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Rethinking double triggering: A phenotype rather than a patient–ventilator dyssynchrony?

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Abstract

Double triggering has traditionally been defined as the presence of two consecutive ventilator cycles separated by an expiratory time equal or shorter than 50% of the inspiratory time. It is considered the consequence of a single inspiratory effort and is recognized as one of the most frequent forms of patient–ventilator dyssynchrony. Epidemiological studies report that this entity occurs in approximately 15–25% of mechanically ventilated patients and is associated with longer duration of mechanical ventilation and ICU stay, although a consistent association with mortality has not been demonstrated. However, growing physiological evidence suggests that double triggering does not constitute a homogeneous entity, but rather a phenotypic expression arising from distinct underlying processes.

This reappraisal revisits the conventional conceptual framework of double triggering and proposes a mechanistic reinterpretation. While in rare cases it may represent truly consecutive neural inspiratory efforts, most episodes appear to result from mismatches between respiratory drive and ventilator timing or flow delivery. Premature cycling, insufficient inspiratory flow or reverse triggering may converge into the same observable pattern of two closely spaced ventilator cycles. Despite similar waveform geography, these pathophysiological mechanisms differ substantially and have a potential impact on tidal volume, transpulmonary pressure, and lung stress.

Reconceptualizing double triggering as a phenotype rather than a discrete dyssynchrony entity carries relevant clinical implications. When this pattern is identified, management should focus on elucidating the underlying mechanism through careful waveform inspection and detailed physiologic assessment. Such a mechanism-oriented, pathophysiology-driven approach may optimize ventilator adjustment and allow a more nuanced interpretation of outcome associations attributed to double triggering.

Keywords: Double triggering, Dyssynchronies, Patient-ventilator interactions

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What we know about double triggering

Double triggering is commonly defined as the delivery of two ventilator breaths in response to a single patient's inspiratory effort, often due to insufficient ventilator inspiratory time relative to patient neural demand or insufficient ventilatory support relative to patient's effort. This pattern frequently produces breath stacking, increasing tidal volume and transpulmonary pressures that may predispose to lung injury. [1] Early physiologic work and clinical observation, including non-invasive ventilation, have long recognized double triggering as one of the key forms of patient–ventilator dyssynchrony alongside ineffective triggering, auto-triggering and delayed cycling. [2]

Epidemiological studies and systematic reviews have demonstrated that patient–ventilator dyssynchrony (PVD) occurs in a large proportion of mechanically ventilated patients, with double triggering reported in approximately 15–25% of cases in some clinical cohorts. [2,3] In a systematic review of 19 studies including 2672 patients undergoing invasive mechanical ventilation, both double triggering and ineffective triggering were significantly associated with longer duration of mechanical ventilation (mean difference 3.29 days; 95% CI 0.13–6.44) and longer ICU length of stay (mean difference 3.65 days; 95% CI 1.20–6.11), although no clear association with mortality was detected in pooled analysis. [3] These findings raise the possibility that double triggering serves as a marker for suboptimal patient–ventilator interaction and may be linked to adverse clinical trajectories.

Prospective cohort data further support this association. In a study of 82 patients with COVID-19 ARDS, double triggering accounted for a median of 0.44% of breaths (IQR 0.19–0.80) and double triggering clusters were independently associated with longer duration of mechanical ventilation and improved ICU survival, suggesting complex interactions between dyssynchrony patterns, disease severity, and outcomes. [4] Similarly, the EPISYNC cohort of 103 mechanically ventilated adult ICU patients identified double triggering clusters in 87% of cases and explored associations with clinical outcomes, reinforcing that double triggering is frequently observed during routine ventilation even if its independent prognostic role remains under investigation. [5]

Cluster analyses of PVD often demonstrate that double triggering rarely occurs in isolation; it coexists with other asynchronies and clinical factors such as sedation depth, ventilator mode, and patient respiratory drive. In observational cohorts, asynchrony index ≥ 10% (including double triggering in its calculation) has been associated with prolonged ventilation and ICU stay, although its relationship with mortality is inconsistent across settings. [3,6]

Thus, the conventional understanding posits double triggering as a distinct dyssynchrony entity with mechanistic links to patient–ventilator mismatch and potential clinical implications but one that may not represent a homogeneous physiological phenomenon.

From entity to phenotype: A mechanistic reinterpretation

True primary double triggering

There are exceptional circumstances in which double triggering may reflect two genuine consecutive neural inspiratory efforts, such as hiccup like patterns or certain neurological dysrhythmias. These rare non-automaton reflex patterns may be accurately captured as separate efforts by the ventilator, representing a true dual neural drive rather than mechanical maladjustment (Figure 1).

However, such cases are uncommon in routine critical care practice, and many double triggering events are observed in the context of mechanical ventilator settings that fail to align with patient demand.

Secondary double triggering

While double triggering has been conceptualized as a discrete category of PVD, deeper physiological analysis suggests that it may often be a manifestation of underlying mismatches in neural and mechanical timing or flow demand, rather than a single mechanistic entity.

Indeed, double triggering can result from several distinct mechanisms:

- Premature cycling, where the ventilator ends inspiration before the patient's neural inspiratory effort concludes, leading to a second trigger once inspiratory drive persists. This pattern was described as a common cause of double triggering in volume-controlled and pressure-controlled modes (Figure 2).
- Insufficient inspiratory flow, particularly when the set flow fails to meet neural demand, prolonging effort and prompting re-triggering (Figure 2).
- Reverse triggering, a reflexive patient effort induced by the ventilator cycle, has been documented to produce stacked breaths. In some exceptional cases it may be clinically indistinguishable from double triggering on basic ventilator waveforms (Figure 3).
- Trigger sensitivity abnormalities, including excessive trigger responsiveness or auto-triggering conditions, can

produce repeated cycle initiation without distinct neural effort.

In this framework, the observable pattern of two consecutive triggered breaths is a common endpoint arising from disparate mechanisms, each with distinct physiological bases. Thus, double triggering may be better conceptualized as a phenotypic expression of underlying dyssynchrony and mismatch rather than a standalone dyssynchrony type with a singular cause.

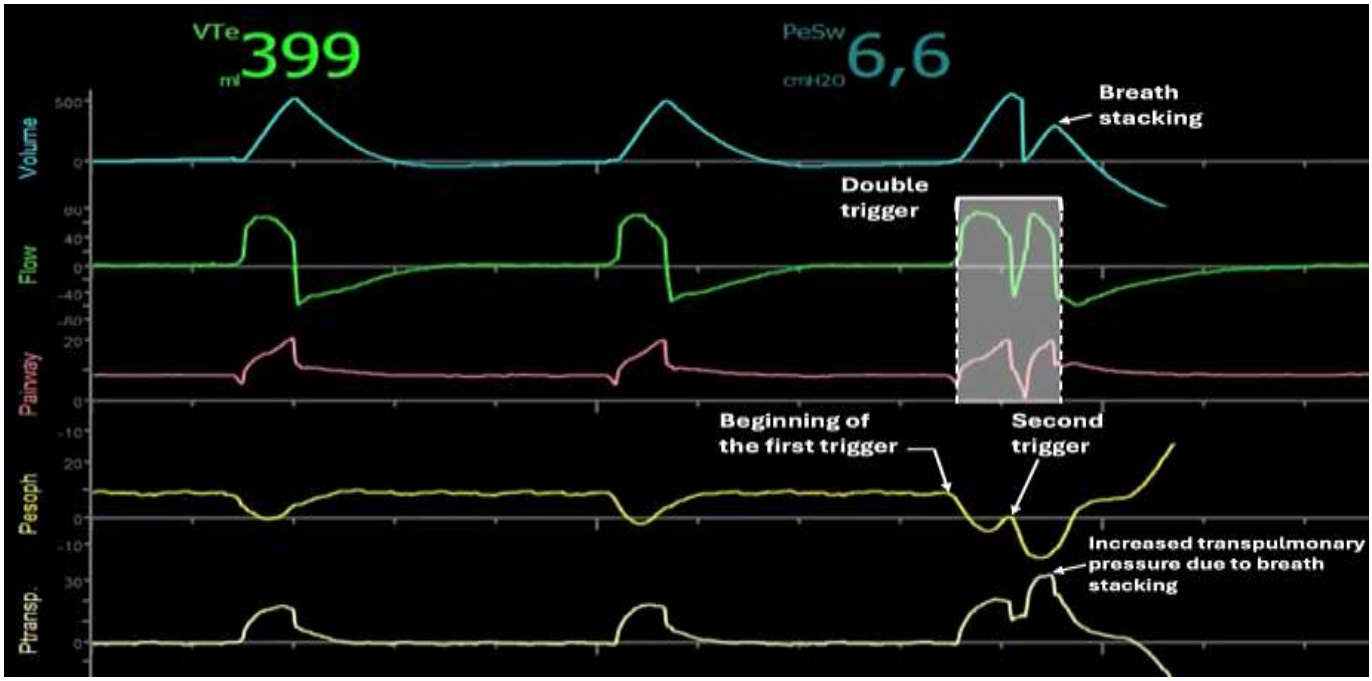

Figure 1: Double trigger due to hippcups. Two consecutive triggers can be observed (box between dotted lines). An increase in transpulmonary pressure secondary to the presence of breath stacking is also observed.

Figure 2: Double triggering due to premature cycling and insufficient inspiratory flow. The nadir of esophageal pressure (maximum negative deflection, indicative of diaphragmatic effort) occurs beyond the cycling point. Additionally, during the first cycle, a pressure "plateau" can be observed (indicative of ongoing patient muscular effort), whereas under normal conditions airway pressure should rise as inspiratory flow is delivered into the system.

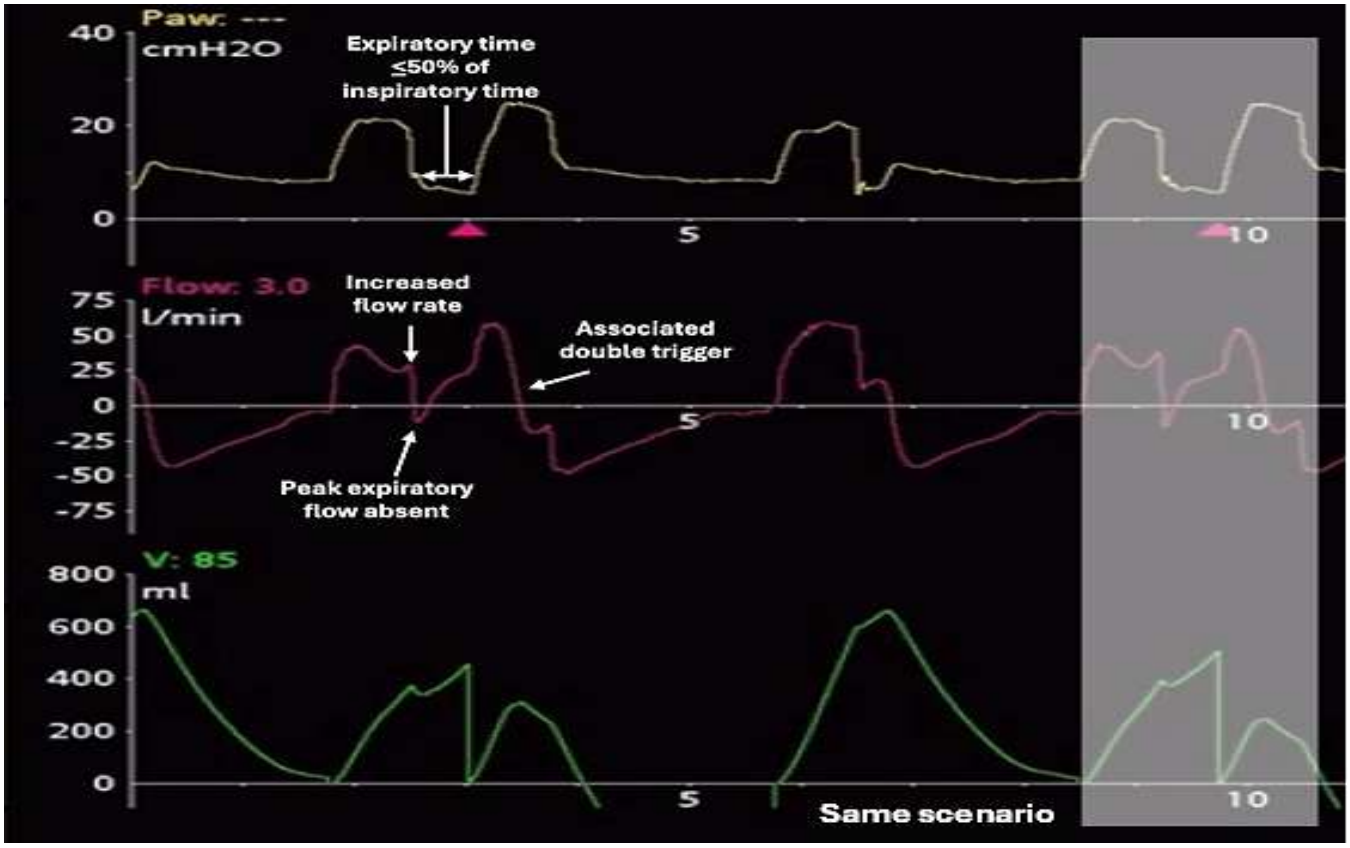

Figure 3: Conventional tracings, as seen on a ventilator screen (without esophageal or transpulmonary pressure). The beginning of the mechanical cycle shows no pressure deflection, suggesting a mandatory cycle. The drop and subsequent increase in flow during the first cycle suggests the presence of a reverse trigger, which ultimately triggers a new respiratory cycle (associated double trigger).

Clinical implications of distinguishing mechanisms

Recognizing that double triggering is often a phenotype rather than a primary mechanistic entity has several clinical implications.

Diagnostic approach

When double triggering is detected on ventilator waveforms, clinicians should not reflexively treat the pattern itself but rather investigate the underlying mechanism. Detailed inspection of pressure–time and flow–time curves, along with adjuncts such as esophageal pressure monitoring (if available), can help distinguish whether the re-triggering arises from premature cycling, inadequate flow delivery, reverse triggering, or trigger sensitivity issues.

This approach moves beyond pattern recognition, aiming at mechanism-targeted diagnosis and precision in ventilator adjustments.

Therapeutic consequences

Treatments aimed purely at suppressing double triggering such as increasing sedation may be ineffective or counterproductive if they do not address the true cause. For instance:

- Adjusting cycling criteria or prolonging inspiratory time may ameliorate double triggering due to premature cycling.
- Increasing inspiratory flow or changing ventilator mode might better match neural demand.
- For reverse triggering, strategies may include sedation depth modulation or changing modes that reduce ventilator-induced entrainment.

Clinical trials targeting overall PVD with specific protocols have shown improved outcomes such as reduced duration of mechanical ventilation and shorter ICU stays, suggesting that proactive management of underlying dyssynchronies can be beneficial. For example, a randomized controlled trial using a protocolized dyssynchrony management approach demonstrated better outcomes in PVD frequency and ventilator days, although specific breakdown for double triggering was not always isolated in earlier reports. [7] Other studies, including a systematic review and meta-analysis, have shown similar results. [8,9]

Interpretation of outcome studies

Systematic reviews indicate that while PVD and subtypes such as double triggering and ineffective triggering are associated with prolonged mechanical ventilation and ICU stay, associations with mortality are less consistent. [3] This may reflect the fact that double triggering, considered in isolation, is a marker of patient–ventilator mismatch and disease severity rather than a causative mediator of adverse outcomes.

Framing double triggering as a phenotype encourages nuanced interpretation of outcome data: rather than attributing causality to the waveform pattern itself, it may be more accurate to view it as an indicator of underlying physiological derangements and ventilator setting mismatches that require comprehensive management.

Conclusion

Double triggering remains a commonly observed pattern in mechanically ventilated patients and is associated with important clinical outcomes such as longer mechanical ventilation duration and ICU stay. However, evidence suggests that it is not a homogeneous, primary dyssynchrony but rather a terminal phenotype arising from diverse mechanisms including premature cycling, flow insufficiency, trigger sensitivity mismatches, and reflex entrainment such as reverse triggering.

Recognizing this distinction has critical implications for diagnosis, management, and interpretation of research. Mechanistic differentiation enables precision in ventilator adjustment, avoiding non-specific interventions and potentially improving patient outcomes.

Future studies should refine diagnostic criteria and integrate mechanistic insights to develop targeted interventions for specific underlying causes of double triggering, moving beyond pattern suppression toward true physiologic optimization of patient–ventilator interaction.

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