A question of flow… how to solve a flow asyncrony on AC-VCV

Manuel is a patient ventilating in volume controlled ventilation (VCV) or better in assist-controlled VCV (AC - VCV):

• VT = 400 ml

• RR = 20 breaths per minute

• I: E = 1:1 (=Ti/Ttot 50%)

These settings produce a respiratory cycle of 3 seconds (60/20 = 3 sec); inspiratory time = 1.5 sec and expiratory time = 1.5 sec.

Suppose at this point we reduce the respiratory rate to 12 bpm.

Taking the set respiratory rate at 12 bpm and leaving the ratio I: E = 1:1 unchanged, this will result in a change in the duration of the respiratory cycle, which will pass from 3 to 5 seconds, resulting in an increase of Ti to 2.5 seconds and of Tesp that will become 2.5 seconds! (Figure 1)

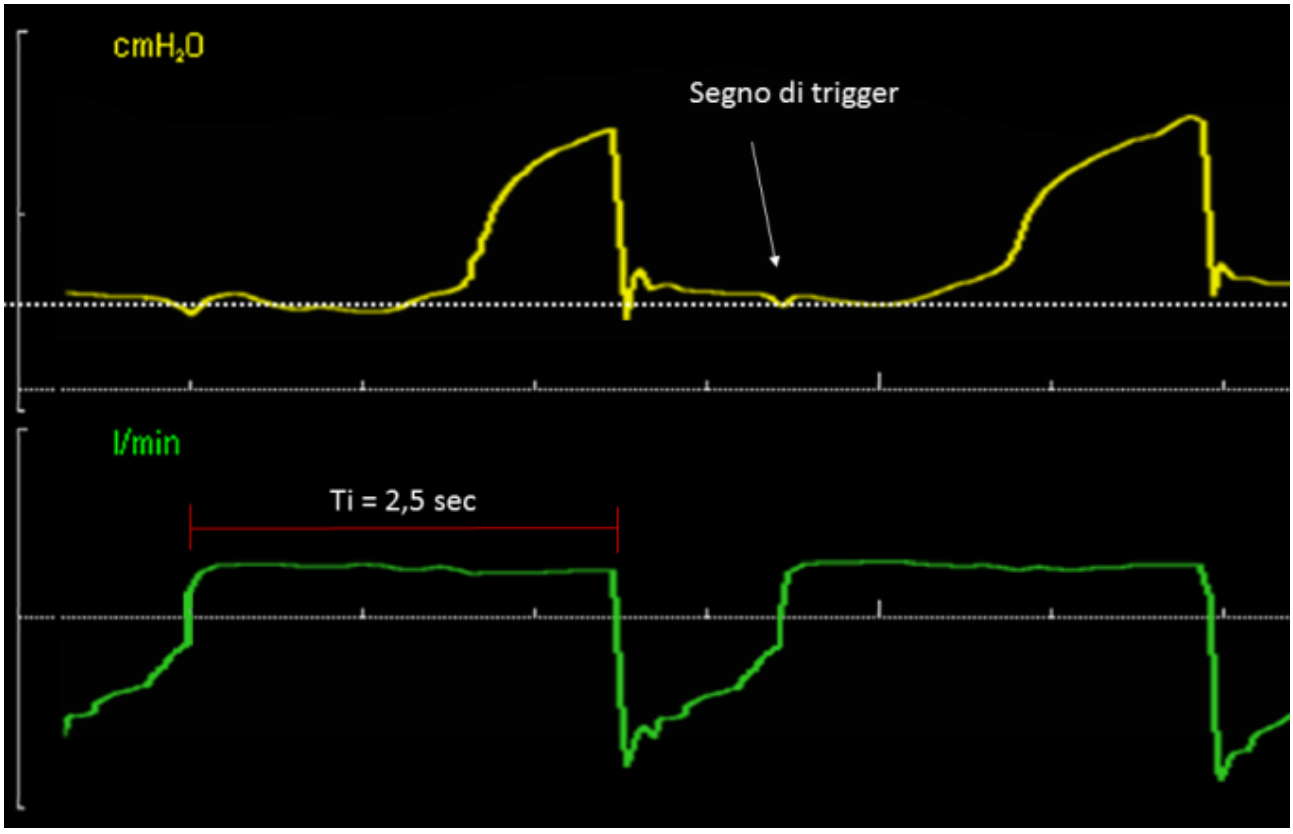

Figura 1. Ventilator waveforms during VCV

Let's use our analysis method.

1. Look at the pressure curves (Paw in yellow) and flow (in green).
2. Identify the inspiratory and expiratory phases. On the flow curve is **inspiratory phase** when the line is above zero and expiratory when it is below.
3. Verify the activation of the inspiratory muscles / trigger activation.Clearly, there is the **trigger sign** on the pressure curve, characterised by a small incision.
4. **Evaluate how the pressure curve changes during inspiration**.Observing the monitoring in Figure 1 we find the first anomaly. During the inspiratory phase the Paw does not increase rapidly: it stays for a second and a half at PEEP level, and THEN undergoes an increase. The missed increase increase in PAW during inspiration is a classic **FLOW asyncrony** sign. The cause could be intense activation of the inspiratory muscles during inspiration or an incorrect setting the ventilator that supplies a flow too low to meet the patient's demands.

What exactly is happening?

The inspiratory flow during VCV is constant, and represents the independent variable; hence it does not undergo any variations based on the patient's demands and depends solely on how the ventilation is set (inspiratory time and current volume). More interesting is the dependent variable, the Paw, which provides valuable information about the respiratory system (chest wall + lungs!) and the degree of patient-ventilator interaction (PVI).

There is an imbalance in the A/C-VCV flow asynchrony: the flow generated by the ventilator is insufficient to meet the patient's demands.

The activation of the inspiratory muscles does not lead to an increase in the flow (since it is the independent variable) but to a visible reduction in PAW!!

Why is that?

We can explain what happens with the beloved respiratory **equation of motion**:

PAW = P0 + *Pel* + Pres + (-Pmusc) =

= Total PEEP + *current volume x elastance* + flow x resistance + (- pressure of respiratory muscles)

If the activity of the muscle (Pmusc) generates an inspiratory flow greater than that delivered by the ventilator, there is a **reduction in the PAW**.

1. **Assess the possible activation of the expiratory muscles**. We look at the end of the inspiratory phase and the beginning of expiration: there is no early activation of the expiratory muscles.

2. **Observe the expiratory phase**. Passive expiration is characterized by an initial flow peak and a downward slope with concavity downwards. In the image there is an active expiration sign because no concavity of the flow trace is noticeable. Another thing to note is the early END of exhalation, manifested by the non-return of the expiratory flow to the zero line: if at the end of the expiration the current volume has not been exhaled completely, dynamic hyperinflation is generated. Under dynamic hyperinflation conditions, at the end of expiration in the alveoli remains a positive residual pressure, called intrinsic positive end-expiratory pressure

What importance to attribute to PEEPi?

Intrinsic PEEP (as well as external PEEP) may reduce heart rate or put the lungs at risk of overinflation at the end of inspiration. It could also reduce inspired airflow speed and increase WORK OF BREATHING during assisted ventilation and spontaneous breathing.

Manuel's ventilation teaches us that a mere change in setting can have negative effects!

When using cycling CONTROLLED systems (Ti/Ttot or I:E) it is important to understand that varying respiratory rates are subject to a variation in inspiratory time. In the case of Manuel, a reduction in RR resulted in an increase in Ti, which leads to a reduction in the delivery rate of the VT set. *If 400 ml is to be delivered in 1.5 sec the flow will be 267 ml / sec, but if it is to be delivered in 2.5 seconds the flow will be 160 ml / sec*.

The Ti remains constant according to the set respiratory cycle time (RR set and I: E), so in order to increase respiratory rate at 18 rpm, Manuel is forced to sacrifice much of the expiratory time!

We stress that this situation can generate:

● **significant increase in the work of breathing**.

● **Dynamic hyperinflation**. Patients after a long inspiratory time often do not spend much time expiring because they need a new inspiration. But ending expiration too early can generate PEEPi.

PRACTICAL ADVICES:

- On some ventilators you directly set the inspiratory time in seconds and not the ratio I: E or Ti/Ttot. Doesn't matter which, just take a look at the ventilation setting to figure out how it was programmed and know how to behave accordingly. For ventilators using the I:E ratio you must pay attention to the variations in the respiratory rate set, as THIS changes the duration of the respiratory cycle and consequently cycling times.
- Always check that the inspiratory time set when the patient was passive is adequte even when he regains respiratory drive.
- Use the I: E ratio only to obtain an appropriate inspiratory time without paying too much attention to the set expiratory time, as the actual ratio changes when the respiratory rate of the patient changes.

How to determine the optimal duration of inspiratory time?

We know that physiological inspiratory time is in the order of 0.8–1.2 seconds. This can guide us in choosing inspiratory time, but it is by no means sufficient, we must necessarily evaluate ventilatory waveforms to understand PVI.

Characteristics of flow asyncronies in ACV

● It is generated during inspiration,

● During inspiration, the flow is delivered at a constant speed, but the pressures do not increase or increase only later, after which part of the volume is already reached to the patient.

POSSIBLE SOLUTIONS to MANUEL'S CASE:

A first simple hint is to understand the reason why this alteration is generated, in fact, we could have 2 causes: an incorrect ventilation setting that generates a too low flow OR excessive demands from the patient. In either case, monitoring is the same (**positive flow but pressures that do not increase**).

In the case of Manuel, the simple reduction in inspiratory time to about one second resulted in a sufficient flow increase to solve the problem (Figure 2).

What happened to Manuel's ventilator?

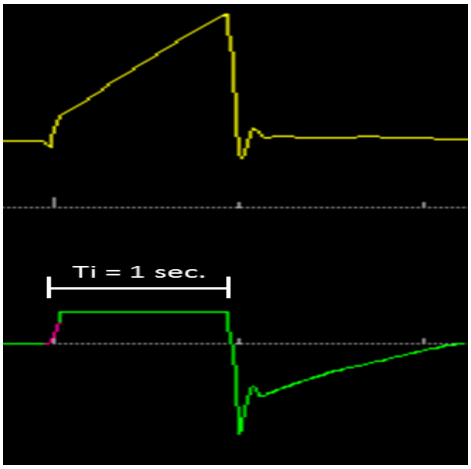

Figura 2. Reduction in inspiratory time to about one second solved the problem.

The ventilator, with this new setting, has less time to deliver the same volume and consequently delivers the air more quickly. The flow generated by the ventilator has a higher speed than that required by the patient, thus immediately increasing the pressure throughout the inspiration and restoring the correct synchrony between the patient and the ventilator. The expiratory phase, moreover, is no longer truncated and the flow curve reaches the zero line. Ultimately, there is no inspiratory flow asynchrony and the exhalation is complete!

Thanks to all dear readers of Triggerlab, Cristian Fusi

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