Modulation and treatment of patient–ventilator dyssynchrony

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Purpose of review

The coupling between ventilator delivered inspiratory flow and patient's demands both in terms of timing and drive is a challenging task that has become largely feasible in recent years. This review addresses the new advances to modulate and treat patient–ventilator dyssynchrony.

Recent findings

Patient–ventilator dyssynchrony is a common phenomenon with conventional modes of mechanical ventilation which influence the duration of mechanical ventilation. Inspection of pressure, volume and flow waveforms represents a valuable tool for the physician to recognize and take the appropriate action to improve patient–ventilator synchrony. New developments have been introduced aiming to improve patient ventilator synchrony by modulating the triggering function and the variables that control the flow delivery and the cycling off.

Summary

Patient–ventilator dyssynchrony may affect patients' outcome. New modes of assisted mechanical ventilation have been introduced and represent a major step forward in modulating patient–ventilator dyssynchrony.

Keywords

assisted modes, cycling off, triggering, ventilatory demands

Curr Opin Crit Care 13:84–89. © 2007 Lippincott Williams & Wilkins.

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Current Opinion in Critical Care 2007, 13:84–89

Abbreviations

AVC	assist volume control
NAVA	neurally adjusted ventilatory assisted
PAV	proportional assist ventilation
P_{di}DV	P_{di}-driven servoventilation

1070-5295

Introduction

Patient–ventilator dyssynchrony is referred to as the uncoupling of the mechanical delivered breath (ventilator) and neural respiratory effort (patient). It is commonly associated with all the conventional modes of assisted mechanical ventilation and influenced by factors related both to the patient and the ventilator [1•]. Patient–ventilator dyssynchrony imposes an additional burden on the respiratory system and may increase the morbidity of critically ill patients [2••]. Inspection of pressure, flow and volume waveforms – provided by the modern ventilators – represents a valuable tool for the physician to recognize and take the appropriate action to improve patient–ventilator synchrony [3••]. In addition, new modes of assisted mechanical ventilation have been introduced aiming to improve the patient–ventilator synchrony by modulating the triggering function and the variables that control the flow delivery and the cycling off [4•].

Patient–ventilator asynchrony during the triggering phase

During assisted modes of support initiation of mechanical breath occurs when a specific condition, determined by the triggering variable, is met. The triggering variable may be pressure, flow, volume and flow waveform [1•,5•,6–9]. Recently two new modes for triggering have been introduced that utilize the diaphragmatic pressure (P_{di}-driven servoventilation; P_{di}DV) and the electrical activity of the diaphragm (neurally adjusted ventilatory assisted; NAVA) as triggering signal [10,11]. With P_{di}DV each breath is triggered by either a preset P_{di} change or a preset flow threshold whichever occurs first [10]. With NAVA the ventilator is triggered when diaphragmatic electrical activity exceeds a predetermined threshold [11]. Currently these two methods remain experimental and are not available for general use.

Patient ventilator asynchrony during the triggering process is expressed in the forms of autotriggerings (triggering in the absence of inspiratory muscle contraction), excessive triggering delay (the delay between the beginning of inspiratory effort and ventilator triggering) and ineffective efforts (the inability of the patient inspiratory effort to trigger the ventilator) [1•,3••].

Autotriggering

Autotriggering is a well known phenomenon inherent to all currently used triggering methods [12,13]. It may result from random noise in the circuit, presence of water

in the circuit, leaks and cardiogenic oscillations [12,13]. Autotriggering usually occurs in patients with low inspiratory drive and breathing frequency, relatively high stroke volume and no dynamic hyperinflation [3••].

Inspection of pressure and flow waveforms may help to identify autotriggering [3••]. The absence of the initial pressure drop bellow end-expiratory pressure is indicative of autotriggering. With flow triggering systems, however, the pressure drop before the mechanical breath may be minimal if the resistance of the ventilator circuit is very low, making the signal less clear [3••]. Triggering occurring synchronously with cardiogenic oscillations, or gross difference between adjoining breaths, may also indicate autotriggering [3••]. Autotriggering is minimized by increasing the threshold of triggering in flow and pressure methods of triggering, augmenting the patient respiratory drive (i.e. by decreasing the sedation level or increasing Pa_{CO2}) and eliminating the leaks.

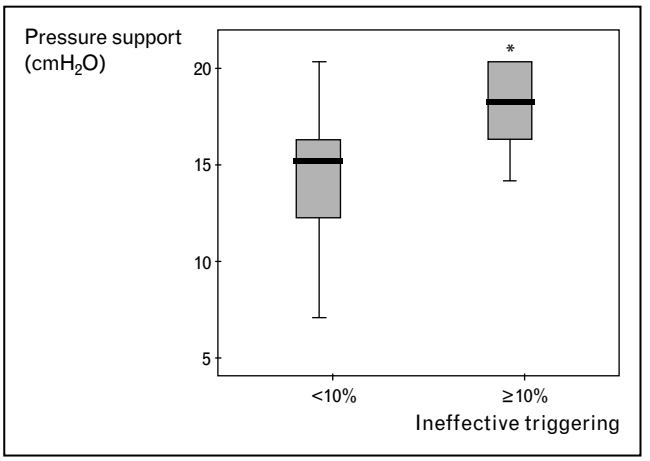
Excessive triggering delay and ineffective efforts

These events are very common and may be detected by the inspection of flow-time or airway pressure–time waveforms [3••]. The point of abrupt decrease in expiratory flow (and to a lesser extent in P_{aw}) signifies the beginning of the triggering phase. The time lag between this point and the point at which P_{aw} starts to increase is the triggering delay [3••,5•]. If the abrupt decrease in expiratory flow is not followed by mechanical breath ineffective effort occurs. Ineffective efforts may also occur during the mechanical inflation phase [2••,3••,5•]. These efforts are identified by observing an abrupt increase in inspiratory flow with pressure support or a transient abrupt decrease in P_{aw} with assist volume control (AVC), although with the latter mode this distortion is not always easy to recognize [3••].

Excessive triggering delay and ineffective efforts are caused by common factors. These are related both to ventilator function and the patient's characteristics of respiratory system [3••,5•]. Dynamic hyperinflation, low respiratory drive and weak inspiratory muscles are the main patient-related factors [14]. Paradoxically, the most important ventilator factors that contribute to triggering delay and ineffective efforts are not linked to triggering function of the ventilator but related to events during the pressure delivery and termination phases. High assist level (Fig. 1) and expiratory asynchrony in the form of delayed opening of exhalation valve (see below) are the ventilator factors which are usually associated with triggering delay and ineffective efforts [2••,15,16].

Earlier studies have shown that inspiratory muscle effort during the triggering phase is significantly lower with flow than that with pressure triggering system [17–19]. In the new generation ventilators, however, the flow and pressure triggering are equally effective systems in terms of modulating the triggering delay and ineffective efforts [20–23]. On the other hand it has been shown that the flow-waveform method of triggering modestly decreases the triggering delay and the number of ineffective efforts [8]. Nevertheless, in the presence of severe airway obstruction and very low expiratory flow the efficiency of this method is reduced due to system technical features [8]. Obviously, excessive triggering delay and ineffective efforts are not an issue with the experimental modes of NAVA and P_{di}DV, for which mechanical inflation occurs almost immediately after the initiation of the patient's effort [10,11].

Figure 1 Relation between the level of pressure support and the incidence of ineffective efforts



Notice that pressure support is higher in patients with a high incidence of ineffective efforts. Reproduced with kind permission of Springer Science and Business Media [2••].

The strategies for decreasing the triggering delay and the number of ineffective efforts are measures that decrease the magnitude of dynamic hyperinflation (e.g. use low tidal volume and long expiratory time, decrease the expiratory resistance, decrease the time that mechanical breaths extend into neural expiration) [2••,3••,16]; interventions which increase inspiratory muscle pressure during the triggering phase (e.g. decrease in sedation level, correction of alkalemia) [2••]; application of external positive end-expiratory pressure (PEEP), which narrows the end-expiratory difference between alveolar pressure and opening airway pressure; decrease the pressure or flow threshold for triggering; and the use of the new generation ventilators (with an average trigger delay between 100–120 ms) [22].

Patient–ventilator dyssynchrony during the pressure delivery and cycling off phases

With the conventional assisted modes of mechanical ventilation (AVC, pressure support), considerable

patient–ventilator asynchrony has been observed, mainly due to dissociation between the patient's respiratory effort and ventilator pressure both in terms of timing (uncoupling between the end of mechanical and neural inspiration) and inadequate or excessive assist [14,24]. In addition with pressure support the pressure rise time (defined as the time that pressure reaches the preselected threshold) may influence the synchronization between the patient and the ventilator [25–28]. In some of the new generation ventilators the rise time is adjustable and this represents a step forward in modulating patient–ventilator dyssynchrony. Rules for setting an optimal rise time, however, are lacking. Notwithstanding this, we should note that studies have shown that both very high and very low rise time may be associated with increased work of breathing [25–28].

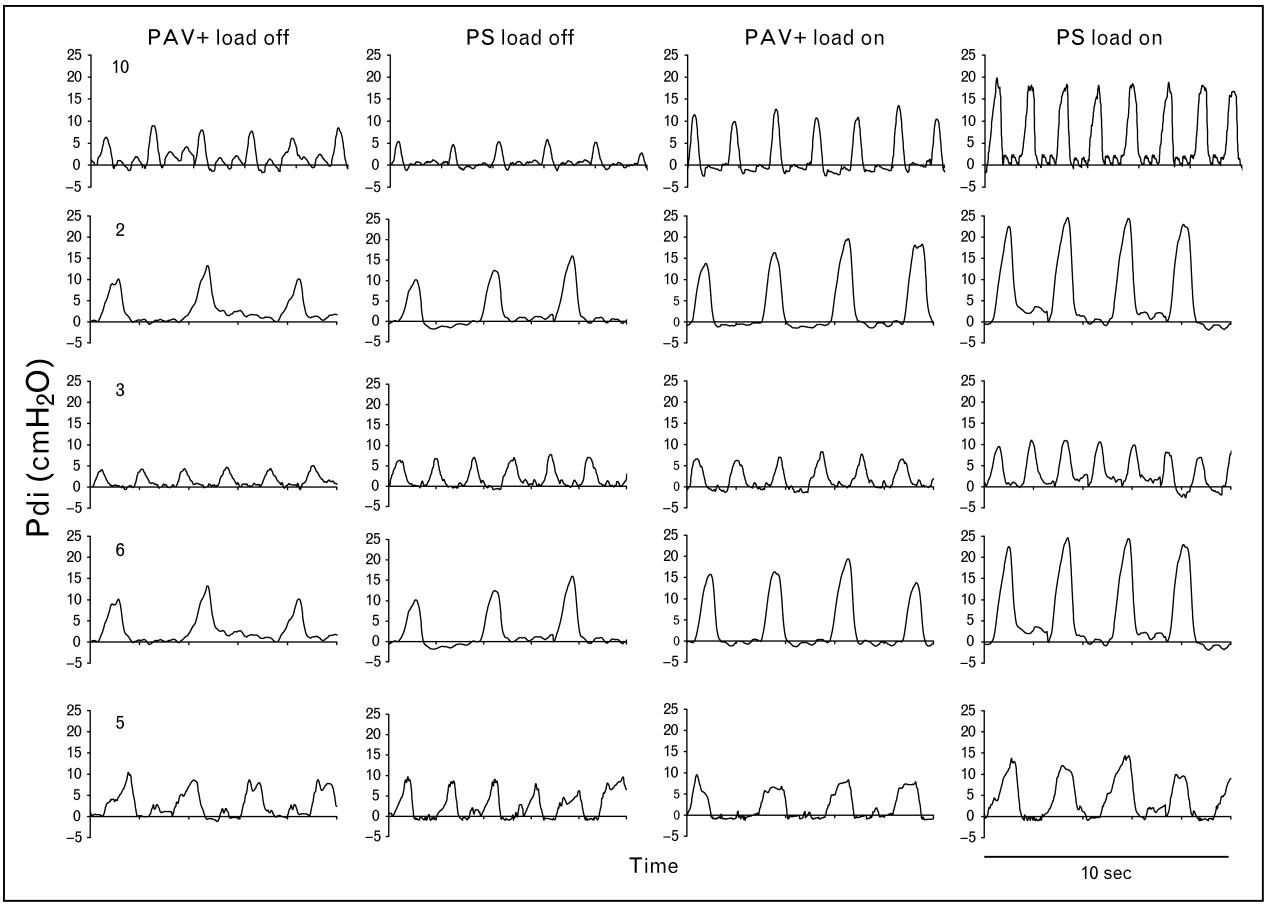
In the last two decades novel modes of assisted mechanical ventilation have been designed in which the pressure delivery is tightly linked to instantaneous flow and volume (proportional assist ventilation; PAV) [29,30], transdiaphragmatic pressure (P_{di}DV) [10] and the electrical activity of the diaphragm (NAVA) [11]. With these modes the ventilator represents an external inspiratory muscle, controlled, directly or indirectly, by the patient neural respiratory output. Although patient–ventilator asynchrony is considerably reduced with all three modes, only PAV is available in commercial ventilators.

With PAV the provided pressure is proportional to instantaneous flow and volume and hence, to pressure generated by the respiratory muscles [29,30]. The proportionality for flow and volume assist is preset and dictates the magnitude of the decrease in the impedance of the respiratory system [29,30]. Studies in normal subjects and mechanically ventilated patients have shown that PAV may efficiently unload the respiratory muscles and enhance the patient's comfort and the coupling between ventilatory demands and assistance [24,29–33]. The proper operation of PAV, however, requires regular and accurate measurements of respiratory system mechanics, otherwise there is risk for either under or over-assist (runaway phenomena). Given the difficulties in measuring respiratory mechanics in spontaneously breathing patients, the widespread use of PAV was significantly limited. To deal with that, software has been developed (PAV+) which automatically adjusts the flow and volume gain factors so as to always represent a constant fraction of the semi-continuously measured values of resistance and elastance of the respiratory system. Recently the efficiency of PAV+ was evaluated in a group of mechanically ventilated critically ill patients [34••]. The patients were randomly ventilated for 30 min with pressure support and PAV+ before and after an approximately 40% and 30% increase in elastic and resistive load of the respiratory system, respectively. As was expected with pressure support the level of pressure assist remained constant and independent of load. On the other hand with PAV+, load application resulted in an approximately 50% increase in airway pressure, as predicted by the alteration in the impedance of the respiratory system [34••]. Furthermore with PAV+ the application of load caused a considerably lower increase in inspiratory effort than that observed with pressure support (Fig. 2). With PAV+, however, the ventilator supported 86 and 66% of the inspiratory effort, without and with load application, respectively. This was due to a load-induced increase in PEEPi which decreased the proportion of supported inspiratory effort, since with this mode inspiratory flow and inspired volume are the signals that control the delivered pressure [34••]. Strategies that shorten the delay between the beginning of inspiratory effort and ventilator triggering (see above) greatly increase the efficiency of this mode to improve patient–ventilator synchrony.

Expiratory asynchrony exists when the end of mechanical inspiration precedes or follows the end of neural inspiration, termed respectively as premature or delayed opening of the expiratory valve [35•]. With premature opening of the expiratory valve, inspiratory muscle contraction continues into mechanical expiration phase [35•]. Zero or small inspiratory flow for some time after opening the exhalation valve indicates that inspiratory muscles continue to contract after the end of mechanical inspiration [3••]. In some cases there is a sharp decrease from the peak expiratory flow which lasts a few milliseconds followed by an increase and then decreases gradually to zero towards the end of expiration [3••]. With pressure support premature termination of pressure delivery is caused by low levels of pressure support, short time constant of the respiratory system, relatively high flow threshold for cycling off and dynamic hyperinflation [3••,35•]. AVC settings that result in a short inspiratory time place the patient at risk for this type of expiratory asynchrony [3••,35•].

With delayed opening of the expiratory valve the mechanical inspiration is continuous into neural expiration. Identification of delayed opening of the exhalation valve in relation to neural inspiration using the basic waveform of P_{aw}, volume and flow is difficult, particularly if the patient does not contract his or her expiratory muscles [3••]. Despite such difficulty with pressure support, a rather sharp decrease in inspiratory flow followed by an exponential decline toward the end of mechanical inspiration indicates that neural inspiration ends well before the exhalation valve opens [3••]. In some cases the end of neural inspiration causes, similar to the case with expiratory muscle contraction, a small increase in airway pressure [3••]. Thus during pressure support, P_{aw} may

Figure 2 Experimental records illustrating the effect of load application on transdiaphragmatic pressure (P_{di}) in five representative patients during PAV+ and during pressure support (PS)



Notice that without load (PAV+ load off and pressure support load off) P_{di} swings were comparable between modes. With load (PAV+ load on and pressure support load on) P_{di} swings were considerably larger with pressure support than with PAV+. Reproduced with kind permission of Springer Science and Business Media [34••].

increase either by abrupt relaxation of inspiratory muscles or contraction of expiratory muscles. In either case this P_{aw} increase is an indication of delayed opening of the expiratory valve [3••]. With AVC a linear increase in airway pressure is a sign of termination of neural inspiration before the end of mechanical inflation [3••]. The consequences of this form of expiratory asynchrony is more notable in patients with obstructive lung disease in whom delayed opening of the expiratory valve may increase dynamic hyperinflation and promote patient–ventilation dyssynchrony during the triggering phase [36]. Also it has been shown that delayed opening of the exhalation valve decreases the patient's spontaneous breathing frequency, possibly by activation of the Hering–Breuer reflex [16]. With AVC ventilator settings that result in long mechanical inflation time, such as high tidal volume, low inspiratory flow and application of end-inspiratory pause may cause delayed opening of the exhalation valve [3••]. With pressure support, this type of expiratory asynchrony is caused by the long time constant of the respiratory system (i.e. patients with obstructive lung disease), high pressure support level and low flow threshold for cycling off.

Until recently with pressure support the flow threshold for cycling off was nonadjustable. Since it has been recognized that with pressure support, similar to AVC, expiratory asynchrony is the rule, the modern ventilator has an adjustable flow threshold for cycling off. Taking into consideration the factors that lead to premature or delayed opening of the expiratory valve with pressure support, modulation of the flow threshold for cycling off may influence the degree and type of expiratory asynchrony. For example in patients with a short time constant (e.g. patients with acute respiratory distress syndrome) decreasing the flow threshold reduces the degree of expiratory asynchrony by reducing the premature opening of the exhalation valve. On the

other hand in patients with a long time constant (e.g. patients with obstructive lung disease) increasing the flow threshold diminishes the amount of expiratory asynchrony by promoting an earlier opening of the exhalation valve [37].

Recently the effect of changing the flow threshold for cycling off on patient–ventilator interaction was studied by Tassaux *et al.* [38•]. Ten patients with obstructive lung disease ventilated on pressure support were studied at flow thresholds of 10, 25, 50 and 70% of peak inspiratory flow [38•]. This study showed that increasing the expiratory thresholds from 10 to 70% decreased the delay in opening of the expiratory valve without the occurrence of premature opening. In addition a high flow threshold was associated with a decrease in dynamic hyperinflation and improvement in patient–ventilator synchrony during the triggering phase, as expressed by a reduction in triggering delay and the number of ineffective efforts [38•].

Advanced software aiming to eliminate expiratory asynchrony with pressure support has been introduced by Du *et al.* [39]. These investigators developed an algorithm which automatically adjusted the flow threshold for cycling off based on data of previous breaths. Currently, however, this feature of pressure support is under investigation [39].

Theoretically with PAV there should be synchrony between the end of neural and mechanical inspiration. Nevertheless, some asynchrony has been demonstrated with PAV, attributed to delay between the ventilator control system's input and output, airway leakage and overestimation of respiratory mechanics [40]. The new version of PAV+, which adjusts continuously the flow and volume assist, has largely overcome the latter problem [34••]. Using the electromyogram activity (NAVA) and transdiaphragmatic pressure (P_{di}DV) as a flow-control variable may eliminate the expiratory asynchrony, but currently these modes are not available in commercial ventilators [10,11].

Conclusion

Patient–ventilator dyssynchrony has been increasingly recognized in recent years. This dyssynchrony may exhibit several forms with different pathophysiologic background. The modern ventilators present features that assist the physician to recognize breath by breath the various types of patient–ventilator dyssynchronies, while on the other hand, have the capability to modulate the patient–ventilator interaction. Finally, new modes of ventilatory support have been introduced which advance the patient–ventilator synchrony by tightly linking the patient's respiratory effort to ventilator assistance.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 99).

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