

CHAPTER 2 Patient Ventilator Dyssynchrony: Clinical Significance and
Implications for Practice

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Implications for Practice

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The cost of providing care to critically ill patients in the United States consumes roughly 15% of all healthcare dollars, or 1% of the gross national product.¹ Contributing to this economic burden are patients admitted to the Intensive Care Unit (ICU) who require mechanical ventilation in patients with complications from their dependence on this technology.^{2, 3} In fact, 50% of ICU patients receive mechanical ventilation.⁴ Often, sedation is required to increase patients' tolerance of the endotracheal tube, reduce anxiety, and facilitate sleep. In particular, sedation is used frequently to reduce patient-ventilator dyssynchrony (PVD).⁵⁻⁹ Sassoon and Foster¹⁰ define PVD as a mismatching between the patient's breaths (neural) and ventilator-assisted breaths (phase asynchrony), as well as the inability of the ventilator's flow delivery to match the patient's flow demand (flow asynchrony). This definition suggests a faulty interaction between the patient and ventilator that is commonly managed by sedation and advanced ventilator modes and adjustments. The correction of PVD is complex and multifaceted given the current capabilities of traditional ventilators. An imperfect solution exists because the sensitivity and responsiveness of both patient and ventilator during the interaction is confounded by factors related to the patient and ventilator. However, in light of the most serious complications (hypoxemia, barotrauma, prolonged mechanical ventilation and discomfort) of PVD, and an imperfect solution for the resolution of PVD at the current time, nurses continue to face the challenge of preventing the consequences of PVD as well as complications due to oversedation and undersedation. In this article we will discuss the factors contributing to PVD; the manifestations, measurement, types and causes of PVD; nursing implications; and future directions for improvement with nursing research questions proposed for consideration.

Factors Affecting Patient-Ventilator Interaction

Patient ventilator interaction is influenced by factors related to the patient (respiratory center output, respiratory system mechanisms, disease states and conditions, artificial airway) and factors related to the ventilator (ventilator triggering, ventilator cycling-off).

Patient-Related Factors

Achieving patient ventilator synchrony during interactive modes of ventilation (assist control [AC] and synchronized intermittent mandatory ventilation [SIMV]) is a daunting task because patients' ventilation is controlled by mechanical, chemical, behavioral and reflex mechanisms that are highly dynamic. These factors can disrupt the patient- ventilator interface because the ventilator responds to the patient's inspiratory and expiratory signals, which affect pressure and flow in the ventilator circuit.¹¹ Given patients' dynamic conditions during critical illness, patient-related factors such as respiratory center output, respiratory mechanics, disease states or conditions, and endotracheal tube type or size influence the patient-ventilator interaction (Table 1).

Respiratory center output. The patient's respiratory center output can produce a decreased or increased drive that may contribute to the development of PVD. Respiratory drive is dependent on both voluntary and autonomic control. Voluntary control is operationalized through the cerebral cortex (forceful inspiration/expiration, breath holding) and thalamus (emotional behaviors associated with intense feelings, fear, pain, anger, sorrow).¹² Autonomic control is operationalized by the brainstem,

Table 1 Patient and Ventilator-related Factors That Affect Patient-ventilator Interaction

Patient-related factors	Ventilator-related factors
<p><u>Respiratory Center Output:</u></p> <p>1. <u>Factors that may Decrease Respiratory Drive-</u> Sedative, Opioid, Hypnotic medications¹⁴ High level of ventilatory assistance¹⁶ Metabolic alkalosis¹⁴ Malnutrition¹⁴ Sleep deprivation¹⁴ Severe Hypothyroidism¹⁴ Idiopathic central hypoventilation syndrome¹⁴ Severe bilateral mid-to lower medulla brainstem injury¹⁴</p> <p>2. <u>Factors that may Increase Respiratory Drive-</u> Chemoreceptor stimulation (hypoxemia, hypercapnia, acidosis states)¹⁴ Increased ventilatory demand from:</p> <ul style="list-style-type: none"> • Increased metabolic states (pain, fever, shivering, overfeeding, sepsis, burns, trauma, hyperthyroidism, metabolic acidosis)¹⁴ • Underlying lung disease¹⁴ <p>Increased workload (weaning, stimuli that increase demand)¹⁴ Pain,¹⁴ Increased psychogenic stimuli or agitation¹⁴ Medications (Theophylline, Doxapram, Acetazolamide)¹⁴</p>	<p><u>Triggering:</u></p> <p>1. <u>Ineffective trigger-</u> a. Excessive Intrinsic Positive End Expiratory Pressure¹⁵ b. Delayed termination dyssynchrony^{27, 28} c. Maladjusted sensitivity level¹⁵</p> <p>2. <u>Auto-cycling-</u> a. Maladjusted sensitivity level¹⁵ b. Excessive water in circuit¹⁵ c. Air leaks in endotracheal tube cuff, ventilator circuit, chest tube¹¹ d. Cardiac oscillations⁴⁰</p> <p>3. <u>Double triggering-</u> a. Premature termination dyssynchrony¹¹</p>

<p><u>Respiratory System Mechanics:</u></p> <ol style="list-style-type: none"> 1. Prolonged patient inspiratory time¹⁰ 2. Shortened patient expiratory time¹⁰ 3. Weak respiratory muscles¹⁴ Poor neuromuscular control¹¹, Wean from high assist 	<p><u>Cycling off:</u></p> <ol style="list-style-type: none"> 1. Inappropriately set cycling variable for patient^{13, 28}
<p><u>Disease states and conditions:</u></p> <ol style="list-style-type: none"> 1. Obstructive Pulmonary Disease, Dynamic hyperinflation states causing increased Intrinsic positive end expiratory pressure¹⁴ 2. Acute Respiratory Distress Syndrome¹⁴ 3. Pain, splinting^{15, 16} 4. Body Posture, Abdominal distension¹⁶ 5. Psychogenic behavioral stimuli causes agitation¹⁴ from: <ul style="list-style-type: none"> • Pulmonary edema, Pulmonary emboli, Pneumothorax¹⁶ • Bronchospasm, Retained airway secretions¹⁶ • Intensive Care Unit Environment,¹⁷ Fear, Anxiety 	<p><u>Ventilator causes of patient agitation:</u></p> <ol style="list-style-type: none"> 1. Ventilator disconnection¹⁶ 2. System leak, Circuit malfunction¹⁶ 3. Inadequate fractional inspired oxygen¹⁶ 4. Inadequate ventilator support¹⁶
<p><u>Artificial Airway in place:</u></p> <ol style="list-style-type: none"> 1. Size, Shape,¹⁵ Narrowing of lumen by secretions¹⁸ 2. Cuff leak^{11, 14} 3. Disconnection from circuit¹⁴ 	<p><u>Dead Space:</u></p> <ol style="list-style-type: none"> 1. Increased dead space in circuit causes increased workload and work of breathing¹⁴

with the pneumotaxic and apneustic respiratory centers in the pons and 2 neuronal groups, called the dorsal (inspiratory) and ventral (inspiratory/expiratory) respiratory groups, located in the medulla.¹² Afferent neurotransmissions from the body are communicated to the medullary neuronal groups from multiple receptors throughout the body. Table 2 describes these receptors specifically and their influence on the autonomic control of respiratory drive. Once the medulla receives impulses, the inspiratory and expiratory centers respond by determining whether inspiration or expiration should be stimulated or inhibited. The pneumotaxic center of the pons fine tunes the rhythmicity of the ventilatory drive and the efferent message is sent to the phrenic and intercostal nerves to stimulate or inhibit ventilation.¹²

If respiratory drive is decreased, the ventilator may not be able to respond to the reduced effort, especially if the clinician does not preset the ventilator sensitivity level at a level that will detect the patient's effort.¹⁰ A reduced drive can be caused by sedatives, opioids and hypnotics. Sedatives, opioids and sleep all increase the time delay between the start of the patient's inspiratory effort and ventilator triggering.¹³ Metabolic states of alkalosis, sleep deprivation, severe hypothyroidism, and bilateral injury of the mid to lower medulla can decrease respiratory drive, thereby influencing the patient-ventilator interaction.¹⁴

If a patient's respiratory drive increases vigorously, however, the duration of inspiratory drive may become longer than ventilator inflation time, causing the ventilator to trigger more than once (double trigger).¹³ An increased respiratory drive can cause the patient to need more flow from the ventilator.¹⁴ If the pre-set flow does not meet patient demand, flow dyssynchrony can occur. An increased respiratory output can be

caused by increased chemoreceptor stimulation, pain, psychogenic alteration, medications and increased ventilatory demand, metabolic rate, and workload.

Respiratory system mechanics. A patient's respiratory mechanics can contribute to dyssynchrony. The patient may have a prolonged inspiratory time. If inspiratory time is longer than the ventilator's pre-set inspiratory time, the patient may take an additional breath because the need for ventilation has not been met, thereby causing double triggering.¹⁰

On the other hand, the patient may have a shortened exhalation time that increases the amount of intrinsic positive pressure at the end of expiration (PEEPi auto-PEEP) because all volume has not been exhaled. This leads to dynamic hyperinflation causing the patient to breathe with high lung volumes and high elastic recoil pressures.¹⁴ The excess pressure on the alveoli at the end of expiration (auto-PEEP) causes increased workload for the patient's diaphragm. Auto-PEEP is a common cause of failure-to-trigger PVD because the patient must overcome excess auto-PEEP by dropping intrathoracic pressure through muscular effort of sufficient magnitude to be sensed by the ventilator.

It is also important to consider the strength of the diaphragm and accessory muscles. Muscular strength may be decreased from diaphragm deconditioning (prolonged ventilator assistance, physical immobility), malnutrition and neuromuscular disease. Weakened respiratory muscles reduce the ability to meet the threshold necessary to initiate a ventilated breath during interactive modes.¹⁴

Table 2 Receptors and Their Effect on Autonomic Respiratory Drive¹²

	Peripheral Chemo-receptors	Pulmonary Stretch receptors	Irritant receptors	Pulmonary Juxtapulmonary Capillary receptors	Baro-receptors	Proprioceptors	Other receptors	Central Chemoreceptors
Location	Aortic arch Carotid bodies	Airway smooth muscle	Epithelium of nose, pharynx, larynx, trachea, bronchi	Pulmonary capillary walls	Aortic arch	Muscles and joints (tendon organs)	Nociceptors in skin, organs Mechano-receptors in skin	Ventro-lateral surface of each side of medulla
Sense	Hypoxemia Acidosis Hypercapnia	Lung inflation Increased pulmonary pressures	Inhaled irritants Mechanical factors: Anaphylaxis Pulmonary congestion	Pulmonary congestion Increased interstitial fluid Chemical irritants Microemboli	Change in blood pressure (BP)	Body movement	Pain Touch	H ⁺ concentration in CSF pCO ₂ crosses blood-brain barrier & yields H ₂ CO ₃ (acid)
Sent to medulla by	Vagus and Glossopharyngeal Nerve	Vagus Nerve	Vagus Nerve	Vagus Nerve	Vagus Nerve	Ascending Spinal Nerves	Ascending Spinal Nerves	Bathing Cerebral Spinal Fluid
Effect on respiratory drive	<i>If Acidotic or Hypoxemic:</i> Increased rate and tidal volume <i>If Alkalotic:</i> Decreased rate and tidal volume	Increased inspiratory Time Increased rate	Hyperpnea Cough Laryngospasm	Rapid, shallow breathing Hypoventilation	<i>If low BP:</i> Hyperventilation <i>If high BP:</i> Hypoventilation	Increased rate and tidal volume	<i>Visceral pain:</i> decrease drive <i>Somatic pain:</i> increase drive	<i>Acidosis:</i> Increased drive <i>Alkalosis:</i> Decreased drive

Abbreviations: CO₂= Carbon dioxide, BP= Blood Pressure, CSF= Cerebral Spinal Fluid, H₂CO₃= Carbonic Acid, H⁺= Hydrogen ion concentration caused by increased chemoreceptor stimulation, pain, psychogenic alteration, medications and increased ventilatory demand, metabolic rate, and workload.

Patient disease states and conditions. Underlying patient diseases and conditions can lead to PVD. For example, patients with obstructive lung disease, high ventilatory demand and patients receiving inverse-ratio ventilation or airway pressure release ventilation can have dynamic hyperinflation.¹⁴ Dynamic hyperinflation moves the diaphragm to a flattened, less dome-like configuration, which limits the patient's ability to generate a forceful inspiration and creates a greater trigger threshold to overcome, leading to failed efforts.¹⁴ Pain and splinting from surgical or other sources may reduce a patient's inspiratory effort and contribute to PVD.¹⁵

In addition, psychogenic mechanisms from agitation,¹⁴ fear and stress may contribute to PVD because of resulting tachypnea and reduced mental capacity from overriding anxiety. Disease and ventilator-related factors can also agitate patients (Table 1). Agitation in patients who are receiving mechanical ventilation can be exacerbated by many factors, such as disease states (pulmonary edema, pulmonary embolism, pneumothorax), body posture, abdominal distension, retained airway secretions, pain and bronchospasm.¹⁶ Last, the environment can be the source of noxious stimuli such as excessive noise, light, or physical stimulation that may contribute to the patient's stress response, thereby increasing anxiety and possible agitation.¹⁷

Artificial airway in place. Finally, the size and type of airway may contribute to PVD.¹⁵ An endotracheal tube of inadequate diameter may increase resistance and limit flow for the patient with high ventilatory demand. The internal diameter of the endotracheal tube can be reduced by the accumulation of secretions and debris that can markedly increase airway resistance.¹⁸ The patient- ventilator interaction is

dependent on the patient's ability to overcome this resistance and the amount of ventilator assistance.¹⁴

Ventilator-Related Factors

Synchronous patient-ventilator interaction requires a ventilator to be sensitive to respiratory efforts and responsive to airflow demand.¹¹ Two major factors contributing to PVD are ventilator triggering (signal opens inspiratory valve) and cycling (signal opens expiratory valve at end inspiration).^{11,19}

Ventilator triggering. The ventilator should ideally respond to a patient's inspiratory effort immediately. The sensitivity trigger is typically set to be stimulated based on pressure, flow or time. Unfortunately, in some situations, the sensitivity level may be set too low to sense the patient's effort. In this situation, the patient's effort may be wasted and not trigger a breath, which may lead to increased respiratory muscle loading.^{19,20} A prolongation of the trigger phase may also occur because of errors in the ventilator's pressure transducer, the ventilator's delay in sampling pressure signals, duration of time from onset of diaphragm contraction to actual decrease in airway pressure, duration of time from decrease of airway pressure to be sensed by the ventilator, and duration of time from when the valve is signaled to when flow reaches the airway circuit.¹¹ These factors will generate phase dyssynchrony.

In phase dyssynchrony, a lag (termed delay time) occurs between the time when the ventilator first senses the trigger and the time when the ventilator responds by delivering gas flow.¹⁵ When the inspiration trigger is driven by a change in pressure (pressure trigger), the delay time by most commercial ventilators may reach 110-120 milliseconds before gas flows into the patient's circuit.¹¹ If the inspiratory trigger delay

becomes too prolonged, the patient may try to increase his inspiratory efforts.¹¹

Therefore to avoid trigger dyssynchrony, it is best for the clinician to set the shortest trigger delay time, which will minimize patients' efforts to trigger an inspiration in interactive modes.¹¹

Newer ventilator modes to improve ventilator triggering. Traditional ventilators can only measure and respond to patient airway pressures at the airway opening and flow downstream on the expiratory limb of the ventilator circuit.^{21,22} The greater the distance from the central nervous system's respiratory center that the ventilator senses the trigger drive, the greater the potential for PVD.¹¹ New, experimental techniques offer different ways and sites to sense and respond to patients' inspiratory signals. Proportional assist ventilation enhances synchrony by using a feedback mechanism to "amplify airway pressure proportionally to inspiratory flow and volume".^{21(p.26)} This mechanism enables the ventilator to track changes in the patient's ventilatory effort, thereby resulting in a more physiologic breathing pattern.²¹ Other factors such as air leaks and water in the ventilator tubing can dampen the signal being sent from the patient to the ventilator during this interaction.¹¹

Neurally adjusted ventilatory assist senses the diaphragm's electric stimulation signal through an esophageal probe on the end of a nasogastric tube that sits close to the diaphragm.²³ The signal is amplified, filtered, and processed by the ventilator's software to then generate adjusted ventilation pressures specific to the patient's initiated diaphragmatic output.²⁴

Methods that are used less frequently may also be effective. Pdi-driven servoventilation adjusts ventilated pressure breaths in response to the patient's

transdiaphragmatic pressures (Pdi).²⁴ This ventilator is triggered through the Pdi or a preset flow threshold, whichever is generated first. Inspiration ends when the inspiratory flow reaches a preset threshold.²⁴ Shape-signal or flow-shape triggering²⁵ depends on a distorted expiratory waveform that is generated when the patient initiates a breath in order to sense a patient's inspiratory trigger. The ventilator algorithm generates a new flow signal that is offset from the patient's actual flow by 0.25 L/s and delays it for 300 milliseconds, thereby allowing the signal to lag behind the patient's actual flow rate so that, once the patient initiates a breath, the sudden decrease in expiratory flow will cross the generated shape signal to initiate a ventilated breath.¹³

Ventilator cycling off. Cycling off terminates the mechanical breath harmoniously when the patient desires to end inspiration and begin exhalation.¹³ Moving from inspiration to expiration on the ventilator is operationalized through volume, pressure, flow or time cycling. To achieve this, clinicians preset ventilator settings of (a) target volumes, (b) peak inspiratory pressures, (c) peak flow rates/flow shape signal, or (d) inspiratory time limit (T_i) or changes in inspiratory:expiratory ratio.¹⁹ The settings to obtain perfect synchrony between the end of inspiration and beginning of expiration, however, are not ideal. Usually termination of ventilator flow occurs either before or after the patient stops inspiratory effort.¹⁷

Premature termination. If a mechanical breath is terminated before the patient desires (premature termination), the patient continues to contract inspiratory muscles, allowing pressure to overcome elastic recoil resulting in the ability to meet the trigger threshold and initiate a new breath, called double triggering.¹³ Premature termination of ventilator flow causes excessive inspiratory muscle work into and during the expiratory

phase and an overestimation of respiratory rate.^{13,14} Nurses should be able to recognize that double triggering may be a cause of increased respiratory frequency.

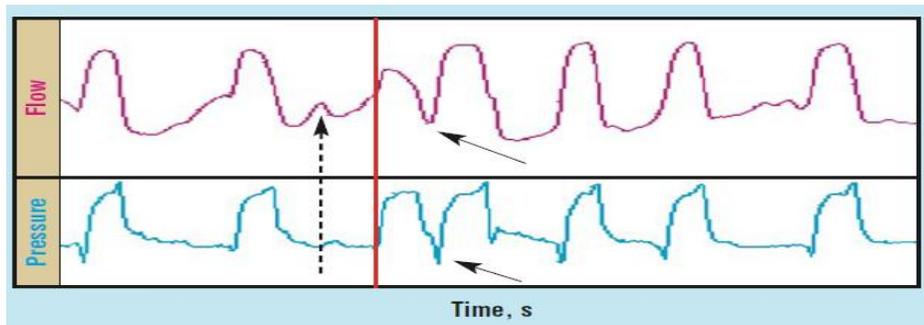


Figure 1. Trigger dyssynchrony, double trigger. Display of flow (top) and pressure (bottom) vs time. Note the start of the third successfully triggered breath (red line). In this breath cycle, patient effort initiates a second breath (solid arrows), represented by a quick decrease in pressure at the trigger threshold, such that flow increases. In addition, an ineffective missed effort (dashed arrow) just before the double trigger is demonstrated by an increase in flow and a decrease in pressure.

Delayed termination. On the other hand, if the mechanical breath does not terminate when the patient's muscular inspiration is complete (delayed termination), the time for exhalation is limited and expiratory workload and sometimes auto-PEEP increase, resulting in possible ineffective and/or failed trigger on the following breath.²⁶⁻²⁸ Delayed termination causes patients to resist or "fight" incoming ventilator flow using their expiratory muscles. This resistance results in increased expiratory load and excessive PEEP_i, thereby leading to possible pneumothoraces, barotrauma and altered cerebral blood flow.²⁶ Indeed, the perfect maneuver would be to program a ventilator breath-by-breath based on the patient's inspiratory time. Such a feature is currently not available on any ventilator, except for the Servo-i ventilator with the option

of neurally adjusted ventilatory assist by Maquet, Inc.® that was recently released commercially.²⁹

A second effect of delayed termination results in ineffective triggering after the breath. If ineffective triggering occurs during exhalation, inspiratory muscles will be contracting when they would normally be lengthening (plometric contraction), which has caused ultrastructural damage to muscle fibers and reduced strength in animal models.^{30,31} Although not studied in human models, muscle injury from ineffective triggering may prolong weaning.¹⁷

Patient ventilator interaction is complex and dynamic making the ideal interface elusive. Despite the advantage of interactive ventilator modes in promoting greater involvement of patients and limiting atrophy of respiratory muscles, a consequence is PVD.

MacIntyre & Branson,²² state ventilators need to be sensitive to patient ventilatory effort and responsive to patient demands.

Prevalence of PVD

PVD, a mismatch of respiratory cycling and flow of ventilation between the ventilator and patient, occurs frequently and is underappreciated.^{14,32-34} Thille et al³⁵ found a high prevalence of dyssynchrony in 62 patients intubated longer than 24 hours, and those with the most dyssynchrony had a longer duration of mechanical ventilation. Indeed, 24% of this sample experienced a significant amount of PVD, which was observed within thirty minutes of data collection.³⁵ The most frequent types were ineffective and double trigger with males and those with chronic obstructive pulmonary disease experiencing more ineffective trigger.³⁵

Use of continuous sedation during mechanical ventilation may prolong hospital stay and influence the prevalence of PVD. Because the assessment of sedation level is not precise, oversedation and undersedation is possible. A variety of approaches, such as daily interruption of sedation (“daily awakening trials”) or intermittent dosing of sedative and opioid medications can reduce the likelihood of excessive sedation.³⁶ However, the use of daily awakening trials may aggravate PVD as they become more alert, potentially placing high-risk patients in jeopardy for complications.

On the other extreme of the sedation scale, de Wit et al,³⁷ conducted a small study of 20 patients receiving mechanical ventilation and reported the incidence of ineffective triggering was significantly correlated with lower scores (i.e., patients more deeply sedated) on the Richmond Agitation Sedation Scale (RASS). This finding suggests that highly sedated patients may reduce their respiratory drive so much that they become ineffective in generating an inspiratory trigger sufficient to initiate a ventilated breath.

Consequences of Patient Ventilator Dyssynchrony

Patient-ventilator synchrony is important for achieving optimal oxygenation and ventilation. Tobin et al⁴ state that patient-ventilator interaction is a major factor in determining how much respiratory muscle unloading can be completed by the machine, and the most effective unloading occurs when the patient and ventilator are synchronous.³⁴ Achieving synchrony during interactive ventilator modes requires ventilator sensitivity and responsiveness to patient’s demands, which is not always possible with current ventilators. Potential outcomes of patient-ventilator synchrony include prevention of hypocapnia, decreased sensation of dyspnea, reduced ineffective

or wasted respiratory efforts, and reduced likelihood of periodic breaths during sleep.²⁰ In addition, prolonged mechanical ventilation and longer hospital stay due to continuous sedation may be avoided.

The resulting unbalanced breathing pattern from PVD can lead to hypoxemia, increased workload on respiratory muscles, cardiovascular compromise, and discomfort.^{11,14,38} In addition, evidence indicates that PVD may result in respiratory muscle injury.^{10,17} In one study,³⁵ patients with PVD had a longer duration of mechanical ventilation, whereas other reports¹⁰ have been unclear. Other specific effects and outcomes of PVD over time are not well known or documented. Because these consequences may aggravate an already existing critical illness and possibly increase ventilator related morbidity,³⁹ nurses should establish the goal of achieving patient-ventilator synchrony. To achieve these goals, nurses must learn how to analyze graphic waveforms for PVD, observe for manifestations of PVD such as those listed in Table 3, investigate and prevent possible causes of PVD, and work with the collaborative team to optimize ventilator settings, sedative titration and psychosocial support to patients.

Measurement/Identification of PVD

Types of Dyssynchrony and Contributing Factors

Categorization of types of dyssynchrony may enable clinicians to more to identify PVD readily and accurately. Nilsestuen & Hargett¹⁵ describe four major types of PVD that can be conceptualized within the different phases of a patient's assisted ventilated breath (Table 3).

Table 3: *Phases of an Assisted Ventilated Breath Associated with Patient-ventilator Dyssynchrony*¹⁵

Phase of an assisted ventilated breath	Type of patient-ventilator dyssynchrony
Patient initiates a breath	Trigger Asynchrony 1. Ineffective effort 2. Failure to trigger 3. Double triggering 4. Auto-triggering
Patient demands air flow from ventilator	Flow Asynchrony
End of inspiration and breath triggering	Termination Asynchrony 1. Premature 2. Delayed
End of expiration	Expiratory Asynchrony

Trigger dyssynchrony. The first phase of a ventilator-assisted breath is its initiation. The factors that influence this stage include patient factors (having an adequate respiratory drive and inspiratory effort) and ventilator factors (having adequate responsiveness to detect the signal-pressure or flow, ability to reach a pressure maximum, appropriately set trigger levels/adequate delay time, and ability to pressurize the circuit).¹⁵ To avoid trigger dyssynchrony, these factors need to be functional. Trigger dyssynchrony is a common category of PVD, and its types can range from failure to trigger to auto-triggering.

Failure to trigger occurs when the ventilator does not sense the pressure or flow trigger. This situation is the result of a poor respiratory drive or excessive PEEP_i that prevents the patient's effort being sent to the ventilator's sensor.¹⁵ Excess hyperinflation causes a larger pressure gradient, and the patient cannot usually overcome the trigger threshold. Therefore, the ventilator does not generate flow for the patient's initiated

breath. This same situation occurs in the patient with chronic obstructive pulmonary disease or when trigger levels are not adjusted appropriately by clinicians. Ineffective triggering is shown in Figure 2.

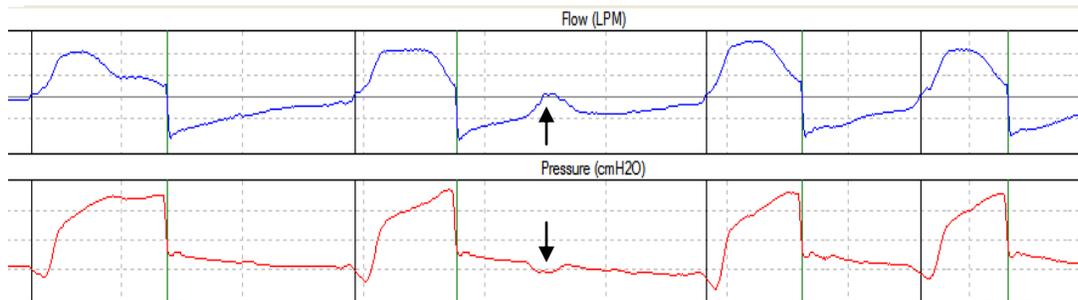


Figure 2. Trigger dyssynchrony, ineffective effort. Display of flow (top) and pressure (bottom) vs time. An ineffective effort, or failure to trigger dyssynchrony event is noted at the arrow. Note the negative deflection of the pressure waveform and the transient positive flow deflection after the second breath.

Double triggering occurs when the patient is triggering the ventilator excessively through an excess demand for flow or volume which the ventilator is not pre-set to deliver.¹⁵ Clinical situations that could lead to this phenomenon include sighs, coughing with breathing, and change in clinical status or inappropriate ventilator settings. Determining the cause of the double triggering is important in order to resolve PVD. If the cause is a temporary situation, such as excessive coughing, the ventilator may be disconnected for a short duration until the episode is over.¹⁵ If double triggering is noted, nurses should consult with the collaborative team to adjust ventilator flow or volume settings to meet the patient's demand (Figure 1).

Auto-triggering is caused by a maladjusted ventilator sensitivity level that recognizes signals other than the patient's initiation of a breath. Examples of triggers that could cause auto-triggering include random noise in the circuit such as water (increased resistance); leaks (i.e., circuit leaks, cuff leaks); and cardiogenic oscillations from patients with larger heart size, higher cardiac output and higher ventricular filling pressures.^{15,40} Factors that promote auto-triggering include are a low respiratory drive and decreased respiratory rate when hyperinflation is absent.¹⁷ Nurses should observe for causes of auto-triggering to prevent its occurrence and collaborate with the team to adjust the sensitivity level appropriately.

Flow dyssynchrony. The second step of inspiration is the demand for air flow. Flow dyssynchrony can occur when the ventilator is not set correctly for the patient's demand. Typically, the flow rate is set too low (eg 40 L/min) compared to the patient's inspiratory demand. One consequence of flow dyssynchrony is the creation of auto-PEEP; auto-PEEP is easily identified when the expiratory flow waveform does not return to zero before the next breath and a subsequent increase in peak inspiratory pressure.¹⁵ Flow dyssynchrony can occur in ventilated settings of volume- or pressure-cycled ventilation. During volume ventilation, the flow pattern is fixed and flow dyssynchrony can be identified by comparing the shapes of the pressure-time waveforms during complete passive breathing and patient-triggered breathing. A "dished out" appearance of the pressure wave during inhalation indicates flow dyssynchrony.¹⁵ (Figure 3)

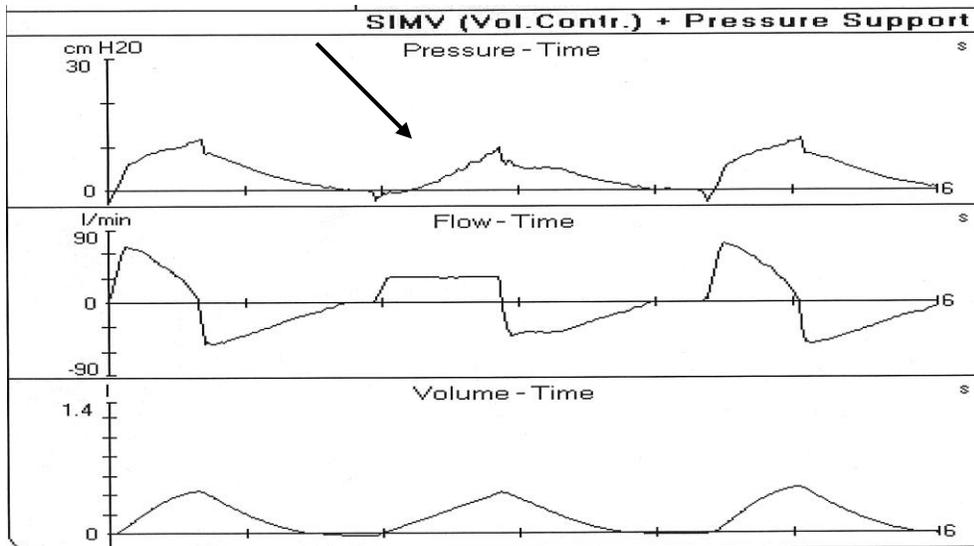


Figure 3. Flow dyssynchrony. Display of pressure (top), flow (middle), and volume (bottom) vs time. During second breath, the ventilator square wave flow is not adequate (pressure trace is dished out). Figure courtesy of Jon Nilsestuen, PhD, RRT, University of Texas Medical Branch, School of Allied Health, Galveston, TX

Termination dyssynchrony. Next in the cycle of a breath is the end of inspiration and breath triggering; it is at this point where termination dyssynchrony can occur.¹⁵ Basically, this type of PVD occurs in all ventilator modes because clinicians cannot set the perfect expiratory time based on the dynamic and changing patient's initiated inspiratory time. Du and Yamada²⁶ state that the most important factor to consider with this type of PVD is the length of time between the patient's inhalation effort and the end of ventilator flow. Termination PVD can be premature or delayed (Figure 4). In premature termination, the flow of air stops before the patient stops inhaling. In delayed termination, the patient is exhaling as the machine continues to deliver a breath.

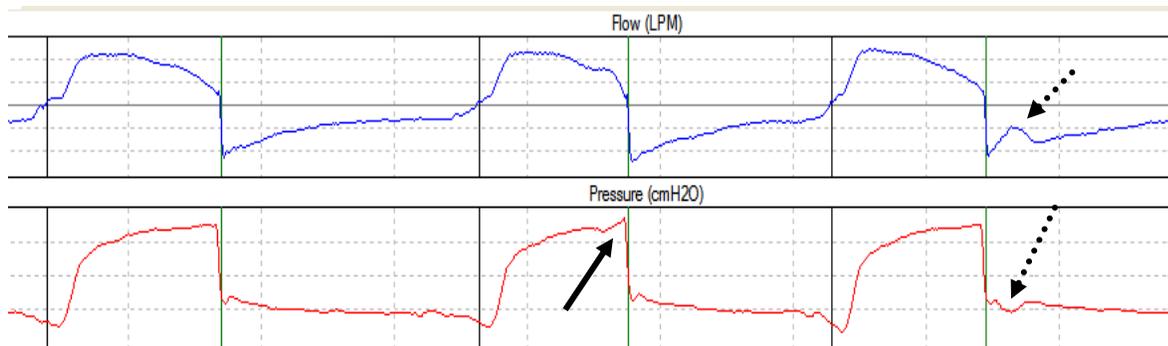


Figure 4. Termination dyssynchrony. Display of flow (top) and pressure (bottom) versus time reveals an example of “delayed termination dyssynchrony” (solid arrow) as the patient attempts exhalation before completion of the inspiratory breath, and “premature termination dyssynchrony” (dotted arrow) as the patient makes an inspiratory effort early in the expiratory phase.

Expiratory dyssynchrony. When the end of expiration is unmatched with the patient’s efforts, expiratory dyssynchrony can occur.¹⁵ During this time period, expiration may be shortened or prolonged. In the event of shortened expiration, the consequence can be air-trapping, auto-PEEP and a possible inability to reach the trigger threshold that leads to failure to trigger.¹⁵ On the other hand, prolonged expiration does not usually cause difficulties for the patient, unless the patient initiates a breath before the expiratory cycle is complete. Prolonged expiration may cause hypoventilation.¹⁵

Manifestations and Measures of PVD

Maintaining patient-ventilator synchrony in the critically ill patients is required to prevent hypoxemia, hypercapnia, cardiovascular compromise, and excessive or inadequate sedation. Thanks to their frequent contact with patients, nurses and

respiratory therapists are usually the first to observe that a patient is experiencing PVD. Although they may both detect changes in status, nurses and respiratory therapists may use different data to identify the change. Nurses describe patients who are “fighting the ventilator”,⁴¹ whereas respiratory therapists may be more likely to recognize PVD by noticing changes in the pressure/flow waveform.^{42,43}

PVD can be detected by noting changes in the volume, pressure and flow graphic waveforms displayed on the ventilator. Although PVD may be detected through waveform analysis,⁴⁴ it is not clear whether nurses evaluate these waveforms accurately or whether nurses use data obtained from waveform analysis in their. Indeed, Burns⁴⁵ reported that few clinicians are proficient in understanding and applying the waveform graphic findings at the bedside. On the other hand, nurses may more often use markers of physiologic instability and agitation as well as patients’ behaviors in general rather than pressure/flow waveforms to identify PVD. Table 4 summarizes the biobehavioral markers and experiences of patients with dyssynchrony. This table links the biological and behavioral manifestations with documented patient reports of PVD events in an attempt to more fully explain and understand this complex phenomenon.

Nasal flaring, forceful exhalation, use of accessory muscles, inspiratory intercostal retractions, paradoxical thoraco-abdominal movements and recruitment of accessory muscles in the neck may all indicate PVD.¹⁴ Biologic measures such as tachycardia, tachypnea, hypoxemia and real time graphic displays of airway pressures have also been used.^{14,15} In addition, patients’ behaviors such as agitation, coughing, or grimacing, as well as frequent ventilator pressure alarms alert nurses to PVD. However,

these patient behaviors and physiologic responses have not been validated as reliable measures of PVD.

The most widely used and objective measure of PVD is the pressure-flow graphic waveform; however clinical application by nurses is not widespread.⁴⁶ Therefore, in the absence of accessible, empirically based measures of PVD, the use of inadequate or excessive sedation may occur, leading to physiologic instability, and agitation, which may result in inappropriate use of sedation.

Table 4. Markers of Patient-ventilator Dyssynchrony

Biological markers	Behavioral markers	Patient reports of breathing with the ventilator
<ul style="list-style-type: none"> Pressure / Flow – time waveforms^{14, 15} 	<ul style="list-style-type: none"> Paradoxical thoraco-abdominal breaths¹⁴ 	<ul style="list-style-type: none"> <u>Difficulty with sensations of altered ventilation</u>: “Even on the ventilator, the rate didn’t bother me, it was the force of air”³⁸
<ul style="list-style-type: none"> Tachycardia^{14, 57} Tachypnea^{14, 58} Decreased oxygen saturation¹⁴ 	<ul style="list-style-type: none"> Nasal Flaring¹⁴ Expiratory muscle activity, forceful exhalation^{14, 57} Inspiratory inter-costal retractions¹⁴ Increased movement of extremities⁵⁹ Coughing⁵³ Agitation¹⁴ Abdominal excursions¹⁴ 	<ul style="list-style-type: none"> <u>Difficulty breathing independently (forced to adapt to the machine)</u>: “Sometimes, it’s too fast for you. So instead of the machine synchronizing with you, you have to synchronize with the machine”³⁸ <u>Realized if they relaxed, their experience was easier</u>: “I never had a feeling of suffocation like I thought I would if I became out of synch with the ventilator”³⁸ <u>Blamed clinicians for not providing information about being “out of synch”</u>: “I was able to, in my own sort of way, put two and two together and figure, as soon as I get in sync here, that will stop”³⁸ <u>Subjects desired to know more about dyssynchrony</u>: “To know that there may be a time where my

- Increased use of accessory muscles¹⁴ breathing and the ventilator's breathing are not going to match³⁸
 - Inspiratory effort without triggering the ventilator¹⁴
 - Subjects desired that nurses provide coping strategies: Patient's would prefer to know that "you can relax, and try to get back in sync with it, or let the nurses know that you're waking up"³⁸
-
- Discomfort , breathlessness, air hunger^{14, 17, 57, 60}

Nursing Implications to Improve the Phenomena of PVD

Monitoring of PVD

Correction of PVD remains a clinical priority. Achieving optimal patient-ventilatory synchrony is enhanced by recognizing the problem, accurately assessing the patient's behavior, adjusting various ventilatory parameters, and optimizing sedative therapy. It is noteworthy, however, that simply increasing the level of sedation, without identifying the cause of PVD or simply making ventilator adjustments, may unnecessarily prolong time receiving mechanical ventilation.⁴⁷ Although waveform analysis has been available over the past decade, the technology is underused.¹⁵ This underuse may be due to interpretation complexity, limited resources in ICU and limited educational preparation of nurses.

Burns⁴⁵ offers a technique to monitor continuous airway pressures using a transducer system connected to the side port of the ventilator circuit. This monitoring technique provides pressure/volume data similar to the data on the ventilator display panel offers a continuous view of the patient's pulmonary dynamics, including evidence of dyssynchrony, auto-PEEP and "breakthrough" respiratory efforts when sedatives and paralytics are used.⁴⁵ The primary benefit of its use is the ability to record and print

these graphics, allowing the novice time to identify and evaluate dyssynchronous patterns. Therefore uses of Burns' technique would augment real-time appreciation and management of PVD, much like the use of cardiac monitoring to detect and manage dysrhythmias.

Patient Ventilator Dyssynchrony as Part of Sedation Assessment

Because sedation is used to optimize the patient-ventilator interaction and reduce dyssynchrony, identifying PVD is paramount for optimal sedation management. However, no definitions of PVD have been uniformly accepted, and evaluation of ventilator synchrony has not been included in commonly used tools for evaluating sedation, such as Ramsay Scale⁴⁸, the Riker Sedation-Agitation Scale⁴⁹ or the RASS⁵⁰, which is primarily used to evaluate level of consciousness and agitation.

National organizations have stressed the need to evaluate and validate measures that ensure all goals of sedation are being met. The American Association of Critical Care Nurses has called for prospective studies to establish and study population-specific, goal-oriented sedation-agitation scales to enhance the consistency of caregiver observations and allow comparison of drug effects in adults.⁵¹ In addition, the Society of Critical Care Medicine's published clinical practice guidelines recommend that a sedation end point, using a validated sedation assessment scale, be regularly redefined with caregivers.⁶ In a systematic review of instruments for measuring the level and effectiveness of sedation in adult and pediatric ICU patients, DeJonghe et al.⁵² reported that although many instruments have been used to measure sedation effectiveness in ICU patients, none have been tested for their utility in detecting change

in sedation status over time (responsiveness), and few of them help clinicians assess sedation.

More recently sedation assessment tools have attempted to include an evaluation of PVD, using measurements of the patient-ventilator interaction. DeJonghe et al.^{52,53} used international focus groups of bedside nurses, residents and intensivists to identify PVD parameters to be included in their Adaptation to the Intensive Care Environment sedation tool⁵³ however, the characteristics chosen have not been tested for validity against graphic analysis of dyssynchrony and reliability has not been determined. Although the Adaptation to the Intensive Care Environment tool uses a 4-item scale for assessing “blockade of inspiratory phase of ventilation”, respiratory rate over 30/min, cough, and use of accessory muscles to assess ventilator synchrony, it is not clear if the inclusion is empirically based. A second tool, the RASS, uses a 10-item scale that is focused on level of consciousness and agitation, but it does include a behavior of “fights ventilator” as a criterion for agitation.⁵⁰

A consensus panel for the American Association of Critical Care Nurses has developed a sedation assessment tool that includes the evaluation of PVD.⁵⁴ This scale ranges from best (“appropriate physiological response achieved from patient-ventilator interface”) to worst (“patient-ventilator dyssynchrony with detrimental physiologic response”), although specific descriptions of physiologic response are not included. Even though newer tools for evaluating sedation are being developed that include measures of PVD, they remain insufficient to measure this phenomenon reliably. Measurement of PVD is a priority for optimal use of sedation; however, sedation scales either do not include it in the evaluation of sedation level or the proposed measure has

not been derived from an empirical foundation. Therefore, future nursing research is needed to identify the biobehavioral markers of PVD.

Collaboration With the Health Care Team

Collaboration between nurses, physicians and respiratory therapists to manage PVD is imperative. Each professional assumes a different responsibility in patient care and contributes valuable information to the team. Uniquely, each team member brings a special school of thought to the phenomenon of PVD. Respiratory therapists are skilled in knowing the details of ventilator operation and modes, assessment of patients' responses and detection of airway pressure monitoring. Physicians are skilled in these similar attributes, as well as therapeutic disease management of patients receiving mechanical ventilation. Nurses are specifically skilled in observing and managing human responses to disease and technology interface, but hold a prominent role in the coordination of patient care. Their role in understanding the larger picture greatly impacts patient outcomes.

It is imperative that nurses commence interventions for improvement of care, which start by recognizing PVD. Establishing routine monitoring of airway pressure and flow real-time waveforms for PVD in patients receiving mechanical ventilation by using continuous airway pressure monitoring would be a significant contribution. Commercial ventilator vendors may consider adding graph paper for printouts of real-time waveforms for analysis and documentation of PVD, similar to that of cardiac monitors used for rhythm strip interpretation. Automated techniques have been used in research to continuously detect ineffective and double triggering; when these methods are clinically realized, clinicians and their patients will benefit from the identification of

PVD.^{55,56} Table 5 describes how to identify different types of PVD, their causes, and nursing contributions for collaboration with the health care team.

Table 5 Identification, Causes, and collaborative Interventions for pPatient-ventilator dyssynchrony

Types/Waves	Cause	Collaborative Interventions
<p>Trigger (before inspiration)</p> <p><u>LOOK FOR:</u> 1. Ineffective trigger: Negative deflection during inspiration or expiration on pressure (P_{aw}) waveform with or without accompanying positive deflection in flow (V) waveform.^{14,61} 2. Double trigger: Within the same breath cycle, a second, stacked breath occurs after a very short inspiratory time on the P_{aw} waveform. The second breath within the cycle has a higher peak airway pressure and may exhibit an increased peak expiratory flow.¹⁴</p>	<p>1. Ineffective trigger attempt: Patients with low respiratory drive do not reach the trigger pressure in the correct time. Other factors: low P_{CO2}, high tidal volume and pH,³⁶ sedation,³⁸ high levels of assist (pressure support or assist control).^{11,35,36}</p> <p>2. Double triggering: Patient effort is greater than volume/flow delivery (inappropriate settings or sudden onset of increased patient demand),^{14,36} shortened ventilator inspiratory time and low ratio of P_{aO2} to fraction of inspired oxygen.³⁶</p> <p>3. Less sensitive trigger level setting.¹⁴</p> <p>4. Poor breathing cycle dynamics: Patient does not receive a positive inflation after inspiratory delay time (due to excessive auto-PEEP, pressure drop across endotracheal tube, COPD).¹⁴</p> <p>5. Ventilator unable to supply flow to pressurize the circuit.¹⁴</p>	<p>1. Ineffective trigger attempt: Assess respiratory drive (neuromuscular/chemical) and correct as able. Evaluate for over-sedation and reduce sedative infusion rate.^{11, 61} Determine if patient is receiving too much assistance, may adjust pressure support ventilation to decrease tidal volume.¹¹</p> <p>2. Double triggering: Adjust flow or volume settings to meet the patient's demand.¹⁴ Consider source of hypoxemia and provide measures to optimize oxygen saturation. Consider sedation.</p> <p>3. Increase trigger sensitivity setting without causing auto-triggering, consider flow-based trigger (may reduce trigger delay <100 ms).¹¹</p> <p>4. Evaluate for auto-PEEP in the expiratory waveform.¹⁴ Add PEEP cautiously (moves the ventilator trigger level closer to the patient's intrinsic airway pressure).^{14,35,61} Optimize use of bronchodilator to reduce PEEP.¹¹ Consider use of flow waveform triggering if auto-PEEP present.⁶¹</p> <p>5. Adjust the slope setting by increasing Inspiratory time and/or flow.</p>
<p>Flow (during inspiration)</p> <p><u>LOOK FOR:</u> 1. Concave appearance during inspiratory phase in the P_{aw} waveform.¹⁴</p>	<p>1. An increased patient respiratory effort causes ventilator to not match the patient's demand.¹⁴</p> <p>2. In volume-controlled ventilation: Incorrect peak flow setting for patient.¹⁴</p> <p>3. In pressure-controlled ventilation: A patient's flow demand and the rate of flow delivery.¹⁴</p>	<p>1. Evaluate pressure time waveform to differentiate this from trigger dyssynchrony. Assess neural drive to breathe, determine causative factors (undersedation, agitation, disease) and manage appropriately.</p> <p>2. Volume-controlled ventilation: "Adjust peak flow setting until patient's demand met, evidenced by pressure waveform resembling that of passive breathing"^{14(p213)}</p> <p>3. Pressure-controlled ventilation: Finely adjust rise time (rate of valve opening) to maximize flow-pressure waveforms.¹⁴</p> <p>4. Consider dual control modes¹⁴</p> <p>5. Consider size and diameter of endotracheal tube</p>
<p>Termination (inspiration terminated when cycling variable reached)</p>	<p>1. Premature termination: During pressure-support ventilation: Caused by low levels of support, short time constant (rate of flow decay influenced</p>	<p>1. Evaluate with flow and pressure time waveforms.¹⁴ May be difficult to see delayed termination during pressure support.⁶²</p> <p>2. Monitor for double triggering in premature</p>

<p><u>LOOK FOR:</u></p> <p>1. Premature: A concavity in the P_{aw} occurs with prolonged high flow during breath, followed by breath with rapid flow deceleration in convex shape.¹⁴</p> <p>2. Delayed: A spike at end of the breath will occur on P_{aw}, coinciding with rapid decrease in flow¹⁴ (patient attempts to exhale).</p>	<p>by resistance and compliance factors of respiratory system²⁹ and dynamic hyperinflation.⁶² During assist volume control: Caused by settings that yield short inspiratory time.⁶²</p> <p>2. Delayed termination: May be more prevalent in patients with COPD⁶²</p> <p>Pressure-support ventilation settings that result in a long time constant (COPD, high pressure support level and low flow threshold for cycling off variable).⁶²</p> <p>Assist-control ventilation settings that result in long inflation time (high tidal volumes, low inspiratory flow).⁶²</p>	<p>termination and missed trigger efforts (ineffective trigger) during delayed termination.^{11,18}</p> <p>3. For premature termination: Consider decreasing the flow threshold in patients with acute respiratory distress syndrome.⁶²</p> <p>4. For delayed termination: Determine if auto-PEEP is present by using expiratory hold maneuver.</p> <p>5. If auto-PEEP is present, consult with team to consider adjusting trigger sensitivity, peak flow, flow pattern, inspiratory time, breath-termination criteria, expiratory time, or respiratory rate, depending on the ventilation mode.¹⁴</p> <p>6. Use a sedation scale to consider sedation and neuromuscular relaxants to weaken patient's effort.²⁹ Monitor patients for ineffective triggering, especially if auto-PEEP is present.</p>
<p>Expiratory (on expiration)</p> <p><u>LOOK FOR:</u></p> <p>1. Expiratory flow waveform does not return to baseline.¹⁴</p>	<p>1. Common phenomena because neural (patient) inspiratory time varies, which affects termination and timing of expiration.</p> <p>2. Premature and delayed termination²⁹</p>	<p>1. Monitor for auto-PEEP by using expiratory hold technique. Adjust trigger sensitivity, peak flow, flow pattern, pressure slope, inspiratory time, breath termination criteria, expiratory time or respiratory rate, depending on mode.¹⁴</p> <p>2. Monitor for trigger dyssynchrony if auto-PEEP is present. If present, consider applying PEEP so as to equal the amount of auto-PEEP.^{13,14}</p>

Last, teaching patients receiving mechanical ventilation how to become acclimated with the ventilator is important. Explaining and realizing sensations that patients may experience is essential. Nurses do not know how the circumstances of the “normal” ventilator experience affect the condition of PVD; however, nurses should provide psychosocial support and maintain excellent communication with patients, especially when other caregivers are in the room.

Nurses may need to use coaching strategies to help patients develop a breath pattern until the cause is found. Patient stressors and the critical care environment should be assessed to determine the degree of conditions that could be changed for the patient's benefit during PVD. Excessive environmental or psychological stressors may affect the interaction between patient and ventilator. Accordingly, early and accurate

identification of PVD will enhance optimal use of sedative therapy and reduce duration of mechanical ventilation.

Future Directions for Research

The collaborative health care team is contributing to the advancement of knowledge in the realm of PVD. Physicians are experimenting with different triggers and modes, and respiratory therapists are identifying strategies to detect PVD and manage it. Nursing literature on this topic are scarce, yet nurses have much to contribute. We plan to describe the biobehavioral markers of PVD, through direct observations and continuous data recordings of heart rate, respiratory rate, end tidal carbon dioxide and oxygen saturation using continuous airway pressure monitoring to detect dyssynchrony. The hope is to recognize those manifestations that can be assessed to detect PVD. New questions about PVD can be raised (Table 6).

In conclusion, clinicians are challenged to recognize PVD and treat it appropriately. Collaborative team work will resolve the identification and treatment of PVD.

Table 6. *List of Potential Research Questions for the Future Direction of Nursing Research of PVD*

What is the effect of daily interruption of sedative in high risk patients on the incidence of PVD?
What is the effect of stress and environmental factors on PVD?
What is the relationship of dyspnea to PVD?
How do nurse's use airway pressure monitoring to detect PVD?
What are the differences in reported practices of nurses, physicians and respiratory therapists in recognizing and managing PVD?
What is the lived experience of the patient who has ventilator dyssynchrony?
What types of PVD occur during sleep / wake cycles in the critically ill mechanically ventilated patient?

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