Identifying asynchronies: Expiratory work
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DOI: https://doi.org/10.53097/JMV.10086

Abstract
Mechanical ventilation is used to improve gas exchange and unload the respiratory muscles allowing for their rest and recovery, which require good synchronization between the patient and the ventilator.

Spontaneous respiratory effort is generally preferred because it reduces atelectasis, improves oxygenation, and may prevent disuse diaphragm atrophy. Nevertheless, vigorous spontaneous effort can cause both lung injury and diaphragm injury (myotrauma). These injuries lead to prolonged ventilation, difficult weaning, and increased morbidity and mortality.

Normal expiration is passive due to the recoil of the lungs and chest wall. In mechanical ventilation, during expiration the ventilator controls the pressure (ie, the target value is PEEP), therefore, we must look at the flow and volume waveforms to see the physiology and patient-ventilator interactions. In expiration the patient-ventilation interaction is not characterized by timing but by work. Expiratory effort (ie, negative Pmus) will deform the flow waveform in a negative direction (away from baseline).

Keywords: synchronization, spontaneous effort, lung injury, myotrauma, expiratory effort

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Conflict of interest/Disclosures: None
Funding: None
Acknowledgement: To Ehab for his support in interpreting this graphic.
Patient–ventilator asynchrony is common in mechanically ventilated patients. It can be defined as a mismatch between patient respiratory effort and ventilator support.  

Although severe patient-ventilator asynchrony is frequent during invasive and non-invasive mechanical ventilation, diagnosing such asynchronies usually requires the presence at the bedside of an experienced clinician to assess the tracings displayed on the ventilator screen applying their knowledge of pulmonary physiology to interpret patients' physical signs together with flow and airway pressure waveforms.  

Prolonged weaning from mechanical ventilation develops in 6 to 15% of mechanically ventilated patients and is associated with increased morbidity and mortality. A major determinant of weaning failure is respiratory muscle dysfunction.  

To avoid ventilation-induced diaphragm dysfunction, clinicians use partially assisted/supported ventilation instead of fully controlled ventilation in critically ill patients. This strategy aims at avoiding diaphragmatic atrophy by maintaining a contractile activity of the respiratory muscles but also requires harmonious synchronization and matching with the patient's demands in terms of ventilator needs.  

Pressure support ventilation (PSV) is a pressure-limited, flow-cycled ventilatory mode whose objective is to unload respiratory muscles and to assist every breathing effort by delivering a predetermined positive pressure level. Expiration begins (cycling off) when inspiratory flow reaches a certain threshold.  

The efficacy of PSV depends on the interaction between the ventilator setting and patient's control of breathing. If the mechanical inspiration ends before the neural inspiration, the patient's inspiratory effort continues beyond the cessation of the ventilator assistance. In this case, the pressure developed by the inspiratory muscles (Pmus) must counteract the elastic recoil of the respiratory system. Although inspiratory muscles are still activated, the respiratory system volume tends to decrease, with a consequent decrease in the elastic recoil. The Pmus could thus be greater than the elastic recoil.  

Expiration is usually passive in patients mechanically ventilated, due to the recoil of the lungs and chest wall and it is always pressure controlled (ie, the target value is PEEP). The expiratory muscles are the “neglected component” of the respiratory muscle pump. During tidal breathing, the expiratory muscles are largely inactive. Their activation during breathing occurs when the (relative) load imposed on the inspiratory muscles increases.  

In a passive patient, we expect to see a smooth exponential decay of the expiratory flow and volume waveforms. Patient expiratory effort (ie, negative Pmus) will deform the flow waveform in a negative direction (away from baseline). Expiratory work may be normal, as when exercising or coughing, but it may also indicate the presence of high resistive load (i.e. COPD), acidosis, or anxiety. Expiratory asynchrony is a common phenomenon during all modes of assisted mechanical ventilation.  

The expiratory muscles, which include the abdominal wall muscles and some of the rib cage muscles, are an important component of the respiratory muscle pump and, in patients receiving mechanical ventilation, are recruited in the presence of high respiratory load and/or low inspiratory muscle capacity or with pulmonary hyperinflation.  

Expiration muscles recruitment progressively increases during a spontaneous breathing trial (SBT) in weaning failure patients, while remaining stable in successfully extubated patients. Recruitment of the expiratory muscles may have beneficial effects, including reduction in end-expiratory lung volume, reduction in transpulmonary pressure and increased inspiratory muscle capacity.  

Activation of the abdominal wall muscles increases abdominal pressure in the expiratory phase. Increased abdominal pressure enhances inspiratory muscle capacity via two mechanisms. First, increased abdominal pressure moves the diaphragm at end expiration to a more cranial position, which results in a more optimal length for tension generation. Second, when the end-expiratory lung volume falls below functional residual capacity (FRC), elastic energy is stored in the respiratory system. This stored energy facilitates the next inspiration (ie, allows more rapid and greater development of negative pleural pressure).  

Figure 1 presents the ventilator waveforms of an 82 year old woman with post traumatic brain injury in liberation phase of prolonged mechanical ventilation with tracheostomy and critical patient weakness.
Figure 1. From top to bottom: pressure-time, flow-time and volume-time curves. We can see a variable inspiratory time characteristic of pressure support ventilation (red rectangle). In expiration time there is expiratory work in which some things happen: a possible early cycling: amputation of the expiratory peak flow (purple arrow), associated to a strong expiratory effort that reverses the expiratory flow from his normal pattern (high at the beginning and less to the end), this effort increases during expiration evidenced by a progressive increase in expiratory flow (blue arrow) and at the end of expiration (orange arrow), there is a strong inspiratory effort that produces a high inspiratory peak flow. This strong expiratory effort causes pressure to rise above set level (green arrow).

References


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