Patient-Ventilator Asynchrony in a Sleeping Patient Receiving Pressure-Support Ventilation

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Introduction

It has been more than 20 years since Prakash and Meij,[1] and MacIntyre[2] described the physiologic responses of patients to pressure-support ventilation (PSV). At that time ventilators allowed for adjustment of only 2 PSV variables: breath trigger and inspiratory pressure. Modern versions of PSV allow for manipulation of the inspiratory rise time and breath-termination (cycle) criteria.[3] These additional adjustments allow the clinician to tailor PSV breath-delivery closer to the patient's neural control of ventilation. The manufacturer sets the limit on inspiratory time (T_I), to preclude protracted inspiratory flow from the ventilator.[3] This paper describes how manipulation of the inspiratory rise time and breath-termination criteria facilitated better patient-ventilator synchrony in a sleeping patient receiving PSV.

Case Summary

A 63-year-old male was receiving mechanical ventilation in the intensive care unit following emergency laparotomy to repair a ruptured viscus. The patient developed abdominal sepsis, which resulted in septic shock and multiple-organ dysfunction. Prolonged respiratory failure was multifactorial from sepsis, basilar atelectasis/infiltrates, and reduced chest-wall compliance due to obesity and anasarca. He eventually underwent tracheostomy and progressed to partial ventilatory support (Servo-i, Maquet Critical Care, Solna, Sweden). On ventilator day 37 the ventilator settings were: continuous positive airway pressure 5 cm H_2O, pressure support 22 cm H_2O, flow-cycle threshold 20% of peak inspiratory flow, rise time 0.2 s, fraction of inspired oxygen 0.4, and flow triggering. With this level of support his respiratory rate was generally 12–16 breaths/min and his tidal volume (V_T) was 500–600 mL. During a routine patient-ventilator check he appeared to be in a deep sleep. He was receiving fentanyl via transdermal patch and had received 1 mg of intravenous hydromorphone 2 hours prior to assessment. The patient-ventilator check revealed an important change in his ventilatory pattern. V_T had risen to approximately 1,000 mL, the recorded respiratory rate had fallen to $<$ 10 breaths/min, and physical examination revealed weak inspiratory efforts that failed to trigger the ventilator.

The ventilator graphics during this occurrence (Fig. 1) show failed attempts to trigger PSV breaths, due to intrinsic positive end-expiratory pressure ($PEEP_I$) and breath-termination at an inspiratory flow of 33% of the peak flow, but the flow-cycle threshold was set at 20% of the peak flow. In Figure 1 the PSV breath terminates before the flow-cycle threshold is met, because the breath is time-cycled. On the Maquet Servo-i ventilator the maximum T_I during a PSV breath is 2.5 s. Unfortunately, the Servo-i graphics display does not show the time units on the horizontal axis, which makes time-cycling more difficult to appreciate. Identifying time-cycling with the Servo-i is a diagnosis of exclusion: T_I appears long, there are no indications of pressure-cycling, and the end-inspiratory flow appears discordant with the flow-cycle threshold.

In response to the graphics in Figure 1 and to eliminate the excessive T_I and V_T, the flow-cycle threshold was increased to 40% of peak inspiratory flow, which is the maximum setting on the Servo-i. That change only shortened T_I from 2.5 s to 2.25 s, and reduced the exhaled V_T by only about 100 mL (Fig. 2). The rise time was then shortened from 0.2 s to 0.15 s, which reduced T_I from 2.25 s to 1.2 s, which reduced V_T to about 570 mL and eliminated failed triggering attempts (Fig. 3).

Discussion

Additional clinician control of PSV breath variables allows the clinician to correct sources of patient-ventilator asynchrony. However, these changes in breath control make

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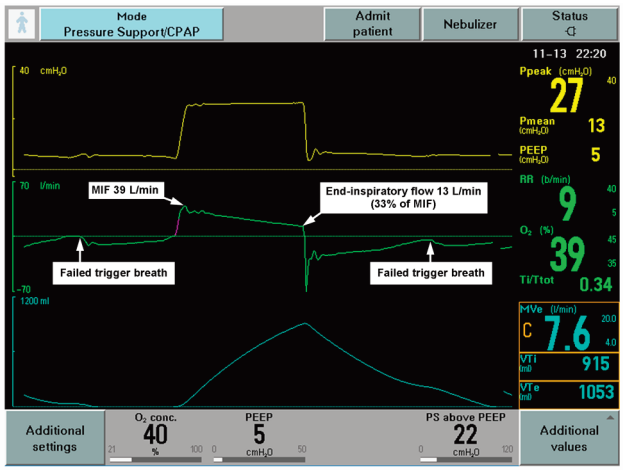

Fig. 1. White labels added to the ventilator pressure/time, flow/time, and volume/time curves to indicate the maximum inspiratory flow (MIF), end-inspiratory flow, and failed trigger breaths in a patient who developed increased tidal volume and reduced respiratory rate during sleep. Without the benefit of physical examination the failed breath triggers could have also been interpreted as biphasic exhalation. Ventilator settings: continuous positive airway pressure 5 cm H_2O, pressure-support ventilation 22 cm H_2O, flow-cycle threshold 20% of peak inspiratory flow, rise time 0.2 s, flow triggering. CPAP = continuous positive airway pressure. P_{peak} = peak pressure. P_{mean} = mean pressure. PEEP = positive end-expiratory pressure. RR = respiratory rate. T_I/T_{TOT} = ratio of inspiratory time to total-breathing-cycle time. MV_E = expiratory minute volume. V_{Ti} = inspiratory tidal volume. V_{Te} = expiratory tidal volume. PS = pressure support.

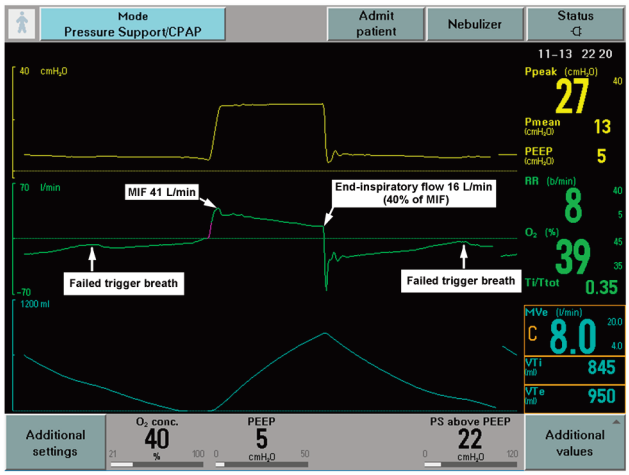

Fig. 2. Ventilator pressure/time, flow/time, and volume/time curves after the flow-cycle threshold was increased to 40% of peak inspiratory flow. Without the benefit of physical examination the failed breath triggers identified above could also be interpreted as biphasic exhalation. (Acronyms and terms are as in Figure 1.)

modern-day PSV more difficult to manage correctly. Clinicians must have a more sophisticated understanding of the dynamics of PSV so that the technology benefits the patient.

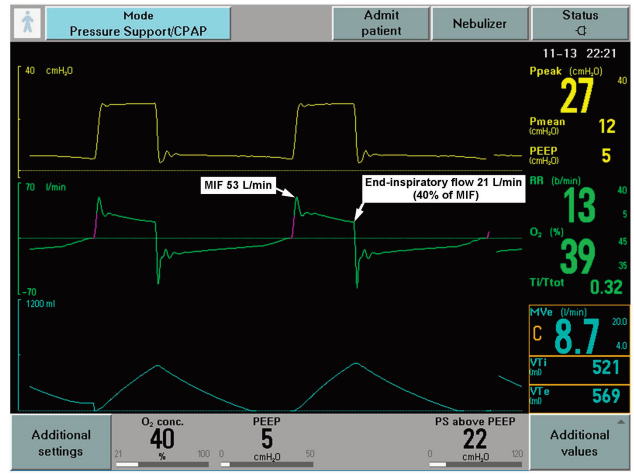

Fig. 3. White labels added to the ventilator pressure/time, flow/time, and volume/time curves to indicate the maximum inspiratory flow (MIF) and end-inspiratory flow after the rise time was reduced from 0.2 s to 0.15 s. (Acronyms and terms are as in Figure 1.)

Flow-cycling of a PSV breath occurs when the inspiratory flow declines to the flow-cycle threshold, which is usually defined as a percentage of the peak inspiratory flow. The flow-cycle setting should be assessed frequently and must be adjusted whenever there is evidence of premature or lagging flow-termination. When the flow-cycle threshold is set too low, pressure-cycling or time-cycling may occur. Pressure-cycling of PSV breaths increases work of breathing (WOB), because in order to terminate the breath, the patient has to exhale against inspiratory flow from the ventilator until the airway pressure meets a threshold above the sum of the end-expiratory pressure and the pressure-support setting (Fig. 4).[4,5] In a spontaneously breathing subject, delaying expiration results in a compensatory lengthening of subsequent expiratory phases, but that compensation is ineffective during mechanical ventilation and results in dynamic hyperinflation.[6] On some ventilators, an airway pressure more than a few cm H_2O above the sum of PSV and PEEP will produce pressure-cycling, whereas other ventilators require airway pressure to reach the upper pressure limit.[3] Pressure-cycling can be identified by a spike in the airway pressure at the end of a PSV breath (see Fig. 4).[5] Pressure-cycling of PSV breaths is a common occurrence in patients with chronic obstructive pulmonary disease (COPD), who have long time constants and increased ventilatory drive.[7,8] In addition, pressure-cycling of PSV breaths is frequently encountered with higher pressure-support levels.[7]

When pressure-cycling is identified, the clinician should increase the flow-cycle threshold and in some cases reduce the pressure-support setting to try to eliminate that source of patient-ventilator asynchrony. In contrast, patients with short time constants, such as those with acute lung injury, can experience increased WOB and patient-ventilator asyn-

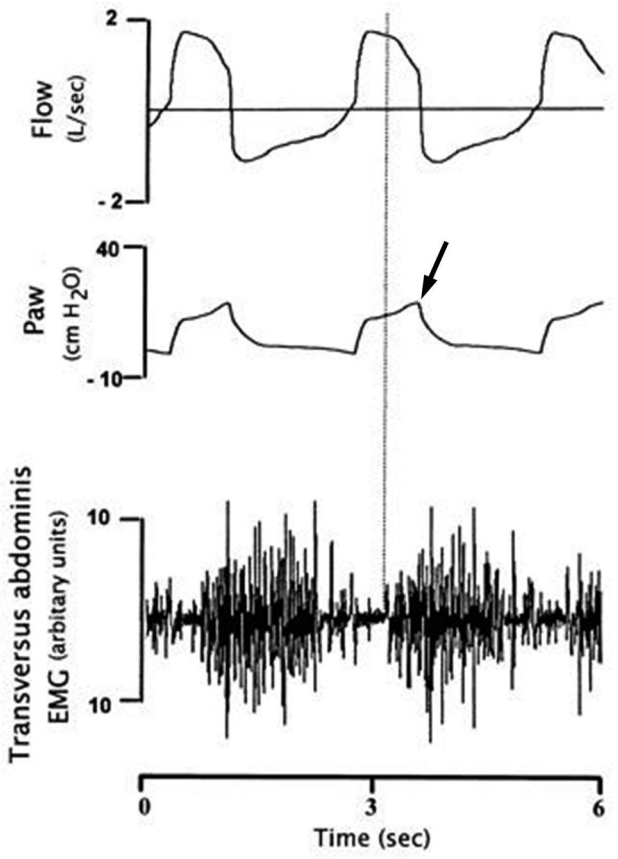

Fig. 4. Ventilator flow/time and pressure/time curves, and transversus abdominis electromyogram (EMG) depicting pressure-cycling asynchrony of a breath delivered in pressure-support ventilation. Active expiratory muscle activity coincides with continued inspiratory flow from the ventilator (vertical line). The pressure spike at the end of the pressure/time curve (arrow) indicates pressure-cycling of a pressure-support breath. P_{aw} = airway pressure. (From Reference 5, with permission).

chrony due to premature breath-termination if the flow-cycle threshold is adjusted higher.[9,10] Double-triggering (ie, one neural inspiration triggers the ventilator twice) is commonly encountered when the cycle threshold is set too high.

Time-cycling of PSV is usually associated with an air leak in the ventilator circuit, around the cuff of an artificial airway, through a bronchopleural fistula, or around a mask, when a critical-care ventilator is used to deliver noninvasive ventilation. A leak during PSV results in sustained high flow from the ventilator, which can overwhelm the patient's ability to cycle the ventilator and thus cause patient-ventilator asynchrony and $PEEP_I$.[11,12] Most critical care ventilators now limit T_I to $\leq$ 4 s, but this was not always the case.[3] For example, before the Puritan Bennett 7200 ventilator was programmed with a PSV time-cycle limit of 3 s, patients would have to fight against flow from the ventilator for a prolonged period until they could pressure-cycle the breath. In addition to leaks, if the patient does not have enough expiratory muscle activity or strength to facilitate pressure-cycling, PSV breaths may also be time-cycled.

The rise-time setting adjusts the time it takes the ventilator to achieve the desired pressure setting during PSV. In other words, it adjusts the initial inspiratory flow, which is almost always the peak flow during the inspiratory cycle. Adjusting the rise time to the correct setting is very important indeed, because an inappropriate rise time can increase WOB and decrease patient comfort. In a study of mechanically ventilated patients with COPD, Bonmarchand et al[13] found that a low initial flow (long rise time) during PSV increased neural ventilatory drive and WOB. An important point for the clinician at the bedside to consider is that these changes in respiratory function were detected by changes in the diaphragmatic electromyogram and airway-occlusion pressure and not by a change in respiratory rate, V_T, or $PEEP_I$. In a study of patients recovering from acute lung injury, Chiumello et al[9] also found that the shortest rise time reduced WOB without changing respiratory rate or V_T.

The latter 2 studies suggest that respiratory rate and V_T are not perfectly sensitive "workometers." However, in a previous study those same investigators found that in patients with acute lung injury the slowest rise time increased respiratory rate and lowered V_T,[14] and patient comfort was affected by the lowest and highest initial flows. Though mechanical ventilation is an arena dominated by numbers, we must be mindful that simply asking the patient how he or she feels can be extraordinarily helpful in getting the ventilator settings right. Painstakingly adjusting variables to achieve nice graphic waveforms may be quite meaningless if the patient's dyspnea is not relieved.

In this case, a low peak inspiratory flow from sluggish ventilatory drive caused by sleep, narcotics, and the set rise time, coupled with the pressure-support setting produced ventilations that were time-cycled before the flow-cycle threshold could be met. Because increasing the flow-cycle threshold to 40% didn't solve the problem, the rise time was shortened. Reducing the rise time increased the initial inspiratory flow and thus reached the pressure-support setting quicker. The peak inspiratory flow was higher, so the flow-cycle threshold, though still set at 40%, was reached at a higher inspiratory flow. In this case shortening the rise time from 0.2 s to 0.15 s increased the peak inspiratory flow from 41 L/min to 53 L/min, so the flow at the flow-cycle threshold (40% of peak inspiratory flow) increased from 16 L/min to 21 L/min. With a faster rise time there was a much more rapid decline in inspiratory flow from the peak, so T_I dropped from 2.25 s to 1.2 s, which decreased V_T to about 570 mL and eliminated failed triggering attempts that had been caused by $PEEP_I$ (see Fig. 3).

Typically one would only shorten the rise time to reduce the WOB in a patient with an increased drive to breathe, such as in an exacerbation of COPD[11] or acute lung injury.[9,14] But in this case, with the maximum flow-cycle threshold of 40% already applied, shortening the rise time facilitated better patient-ventilator synchrony in a sleeping patient who had a sluggish ventilatory drive.

Certainly, another approach to this problem is to reduce the pressure-support setting; however, changing the rise time is an immediate option for the respiratory therapist if a physician order to titrate the pressure-support level is needed. Perhaps the best option to correct this type of patient-ventilator asynchrony is to change modes completely. PSV during sleep is associated with central apneas due to hypocapnia and patient-ventilator asynchrony, which decrease sleep quality.[15-16] Both volume controlled-continuous mandatory ventilation[15] and proportional-assist ventilation[16] reduce patient-ventilator asynchrony and improve sleep quality, compared to PSV.

Ventilator manufacturers have given the clinician more control over PSV delivery, to overcome the shortcomings of antecedent programming. Consequently, clinicians can better tailor PSV to be in concert with the patient's neural control of ventilation and reduce patient-ventilator asynchrony and imposed WOB.

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