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Bedside detection of patient-ventilation asynchrony

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ABSTRACT

Patient-ventilator asynchrony is common but under-recognized and under-reported. The frequency of PAV is reported around 23%, but up to 93% of patients have at least one episode of PVA. While sporadic asynchronies may have uncertain clinical impact, when they amount to more than 10% of the total breaths, PVA can increase the need for sedation and reduce sleep quality. In addition, they can impact on outcome by prolonging mechanical ventilation and increasing both ICU and hospital mortality.
The purpose of this review is threefold: 1) to characterise different types of patient-ventilator interaction; 2) to describe mechanisms leading to asynchrony; and 3) to describe ventilator modification to reduce patient-ventilation asynchrony.

Key words: ventilator waveforms, asynchronies, patient-ventilator interactions

INTRODUCTION

During assisted or supported breathing, the total pressure applied to the respiratory system to overcome the patient's inspiratory pressure load and generate inspiratory flow is shared between the patient's muscular pump and the mechanical ventilator. Any discrepancy between the volume, timing and duration of the set mechanical breath and the magnitude of the support required by the patient, will fail to relieve the work of breathing and can have important consequences in terms of dyspnea, sleep quality and outcome, including mortality. (1-6)
While traditionally, patient-ventilator asynchrony (PVA) has been detected by oesophageal and gastric pressure monitoring, the bedside clinician can detect such asynchronies less invasively by observing the flow and pressure waveforms that are routinely displayed on mechanical ventilators, as well as inspecting the patient's breathing pattern. (3, 5, 7, 8)
Patient-ventilator asynchrony is common but under-recognized and under-reported. PVA can occur with every mode of ventilation and in every phase of breathing. The frequency of PAV is reported around 23%, but up to 93% of patients have at least one episode of PVA. In patients on non-invasive ventilation (NIV), a high prevalence of PVA has been reported (58% of patients). Trigger asynchrony is the most frequent PVA reported (26-86% of mechanically ventilated patients). Double triggering and cycling asynchrony each account for around 10% of PVA. While sporadic asynchronies may have uncertain clinical impact, when they amount to more than 10% of the total breaths, PVA can increase the need for sedation and reduce sleep quality. In addition, they can impact on outcome by prolonging mechanical ventilation and increasing both ICU and hospital mortality. (7-11)
The purpose of this review is threefold: 1) to characterise different types of patient-ventilator interaction; 2) to describe mechanisms leading to asynchrony; and 3) to describe ventilator modification to reduce patient-ventilation asynchrony.

MECHANISMS OF PATIENT-VENTILATION ASYNCHRONY

Three variables determine a breath delivery on a mechanical ventilator: 1) breath initiation: trigger (the signal initiating pressure/flow delivery); 2) flow delivery: the control variable (the algorithm that controls the delivery of flow/pressure during inspiration); 3) breath termination or cycling off variable (the signal terminating the delivery of flow or pressure).
When interpreting ventilator waveforms, it is important to differentiate between dependent and independent variables, based on the set mode of ventilation. (1, 3-5, 11-17)

Table 1. Patient – ventilator asynchrony in relation to the different phases of breathing

Phase of assisted ventilated breath	PVA
Initiation of breathing	Trigger asynchrony: Delayed triggering Ineffective triggering Double triggering Auto Triggering Reverse triggering or 'entrainment'
Gas Delivery	Flow asynchrony
End of Inspiration	Termination asynchrony Premature cycling Delayed cycling

TRIGGER DELAY AND INEFFECTIVE INSPIRATORY TRIGGER (IIE)

The triggering time delay is the time from onset of triggering effort to the onset of flow delivery. Ineffective trigger is an inspiratory effort that does not trigger the ventilator. Ineffective triggering (or wasted effort) is one of the most frequent asynchronies (88% of all asynchronous breaths). IIE can be seen during mechanical expiration as well as during inspiration. In volume assist IIE, it can be detected by observing the pressure waveform; while in pressure support ventilation (PS) it is best observed in the flow waveform. An abrupt increase in flow suggests IIE.

IIE is generally caused by dynamic hyperinflation with intrinsic PEEP (PEEPi) -generated by low elastic recoil; high tidal volume and short expiratory time.

In the presence of PEEPi the respiratory system fails to reach equilibrium at the end of expiration. The elastic threshold load (PEEPi), is imposed on the respiratory muscles at the beginning of inspiration requiring additional efforts (pressure) to overcome PEEPi.

The ventilator can trigger inspiration only after PEEPi is overcome. The time spent by the respiratory system to dissipate the elastic threshold load caused by PEEPi causes the delay between the initiation of inspiration (muscle contraction) and triggering. If PEEPi is higher or the patient is weak or too sedated, the PEEPi is not overcome and the respiratory effort is unable to cause triggering. (3, 6, 8, 11, 18)

Modification of the waveforms:
Ineffective inspiratory Efforts

- Flow: positive deflection of the expiratory flow waveform
- Pressure (less sensitive): negative deflection during pressurisation and inspiration.

Trigger delay

- Flow: positive deflection of the expiratory flow
- Pressure (less sensitive): the time between the negative deflection of flow waveform and beginning of pressurisation.

Causes:

- Low respiratory drive
- Low PaCO2
- High tidal volumes
- Over-sedation
- High level of assistance
- PEEPi

How to correct IIE:

- Decrease PEEPi: Lower tidal volume or decrease the level of pressure support,
- particularly in COPD
- Lengthen the expiratory time
- Decrease resistance of the airways (i.e., bronchodilators)
- Decrease sedation levels (10)
- Increase external PEEP
- Decrease trigger threshold (increase trigger sensitivity). The trigger should be as sensitive as possible without causing auto-triggering (see below).
- Assess respiratory drive
- Use larger endotracheal tube (generating greater pressure to overcome increased airway resistance from smaller tubes requires time and greater effort)
- Use a 'flow-waveform' trigger system

AUTO-TRIGGERING

These are mechanical breaths delivered by the ventilator in the absence of the patient's triggered inspiratory effort. Auto-triggering causes discomfort and can increase PEEPi.

Causes:

- Any distortion in flow/pressure in the presence of low respiratory drive and respiratory rate in the absence of PEEPi;
- Low threshold trigger
- Circuit leaks
- Water in the circuit
- Vigorous cardiac oscillations (19)
- Hiccups

Modification of waveforms:

- Inspiration is not preceded by a pressure drop below PEEP
- Variation in peak inspiratory flow in the absence of dynamic hyperinflation
- Breath by breath variability in tidal volume
- Variation in shape of flow waveform (Flow– time)

DOUBLE TRIGGERING

Double triggering is defined as two consecutive inspiratory cycles not separated by an expiration, occurring within a timeframe of less than half of the mean inspiratory time. If the patient's respiratory drive increases, the duration of inspiratory effort becomes longer than the mechanical inflation time. This will cause the ventilator to be triggered more than once within the same neural inspiration. Therefore, double triggering is multiple consecutive breath cycles in which only the first is triggered by the patient. The tidal volume of the second breath will be smaller than the first in pressure support, while in volume assist ventilation, the tidal volume will be the similar to the first breath, and will be associated with higher airway pressure

Causes:

- Patient's effort is greater than the support delivered
- Longer neural inspiratory time compared to the to the inspiratory time of the mechanical breath
- Oversensitive trigger settings
- Ventilator unable to pressurise the circuit appropriately

REVERSE TRIGGERING

Reverse triggering – or respiratory – entrainment, is a phenomenon that occurs when a ventilator insufflation elicits a diaphragmatic contraction and a spontaneous breath with a ration varying between 1:1 to 1:3. (20, 21)

PHASE OR CYCLE ASYNCHRONY

A phase/flow/cycle asynchrony occurs when the delivery of a mechanical breath is inappropriate for the patient's demand. In volume assist, inadequate flow delivery can cause redaction in the instantaneous pressure-time airway curve giving a 'concave' or 'sucked down' appearance of the assisted breath (in comparison with the controlled breath), indicating that the flow provided by the mechanical breath is lower than required for muscle unloading.

The pressure waveform will then decrease with any increase of the patient inspiratory effort. In PS/ PC ventilation the above considerations apply to the flow waveform. In addition, the respiratory efforts during inspiration determine the duration of inspiration, as the inspiration time in PS is dependent on the decline of inspiratory flow up until the cycling off criterion - set as a percentage of peak inspiratory flow. In PS, two signs may indicate flow asynchrony: 1) a 'square' or constant inspiratory flow

waveform appearance caused by large continuous inspiratory effort; 2) changes in tidal volume for the same driving pressure – the larger the tidal volume, the better the synchrony.
In contrast, excessive flow or pressure can overwhelm the respiratory centre and cause episodic breathing or an abrupt termination of inspiration, with early activation of expiratory muscles. (1, 2, 9, 11, 12, 22-24)

EXPIRATORY TIME ASYNCHRONIES:

Cycling from inspiration to expiration is determined – depending on the mode of ventilation-by time, volume or flow. In pressure support mode, the transition from inspiration to expiration (cycling) occurs when the inspiratory flow decreases to a set proportion (usually 25%) of the peak inspiratory flow, so that the inspiratory time is dependent on the effort. In ideal conditions, cycling coincides with the end of neural inspiration. However, these signals rarely completely match the patient's own neural inspiratory time. As a consequence, the mechanical inspiration can be longer (i.e., terminated later – delayed cycling) or shorter (i.e., terminated earlier- premature cycling) than the neural inspiratory time. (1, 2, 9, 11, 12, 22-24)

PREMATURE (SHORT) CYCLING

Premature cycling, occurs when there is persistence of an inspiratory flow after termination of a mechanical breath. This indicates a shorter mechanical inspiratory time (premature cycling) - compared to the neural inspiratory time. The patient will continue to contract inspiratory muscles during mechanical expiration. The effort reduces airway pressure and increases flow, resulting in a 'notch' in the early part of the expiratory flow with a decrease in the peak expiratory flow. In addition, inspiratory muscle activation can trigger inspiration – resulting in double-triggering.

Waveform identification of premature cycling

- Flow: positive inflection at the end of expiration
- Pressure: Negative deflection 'notch' at the end of expiration

Causes:

- Low level of PS
- Short time constant (low lung compliance)
- Setting a short cycling off criterion
- Dynamic hyperinflation - which reduces expiratory flow and therefore causes early termination of inspiration.

DELAYED CYCLING

Occurs when the ventilator breath cycle is longer than the patient's inspiratory time. If the mechanical breath persists after completion of muscular inspiration, the airway pressure and flow waveforms will reflect the summation of two opposing forces: the mechanical inspiration and the activation of expiratory muscles – giving the appearance of the patient fighting the ventilator. This asynchrony can be difficult to identify. Delayed cycling can exacerbate dynamic hyperinflation and as a consequence cause triggering asynchronies.

Waveform identification of delayed cycling in pressure support

- A rapid decrease in inspiratory flow
- An increase in airway pressure at the end of inspiration (spike) due to a combination of abrupt relaxation of the inspiratory muscles and active contraction of the expiratory muscles.
- The increasing pressure can itself cause an inspiratory termination

Causes:

- Excessive PS
- Long expiratory time constants

In patients with acute respiratory distress syndrome with low compliance and short expiratory time constant, increasing the pressurisation rate (ramp or rising time) has a greater impact on reducing work of breathing compared to adjusting the cycling off criterion. In contrast, in COPD increasing the cycling off criterion (earlier cycling) has a significant impact on lung mechanics, reduction of PEEPi and work of breathing. (6, 8, 25, 26)

CONCLUSION

During assisted mechanical ventilation, patient and ventilator interaction needs to be monitored. Asynchronies are prevalent and if frequent can affect important patient-centred outcomes including mortality. A systematic approach to detection of common abnormalities in the flow and pressure waveforms can allow the clinician to diagnose and correct asynchronies. Holistic management of sedation, sleep and ventilator settings is required to harmonise the interaction with the ventilator and aid liberation from mechanical ventilation. Newer modes of ventilation (e.g., PAV+ - based on analysis of respiratory mechanics; and NAVA – based on neural respiratory output), that are designed to detect inspiratory muscle activity may improve synchrony and support the breath in proportion to the patient's efforts.

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