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Use of the esophageal catheter to detect asynchronies

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Abstract

Patient-ventilator asynchronies are more usual than we believe in critical care patients, and recognition of such an interaction can be challenging in clinical practice, especially if we don´t have advanced monitoring tools such as esophageal pressure tracing at the bedside.

Reverse trigger, early cycling, and work shifting are types of asynchronies that must comply with some characteristics in the interaction between ventilator and patient, that could be difficult to detect only with visual analysis of usual waveforms curves showed in mechanical ventilators even for trained operators.

In this sense, esophageal catheter is a very useful tool to correct detection and management, if required, of patient-ventilator interactions.

Keywords: asynchrony, esophageal pressure, reverse trigger, work shifting.

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Patient-ventilator asynchrony (PVA) is a condition when ventilation is not adequate, and there are discrepancies between the phases of the respiratory cycle and patient breathing effort (1). They are common in intensive care units with an incidence of up to 25% (2).

We call early trigger when a machine-triggered inspiration precedes the patient trigger effort. The patient effort may occur any time during inspiration or early during expiration (3). Reverse trigger (RT) corresponds to a specific type of interaction between the patient and the mechanical ventilator, where the diaphragm contracts in a delayed manner or at a certain time after passive insufflation started by the ventilator (4) in a relation referred to as “entrainment” (5). “Respiratory entrainment” (also called “respiratory phase locking”) refers to the establishment of a fixed repetitive temporal relationship between the neural and mechanical respiratory cycles (6). On the other hand, the term early trigger describes the event in terms of the signals, not the physiology (3).

It has been seen that RT mainly occurs in patients under deep sedation or who are in transition from controlled to spontaneous ventilation (7). The pathogenesis involves the activation of vagally mediated pulmonary reflexes, along with cortical and subcortical influences (8).

Recent studies have shown that RT may exhibit different phenotypes (9). The most frequent one is characterized by activation that produces the greatest contraction peak during the expiratory phase (7). Called mid-cycle RT (see figure 1), it occurred when inspiratory efforts began during inflation, but maximal inspiratory muscle pressure (Pmus) generation occurred during lung deflation, the patient then relaxes back to the passive chest wall compliance curve by end-expiration before the subsequent passive ventilator-delivered breath (9).

For RT to be considered as such, the cycle must be mandatory in the absence of signs of patient effort (7). In pressure control mode, we can identify some features (see figure 1) such as variations in the flow-time curve; for example, a rise in flow during the inspiratory phase time interval in the decelerated flow, indicating contraction of the diaphragm, which at the same time may or may not correspond to a mild drop in the pressure-time curve (7), which can generate some degree of work-shifting (orange arrows in figure 1). In the flow-time curve we can also observe an amputation of the peak expiratory flow and a rise in flow in the first third of the cycling phase (7), giving an image similar to that of an early cycling (red line in figure 1), however the primary phenomenon is the RT. If contraction of the diaphragm is powerful enough and close to the end of the inspiratory cycle, it may give rise to a new trigger: double cycling (7).

For monitoring purposes, clinicians rely mostly on airway pressure and flow waveforms (10) (7). They provide a gross estimate of patient–ventilator synchrony, but a considerable number of asynchronies remain undetected (11). These measurements may mask profound PVA and do not allow respiratory muscle effort assessment (10). There is a need for additional signals reflecting patients’ inspiratory effort to facilitate recognition of these events (11) .

The reference standard for the identification of RT is the use of the esophageal balloon (Peso), or the electrical activity of the diaphragm (EAdi) (7). The esophageal catheter provides a direct observation of the patient’s inspiratory effort (12). Monitoring Peso in this context enables detection of every inspiratory effort, and hence understands the interaction and synchrony between patient and ventilator (10). Use of Peso has allowed the description of RT (5); without Peso, it would have been impossible to detect such asynchronies (12). Using Peso, we detect RT through the positive change in the Peso signal at the start of mandatory respiration, followed by a negative swing in the Peso signal after the start of patient´s respiration (7) (see figure 1).

Peso measurements have enhanced our understanding of the pathophysiology of patient–ventilator interaction (10).

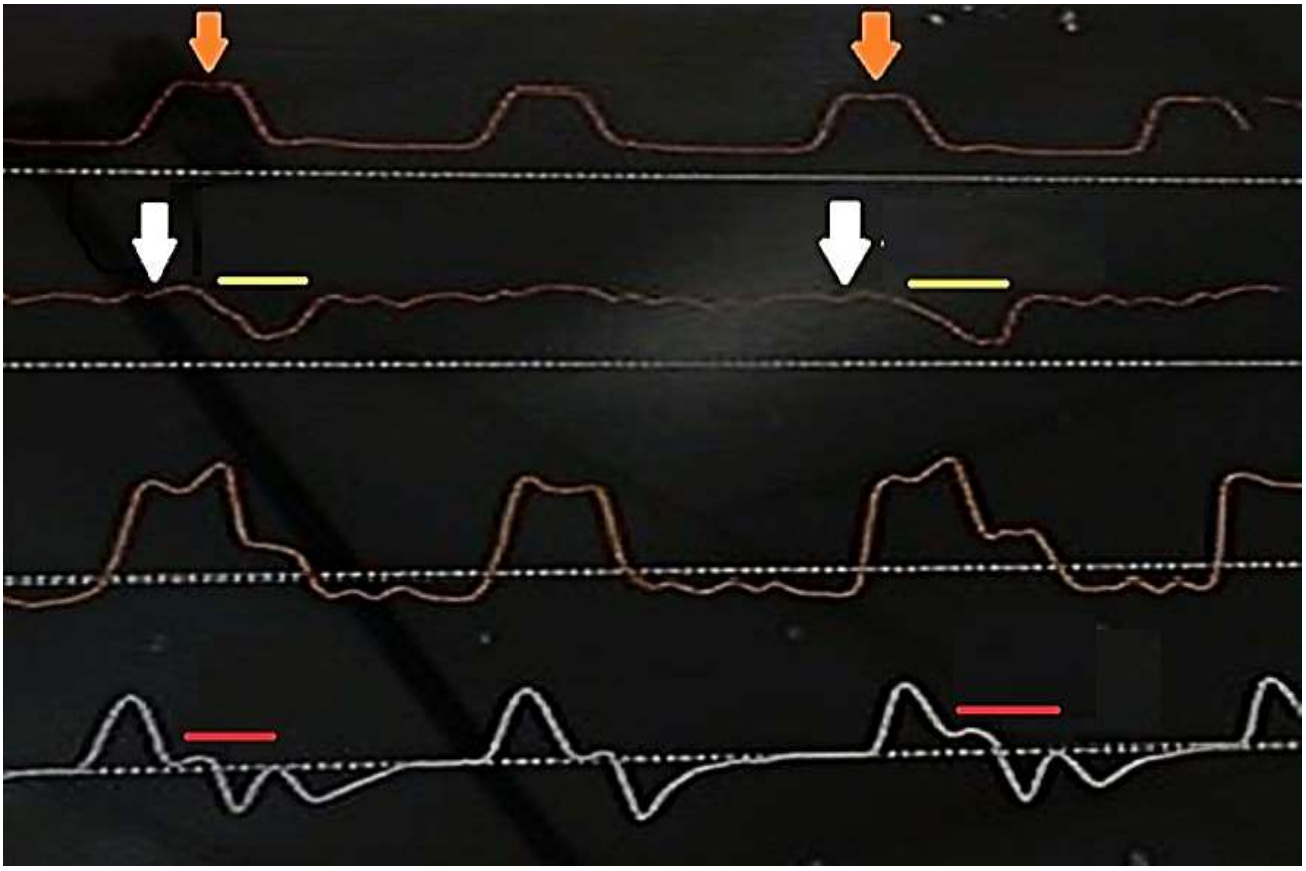

Figure 1. From top to bottom: airway pressure-time, esophageal pressure-time, transpulmonary pressure-time and flow-time curves. There is a reverse trigger in the first and third breaths. We can see the beginning of mandatory ventilations (white arrows), start and end of patient activity (yellow and red lines). Orange arrows indicate the electrical activity of the diaphragm onset.

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