indicative, not prescriptive, benchmark, trigger delays measured from airway pressure/flow of approximately less than 100 milliseconds are generally considered physiologically efficient in adult patients under clinical conditions, while delays greater than 300 milliseconds have been associated in prior physiological studies with increased patient effort and asynchrony index. [4] These figures should be interpreted cautiously: they depend heavily on the reference signal used, ventilator trigger algorithms, and patient population.

Early triggering

Early triggering describes a temporal relationship in which ventilator inspiratory signal onset precedes the patient inspiratory signal onset. Signal sequence alone, however, does not establish the mechanism. The same sequence may represent inappropriate early triggering relative to the selected patient reference, unintended (false) triggering, an intentional mandatory mechanical breath, or a mechanical breath followed by reverse-triggered patient activity. The expected relationship between the breath (ventilator signal) and the patient signal, as well as the direction of interaction, must first be determined (see " Direction of Signal Interaction," below).

Late triggering

Late triggering occurs when the onset of the patient inspiratory signal precedes the onset of the ventilator inspiratory signal by an interval that exceeds what is intended or physiologically appropriate. Trigger delay is the measured temporal interval; late triggering is the interpretation that this interval is abnormal (Figure 3).

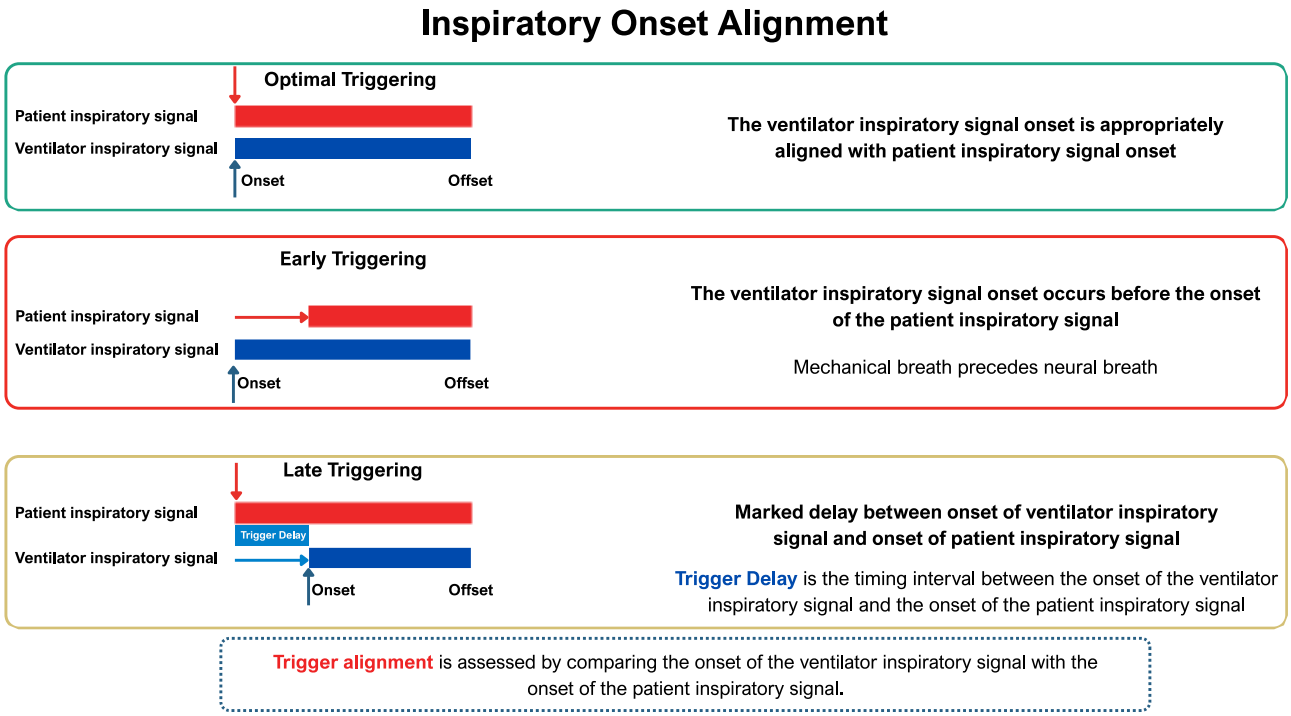

Figure 3: Inspiratory-onset alignment.

Trigger asynchrony as a signal abnormality

Trigger asynchrony can therefore be separated physiologically into two broad mechanisms: (1) loss of expected correspondence, represented by failed triggering, unintended (false) triggering; and (2) abnormal onset alignment, represented by early triggering and late triggering. A missed signal and signal onset misalignment are physiologically different phenomena, even though both may ultimately appear clinically as "abnormal triggering" (figure 4).

Inspiratory-offset alignment

Patient-ventilator interaction continues throughout inspiration. The termination of patient inspiratory activity and termination of mechanical inspiratory assistance form a second temporal relationship. The cycling-off reference point compares ventilator inspiratory signal offset with patient inspiratory signal offset. The interval separating these events represents the cycling-off delay. Cycling-off delay is a measurement; cycling-off mismatch is an abnormal timing relationship.

Optimal cycling-off

During optimal cycling-off, the mechanical inspiratory-signal offset occurs in an appropriate temporal relationship to the offset of the patient inspiratory signal. Exact simultaneity may not be achievable at the bedside. The relevant objective is an appropriate temporal relationship that avoids clinically important persistence of patient inspiratory effort after mechanical cycling-off or continued mechanical insufflation after the end of patient effort (Figure 5).

Early cycling

Early cycling occurs when the ventilator inspiratory signal offset precedes patient inspiratory signal offset. Mechanical assistance therefore ends while patient inspiratory activity persists (Figure 5). The persistent effort can oppose expiratory flow, increase respiratory muscle work, deform expiratory flow waveform, generate another trigger, and contribute to multiple triggering or breath stacking. Early cycling therefore illustrates how an inspiratory timing abnormality can propagate into the expiratory phase.

Late cycling

Late cycling represents the opposite relationship: the patient inspiratory signal offset precedes ventilator inspiratory signal offset. The ventilator therefore continues mechanical insufflation after the patient inspiratory activity has ended. The relevant measurement is the cycling-off interval, whereas late cycling describes the abnormal state (Figure 5).

The consequences depend on respiratory mechanics, assistance magnitude, patient effort and expiratory muscle recruitment. In patients with obstructive lung mechanics, prolonged mechanical inspiration may reduce the effective expiratory time and contribute to dynamic hyperinflation. [12] In other patients, expiratory muscle recruitment may develop because the ventilator continues inspiration while the patient is attempting to expire.

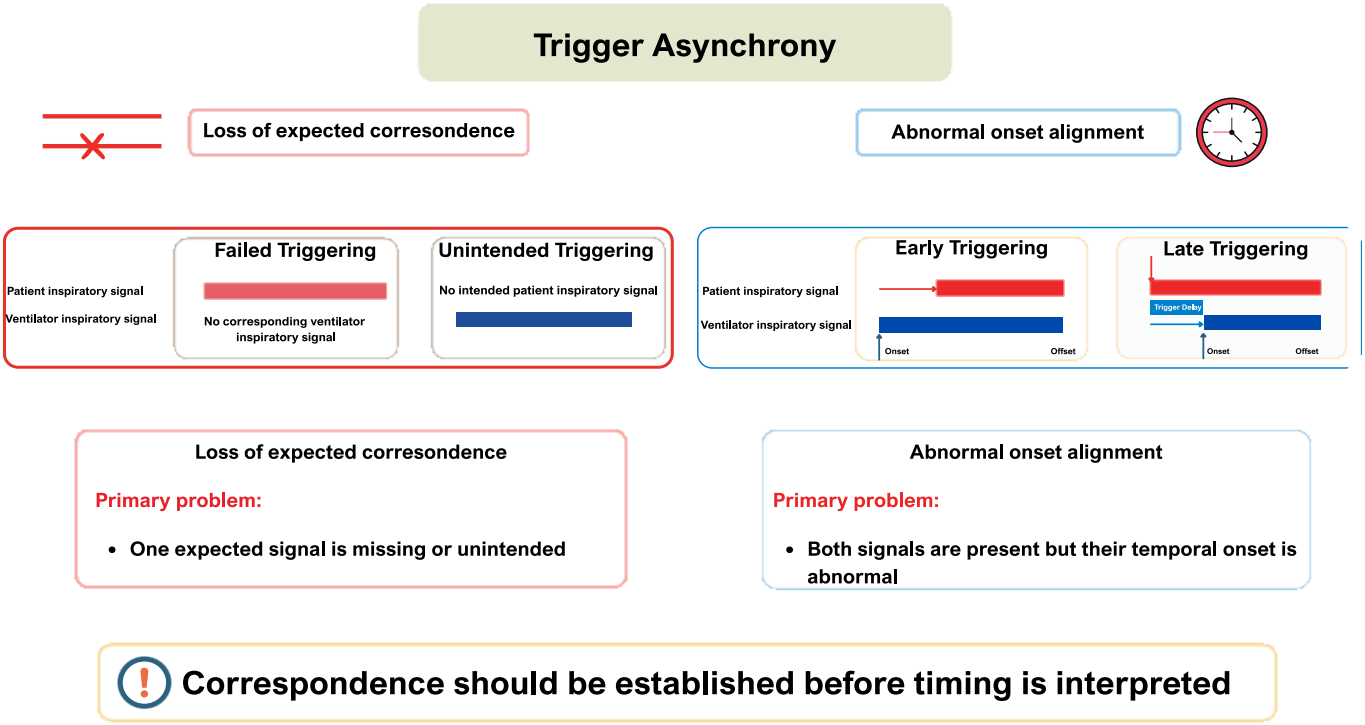

Figure 4: Trigger asynchrony as a signal abnormality

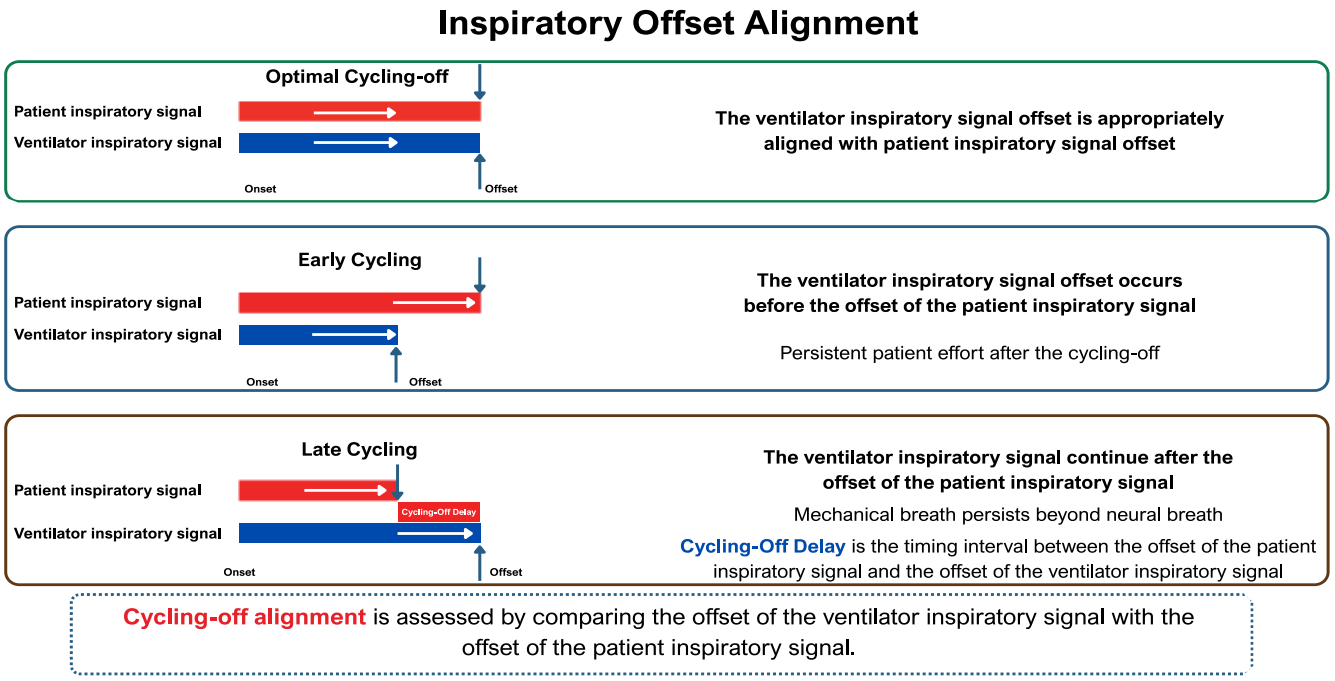

Figure 5: Inspiratory-offset alignment

Timing alignment across the inspiratory phase

Triggering and cycling should not be interpreted as unrelated events. A patient inspiratory signal possesses an onset, duration and an offset; the ventilator inspiratory signal similarly possesses an onset, duration, and an offset. Trigger mismatch reflects inappropriate alignment at the beginning of inspiration, whereas cycling-off mismatch reflects inappropriate alignment at its termination. Both abnormalities may coexist within the same breath.

Timing alignment therefore describes the temporal relationship across the complete inspiratory interval rather than isolated points on the waveform.

Timing alignment does not equal optimal PVI

A patient may exhibit one-to-one trigger correspondence, appropriate trigger timing, appropriate cycling-off timing, and no obvious conventional timing asynchrony, yet still receive physiologically inappropriate ventilator-delivered assistance.

A patient with high respiratory drive may be markedly under-assisted despite optimal triggering and cycling-off timing; conversely, a patient may be temporally synchronous but receive ventilator-delivered assistance that is excessive relative to the intended unloading target.

Temporal synchrony is therefore an important component of PVI, but not a sufficient definition of optimal interaction.

Magnitude alignment

Magnitude alignment addresses a different question from timing. Timing defines when assistance is delivered, while magnitude defines how much assistance is delivered relative to what the patient requires and what the clinician intends to unload.

Magnitude alignment is a patient-specific state in which the ventilator-delivered inspiratory assistance appropriately matches the patient's respiratory demand and the intended unloading goal.

Respiratory Demand and Assistance

Respiratory demand should not be treated as a single measurable variable. It reflects the integrated influence of central respiratory drive, metabolic demand, gas exchange requirements, respiratory system load, respiratory muscle capacity and therapeutic objective. Magnitude assessment therefore requires integration of several patient and ventilator signals rather than reliance on isolated ventilator settings.

Signals relevant to magnitude alignment

Potential patient-side measures include EAdi, P0.1, esophageal-pressure swing, derived Pmus, pressure-time product, airway-occlusion derived estimates of effort, respiratory rate. P0.1 can provide a clinically practical estimate of respiratory drive, whereas esophageal-pressure measurement provides more direct information regarding mechanical effort. [3,7,8,17]

Relevant ventilator information includes applied inspiratory pressure, delivered inspiratory flow, tidal volume, inspiratory duration, minute ventilation, and breath-to-breath response to changes in assistance. The physiological relationship between ventilator-delivered assistance, patient effort, respiratory system mechanics, and delivered tidal volume is non-linear and patient-specific. [14]

Adequate inspiratory assistance

Adequate inspiratory assistance represents the desired relationship in which ventilator-delivered inspiratory assistance is sufficient to support patient respiratory demand and achieve the intended unloading target. It requires both timing alignment and magnitude alignment: the appropriate level of assistance must be delivered at an appropriate time.

Under-assistance

Under-assistance is a state in which ventilator-delivered inspiratory assistance is insufficient relative to the patient's respiratory demand or intended unloading target. Potential manifestations may include persistent or excessive inspiratory effort, increased respiratory drive, high respiratory rate, dyspnea, and persistence of patient inspiration beyond mechanical cycling.

Under-assistance should not be defined by a single ventilator setting. Insufficient inspiratory flow may be one mechanism contributing to under-assistance, but under-assistance represents the broader relationship between patient demand and ventilator contribution.

Over-assistance

Over-assistance is a state in which ventilator-delivered inspiratory assistance is excessive relative to the patient's respiratory demand or intended unloading goal. Potential manifestations may include marked reduction in inspiratory effort, reduced respiratory rate, increased tidal volume, respiratory alkalosis, late cycling, dynamic hyperinflation, and subsequent failed trigger in

susceptible patients. Over-assistance is a relational state, not defined by a specific level of pressure assistance. The same ventilator settings may be excessive for one patient and inadequate for another.

Avoiding both insufficient and excessive respiratory effort is also relevant to lung- and diaphragm-protective ventilation, because abnormally low respiratory effort is associated with diaphragm disuse whereas excessive effort may impose injurious load on both diaphragm and injured lung. [16]

Interactions between timing and magnitude

Timing and magnitude abnormalities frequently interact. Under-assistance may increase respiratory drive and prolong the patient's neural inspiratory time which leads to persistent inspiratory activity extending beyond ventilator inspiration and thereby may contribute to early cycling or double triggering phenomenon. Over-assistance may reduce patient respiratory activity and may contribute to failed triggering. Early cycling reduces the duration of delivered assistance and may contribute to under-assistance. One waveform abnormality may therefore be the downstream expression of another disturbance in signal alignment.

The clinician should avoid treating individual waveform labels as necessarily independent events.

Timing and magnitude alignment dimensions

If abnormal PVI can be understood through inappropriate signal relationships, optimal PVI can be approached through appropriate alignment. Optimal inspiratory interaction should include appropriate signal correspondence, appropriate trigger alignment, appropriate cycling-off alignment, and appropriate magnitude alignment, interpreted in relation to the patient and the intended clinical goal (Figure 6).

A patient can therefore be synchronous but under-assisted, or synchronous but over-assisted. Likewise, a patient may exhibit a modest timing mismatch while receiving an otherwise appropriate magnitude of assistance. PVI therefore exists across multiple dimensions rather than as a binary synchronous/asynchronous state (Figure 6).

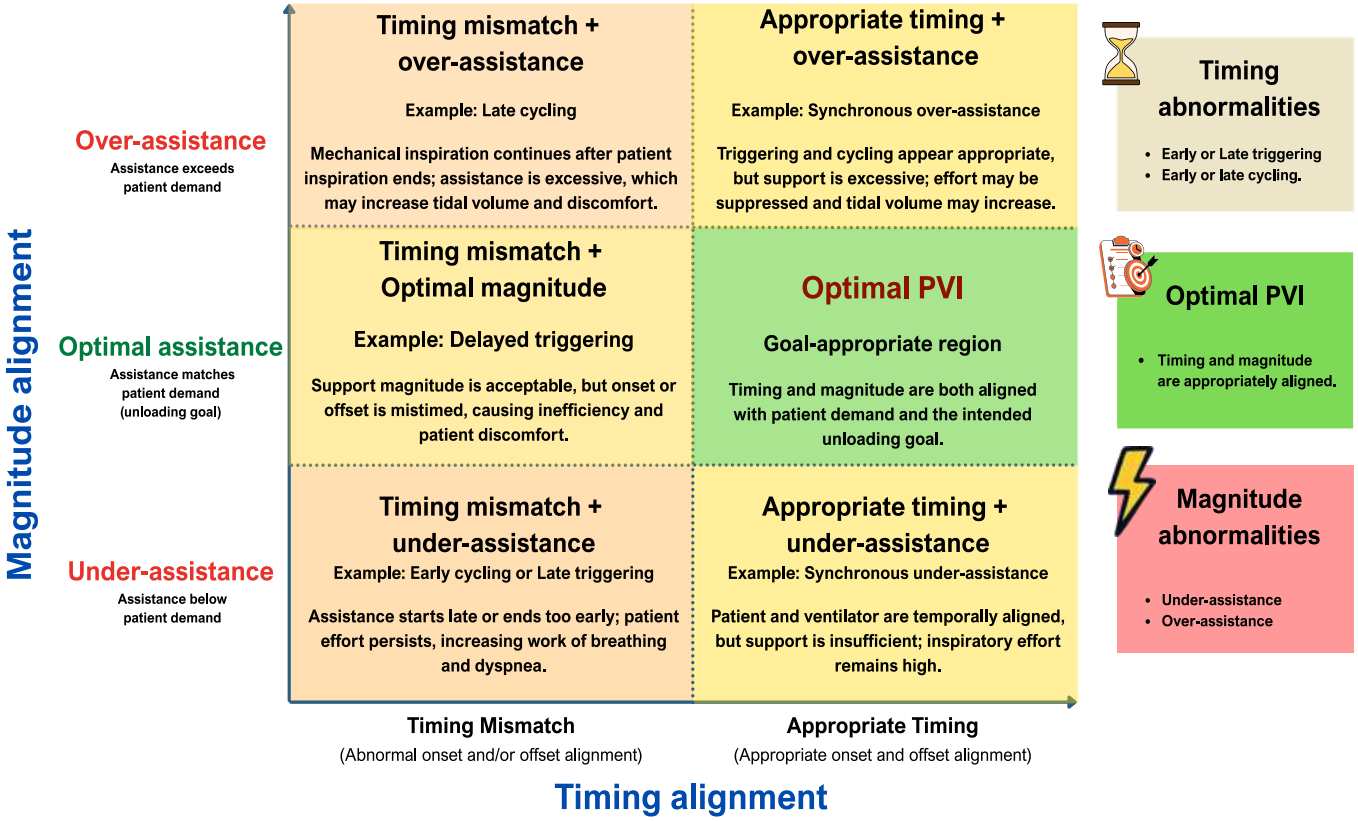

Figure 6: Timing and magnitude alignment dimensions. Two-dimensional diagram with timing alignment on the x-axis and magnitude alignment on the y-axis, illustrating that appropriate timing can coexist with under-assistance or over-assistance and that optimal PVI occupies the goal-appropriate region.

Alignment, mismatch, and signal loss

The major inspiratory abnormalities can be described physiologically as three fundamental disturbances: loss of expected signal correspondence, timing mismatch, and magnitude mismatch (Table 2). These abnormalities may occur independently or in combination.

Direct signals versus surrogate signals

A major challenge in PVI assessment is that the true physiological patient signal is often not directly available. EAdi, esophageal pressure, Pmus, and gastric pressure can provide relatively direct information about neural or muscular signals. Airway pressure and flow can often infer patient activity without invasive monitoring, but these signals are influenced by respiratory system mechanics. The distinction is therefore not that one signal is useful, but another is not. Rather, signals differ in physiological concepts they represent and in the confidence with which timing and magnitude can be inferred. Whenever a PVI abnormality is reported, the patient reference signal should therefore be specified.

Signal alignment is reference-dependent

Alignment must always be interpreted relative to the specified reference signal. Neural inspiratory onset precedes mechanical pressure generation. Mechanical pressure generation precedes a detectable change in airway flow. Similarly, neural inspiratory termination precedes complete relaxation of the respiratory muscles.

Therefore, trigger and cycling intervals measured using EAdi are not necessarily equivalent to intervals derived from esophageal pressure-derived measures or inferred from flow. Standardization of PVI requires not only standardized terminology but transparent reporting of the signals and reference points used to define each abnormality.

Measurement versus abnormality

Another essential distinction is between a measurement and the interpretation of that measurement. Trigger delay, cycling-off delay, and similar intervals are measured temporal values; late triggering, cycling-off mismatch, under-assistance, and over-assistance are the interpretation that a given measured value is physiologically inappropriate.

The distinction matters because physiological delay is unavoidable: signal transmission, muscle electromechanical coupling, ventilator detection, control-system processing, and pneumatic response all require time. Optimal PVI should therefore represent physiologically appropriate alignment rather than mathematical simultaneity.

Table 2: Different PVI Abnormalities Viewed Through Signal Alignment

PVI abnormality	Phase	Patient signal	Ventilator signal	Principal signal abnormality
Failed trigger	**Inspiratory**	Present	Corresponding ventilator inspiratory signal absent	Loss of correspondence
Unintended / false triggering		Intended patient signal absent	Ventilator inspiratory signal present	Loss of correspondence
Early triggering		Patient onset after ventilator onset	Present	Onset timing mismatch
Late triggering		Patient onset precedes ventilator onset excessively	Present	Onset timing mismatch
Optimal triggering		Present	Present	Appropriate correspondence + onset alignment
Early cycling		Patient inspiration continues	Ventilator inspiration terminates	Offset timing mismatch
Late cycling		Patient inspiration terminates	Ventilator inspiration continues	Offset timing mismatch
Optimal cycling-off		Present	Present	Appropriate offset alignment
Under-assistance		Demand/effort exceeds intended relationship	Assistance insufficient	Magnitude mismatch
Over-assistance		Demand/effort low relative to delivered support	Assistance excessive	Magnitude mismatch
Active expiration	**Expiratory**	Expiratory muscle contraction	Airway pressure/flow perturbation	Signal misattribution
Persistent inspiratory activity after cycling-off		Inspiratory activity continues beyond cycling-off	Expiratory signal (flow, pressure) already initiated	Propagated offset timing mismatch
Expiratory muscle relaxation-induced triggering		Expiratory muscle relaxation (passive recoil signal)	Ventilator inspiratory signal present (unintended)	Loss of correspondence (misattributed direction)

Expiratory-phase interactions and cross-phase propagation

Most discussions and publications of PVI focus predominantly on inspiration. Triggering, inspiratory pressure and flow delivery, and cycling-off have received extensive attention. Yet the interaction between patient and ventilator does not terminate when mechanical inspiration ends.

Mechanical expiration begins at ventilator cycling-off and extends until the next mechanical inspiration onset. During this period, the ventilator measures expiratory flow and maintains airway pressure near the selected PEEP level, while patient respiratory muscles may be absent, actively recruited, relaxing, or transitioning toward the next inspiratory effort. The expiratory phase should therefore be regarded as an integral component of PVI assessment rather than merely the interval between two inspiratory events. Recognition and appropriate interpretation of expiratory phase patient and ventilator signals are essential for comprehensive understanding of PVI across the entire respiratory cycle.

Ventilator expiratory signals

Expiratory flow

Following cycling-off, expiratory flow becomes the principal time-varying ventilator-measured signal during the expiratory phase. During passive expiration, and in the absence of major nonlinear respiratory system behavior or expiratory flow limitation, the flow-time waveform typically demonstrates a smooth exponential decay determined largely by respiratory system mechanics.

The passive pattern can serve as a useful mechanical reference against which deviations or waveform deformations related to active respiratory muscle activity may be recognized.

Active expiratory muscle activity can deform the expiratory flow waveform. Potential appearances include acceleration of expiratory flow, loss of exponential decay, alteration in waveform concavity, and waveform deformation. These are indirect manifestations and should be interpreted alongside respiratory mechanics and, when necessary, direct physiological signals.

Expiratory airway pressure

During expiration, airway pressure is maintained near set positive end-expiratory pressure (PEEP) level by the ventilator. Superimposed patient respiratory muscle activity, whether expiratory or inspiratory, can affect this pressure and produce perturbations in airway pressure and flow. Consequently, expiratory airway pressure and flow should be regarded as ventilator measured interaction signals reflecting the combined influence of ventilator-applied pressure, patient respiratory muscle activity, respiratory system mechanics rather than representing either patient or ventilator signal in isolation.

Patient expiratory signals

Patient respiratory muscle activity during expiratory phase may take several forms, including active expiratory muscle recruitment, persistence of inspiratory muscle activity beyond ventilator cycling-off, relaxation of previously active expiratory muscles, and subsequent reactivation of inspiratory muscles as the next breath approaches.

Direct characterization of these physiological signals may require measurements such as gastric pressure, esophageal pressure, EAdi, or electromyographic assessment of expiratory muscles. In contrast, changes observed in ventilator-measured airway pressure or flow provide indirect evidence of underlying respiratory muscle activity and should be interpreted in conjunction with respiratory system mechanics.

Passive expiration and signal correspondence

During passive expiration, the absence of an active patient expiratory muscle signal is itself the expected physiological state. Expiratory flow occurs predominantly due to respiratory system elastic recoil, while the ventilator maintains the set PEEP. Therefore, the presence of ventilator expiratory signal in absence of patient expiratory signals does not represent loss of signal correspondence or abnormal PVI. Rather, this represents appropriate expiratory patient-ventilator interaction.

Active expiration

Active expiration introduces an additional patient signal into the expiratory phase, arising from recruitment of abdominal and other expiratory muscles. This is more likely to occur when respiratory drive or respiratory system loading is increased during expiratory phase.

Direct identification of patient expiratory signals may require physiological measurement such as gastric pressure, with an expiratory rise in gastric pressure providing particularly useful evidence of abdominal expiratory muscle recruitment. The effect of active expiration on ventilator signals may resemble abnormalities traditionally attributed to inspiratory activity. A physiological observational study demonstrated that active expiratory muscle activity could generate airway pressure and flow patterns resembling failed triggering or

unintended triggering. [15] Gastric pressure monitoring was important in establishing that these waveform abnormalities originated from expiratory muscle activity rather than inspiratory muscle recruitment. [18]

Similar ventilator waveform abnormalities may arise from different patient physiological signals; identifying the signal responsible for the waveform change is therefore essential before assigning the mechanism of abnormal PVI.

Persistent inspiratory activity after cycling-off

The expiratory phase may contain a residual patient inspiratory signal during early cycling. In this situation, the cycling-off point marks the onset of the ventilator expiratory signal, while the patient-inspiratory signal continues beyond the cycling-off point. The continuing inspiratory effort therefore becomes superimposed on the expiratory phase. This persistent patient signal may oppose the development of expiratory flow, distort the expected expiratory flow waveform, shorten the effective expiratory time, and, if sufficiently strong, trigger additional mechanical breath initiation.

From an interacting-signal perspective, this represents propagation of an inspiratory offset mismatch across the inspiratory-expiratory phase boundary. PVI abnormalities may extend across respiratory phases and may influence the signals observed in the subsequent phase.

Expiratory muscle relaxation-induced triggering

A particularly important example of expiratory signal interaction is expiratory muscle relaxation-induced ventilator triggering (ERIT). During active expiration, contraction of the abdominal and other expiratory muscles generates a positive pressure signal. When these muscles relax near end-expiration, the resulting fall in abdominal and pleural pressure may produce a corresponding change in esophageal pressure, airway pressure, and flow. If these changes are sufficient, they may trigger initiation of a new mechanical breath despite the absence of an intended patient's inspiratory effort. [19] The ventilator is therefore responding to a patient-generated expiratory relaxation signal rather than the expected patient-inspiratory signal.

Directionality as an interpretive modifier

Directionality differs conceptually from correspondence, timing, and magnitude because it does not represent an additional alignment domain. Rather, it is an interpretive modifier used when the temporal sequence of signals does not by itself establish which system initiated the interaction. Most patient-triggered assisted breaths follow the expected signal direction:

Patient-inspiratory signal → ventilator-inspiratory signal

The patient initiates respiratory activity, the ventilator detects its mechanical expression, and mechanical assistance follows. However, signal interaction does not always proceed in this direction.

In some circumstances, the ventilator signal precedes and influences subsequent patient respiratory activity:

Ventilator-inspiratory signal → patient-inspiratory signal

Recognition of signal direction is therefore essential when interpreting PVI. A ventilator inspiratory event occurring before a patient-inspiratory signal cannot be classified solely from temporal order. It may represent an intentionally delivered mandatory breath, unintentional triggering, or ventilator-to-patient coupling such as reverse triggering. Determining which event initiated the interaction is necessary before assigning its physiological meaning.

Reverse Triggering: ventilator to patient signal coupling

Reverse triggering provides an established example of reversed signal directionality. In this phenomenon, a ventilator delivered breath precedes and is followed by patient-inspiratory effort. [20] The defining feature is not simply that the ventilator signal occurs earlier than the patient signal, but that the direction of interaction has changed.

Towards optimal patient-ventilator interaction

If abnormal PVI can be understood as inappropriate relationships among interacting patient and ventilator signals, optimal PVI can be approached by identifying the physiological relationships that should be preserved across the respiratory cycle. Optimal PVI is characterized by expected signal correspondence, appropriate inspiratory-onset alignment, appropriate inspiratory-offset alignment, and appropriate magnitude alignment of ventilator-delivered assistance relative to patient respiratory demand and intended unloading goal. During expiration, optimal interaction requires coordinated signal behavior without clinically important loading, inappropriate persistence of inspiratory activity, unintended triggering, or other physiologically inappropriate signal interactions.

Optimal PVI is Patient-Specific

There is unlikely to be a universal ventilator setting that defines optimal interaction. Appropriate ventilator

contribution depends on respiratory drive, respiratory system load, respiratory muscle capacity, severity of lung injury, gas exchange requirements, and intended unloading targets.
A level of pressure support that represents over-assistance for one patient may represent under-assistance for another. Optimal PVI is therefore a relationship rather than a ventilator setting.

Optimal PVI is dynamic

Respiratory physiology changes continuously. Respiratory drive can increase or decrease. Respiratory system mechanics evolve. Sedation changes. Metabolic demand changes. Respiratory muscle function can deteriorate or recover. The same ventilator settings can consequently generate different PVI states over time.

Practical bedside application

The signal-based framework can be translated into a stepwise algorithm (Figure 7). The sequence intentionally begins with observations and signals and ends with assignment of conventional PVI phenotype or mechanism (Figure 7).

1. Identify the signals available at bedside, and specify which signal will serve as the patient reference signal
2. Establish signal correspondence: for each patient-inspiratory signal, is there a corresponding ventilator inspiratory signal, and vice versa? If correspondence is absent, classify as failed trigger or unintended/false trigger before proceeding.
3. Assess onset alignment: if correspondence is present, assess the trigger delay and judge whether it is physiologically appropriate for the reference signal used (early vs. late triggering).
4. Assess offset alignment: assess the cycling-off interval and judge whether ventilator offset appropriately matches patient-inspiratory offset (early vs. late cycling)
5. Assess magnitude alignment: independent of timing, evaluate whether the delivered assistance matches respiratory demand and intended unloading goal (under- vs. over-assistance), using available muscle effort measurements or surrogates.
6. Extend assessment into expiration: examine expiratory flow/pressure waveforms for evidence of active expiration, persistent inspiratory activity after cycling-off, or ERIT before attributing an apparent inspiratory-phase abnormality to inspiratory mechanisms alone.
7. Determine direction of interaction: when the temporal sequence is unusual (ventilator event preceding patient signal), determine whether this reflects an intended mandatory breath or a ventilator-to-patient coupling phenomenon such as reverse triggering.

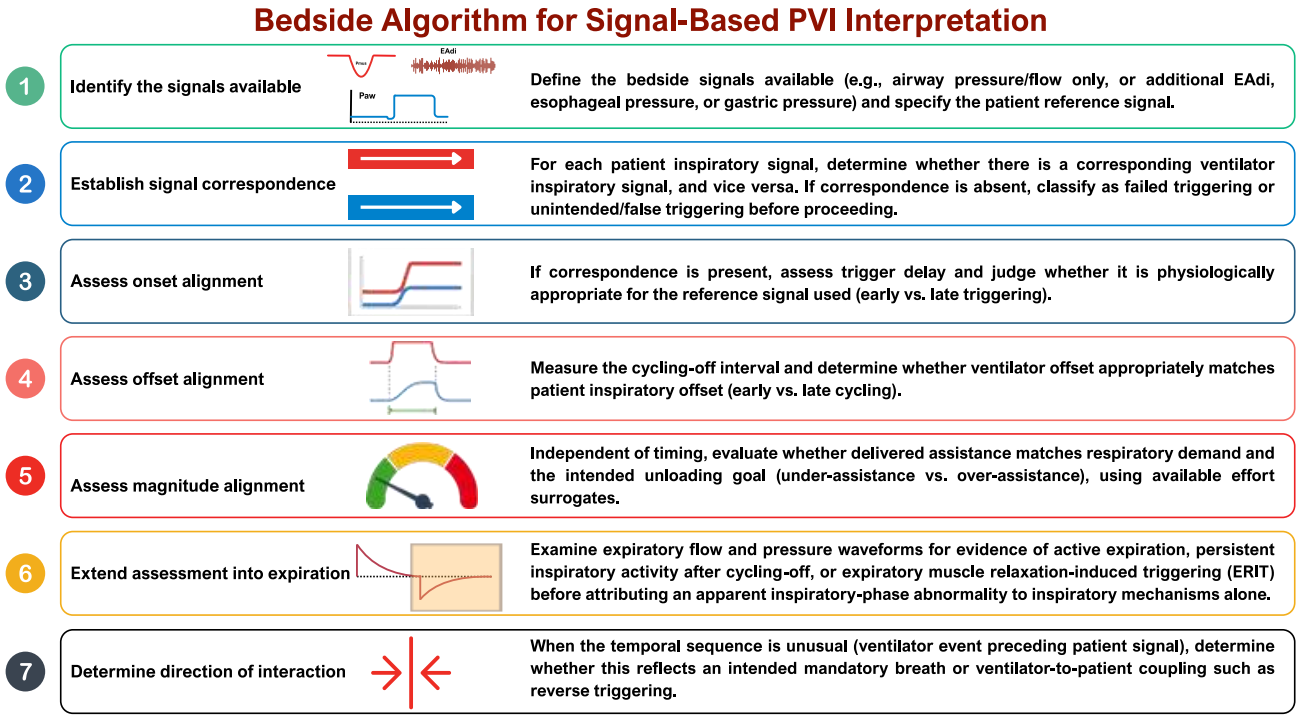

Figure 7: Proposed stepwise bedside algorithm for signal-based PVI interpretation

Conclusion

Patient-ventilator interaction is fundamentally an interaction between physiological and mechanical signals generated within two dynamically coupled systems: the patient and the ventilator. The patient generates signals reflecting respiratory drive, neural respiratory activation, inspiratory and expiratory muscle activity, respiratory effort, and resulting mechanical response. The ventilator generates control events and measures interaction signals reflecting triggering, mechanical inspiration, pressure and flow delivery, volume delivery, cycling-off, and expiration.

Understanding PVI requires first identifying which signals are being compared. Signal correspondence determines whether expected events are present. Inspiratory-onset alignment determines the triggering relationship. Inspiratory-offset alignment determines the cycling-off relationship. Magnitude alignment determines whether ventilator-delivered assistance matches the patient's respiratory demand and intended unloading goal.

Expiration adds further interaction involving active expiratory muscle contraction, expiratory flow deformation, persistent inspiratory activity after cycling-off, and expiratory muscle relaxation-induced triggering. Reverse triggering demonstrates that the direction of interaction itself may also require consideration.

Accordingly, abnormal PVI patterns are manifestations of different relationships among interacting physiological and mechanical signals. The goal of PVI assessment should therefore extend beyond questioning whether the patient and ventilator are synchronous. The more physiologically meaningful questions are: Which signals are interacting? Do the expected signals correspond? Are their onset and offset appropriately aligned? Does ventilator assistance appropriately match respiratory demand? What is occurring during expiration? And which system is driving the interaction? Answering these questions provides a physiological path towards optimal patient-ventilator interaction.

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List of Abbreviations:
EAdi: Electrical activity of the diaphragm.
ERIT: Expiratory muscle relaxation-induced ventilator triggering.
P0.1: Airway occlusion pressure measured 100 milliseconds after the onset of inspiratory effort against an occluded airway.
PEEP: Positive end-expiratory pressure.
Pmus: Respiratory muscle pressure.
PVI: Patient-ventilator interaction.