

ASV in a COPD Patient

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Case Report

Mrs. H. A., a 74-year-old woman with a history of chronic obstructive pulmonary disease (COPD), formerly a heavy-smoker, and previously treated at home with corticosteroids and long-term oxygen therapy, was admitted to an intensive care unit with acute respiratory distress. She had a three-day history of worsening dyspnea, and increased purulence and volume of tracheal secretions.

Chest radiography showed hyperinflation without acute infiltrate. Measurement of arterial blood gases while the patient was breathing room air showed severe acute respiratory acidosis ($\text{pH} = 7.18$; $\text{PCO}_2 = 13 \text{ kPa}$). The cause of the acute respiratory failure was assumed to be a respiratory tract infection. Non-invasive ventilation (NIV) was administered, but neither clinical status, nor arterial blood gas status, improved.

The patient was sedated and orally intubated. The ventilator was set to ASV. Based on her height, ideal body weight was set to 49 kg. Percentage minute volume was set to 110%. The resulting minute ventilation was 5 l/min. FiO_2 was set to 50%, and PEEP was set to zero. The measured expiratory time constant (RCe) was 3.4 seconds. The tidal volume-frequency (Vt-FR) combination given by the ventilator was 776 ml and 7 breaths per minute, with an I:E ratio of approximately 1:2 as shown on Figure 1.

With this Vt-FR combination, inspiratory pressure (Pinsp) was 23 cmH_2O , plateau pressure (Pplat) was 17 cmH_2O , and static intrinsic PEEP (PEEPi, stat) was 6.9 cmH_2O (Figure 2). Respiratory acidosis normalized with persistent chronic hypercapnia.

The patient was sedated for two days. At the end of this time, oedema had reduced and the volume of tracheal secretions had decreased. Sedation was stopped, and the patient started to breathe actively. To compensate for the extra work of breathing due to intrinsic PEEP, 6 cmH_2O external PEEP was set. The RCe was then 1.58 s. The Vt-FR combination was 557 ml at 13 breaths per minute. Pinsp was 20 cmH_2O (Figure 3).

On day 5, RCe decreased to 1.39 s and Pinsp was 13 cmH_2O . After a two-hour successful T-piece trial, the patient was extubated. Because of her unstable respiratory condition, she was trained for NIV before being transferred to the respiratory ward.

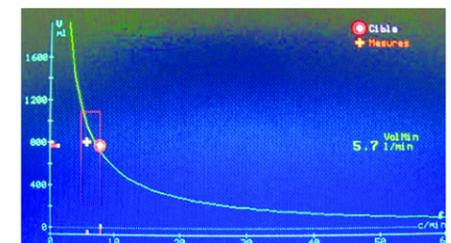
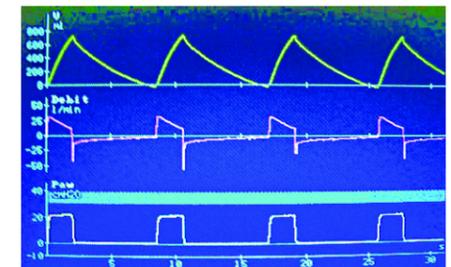


Figure 1: Above: Volume, flow and pressure traces during ASV ventilation. Below: The special ASV monitoring screen showing (on the tidal volume-respiratory frequency curve) the optimal Vt-FR combination calculated by the ASV algorithm (red circle). The patient's current tidal volume and respiratory frequency (yellow cross) is shown near the curve.

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Discussion

Adaptive Support Ventilation (ASV) is a comprehensive “package” including the following features:

1. Automatic detection of spontaneous activity (the ventilator is able to switch back and forth from full to partial ventilatory support).
2. Wide-ranging, automated, pressure modes (from pressure-controlled ventilation to pressure-support ventilation).
3. A closed-loop algorithm to maintain the minute ventilation (as set by the operator) at the optimal tidal volume-respiratory frequency combination, according to the patient's respiratory mechanics.
4. Lung-protective rules to limit the range of tidal volume-respiratory frequency combinations, depending on the patient's respiratory mechanics.
5. The ability to switch back to manual control at any time.

Intrinsic requirements for such a comprehensive concept are breath-to-breath measurement of the patient's respiratory mechanics based on the volume-flow loop method¹, and dynamic compliance based on tidal pressure/volume relationship.

ASV has been tested in many situations. The two major studies are from Tasseaux and coworkers² and from Sultzer and coworkers³.

In the Tasseaux study² the objective was to compare the effects of ASV to the effects of synchronized intermittent mandatory ventilation plus pressure support (SIMV-PS) on patient-ventilator interactions in patients undergoing partial ventilatory support. The authors concluded that in patients with increased respiratory muscle loading, and for comparable minute ventilation, ASV was associated with decreased inspiratory load and improved patient-ventilator interactions.

The aim of the Sultzer study³ was to test the hypothesis that a protocol of respiratory weaning based on ASV could reduce the duration of tracheal intubation after uncomplicated cardiac surgery. A group of patients being given ASV was compared to a control group in a randomized controlled study. Forty-nine patients were enrolled. The primary outcome of the study, the duration of tracheal intubation, was shorter in the ASV group than in the control group (median [quartiles]: 3.2 [2.5-4.6] as compared to 4.1 [3.1-8.6] h; P < 0.02). Fewer arterial blood analyses were performed in the ASV group (median number [quartiles]: 3 [3-4] as compared to 4 [3-6]), suggesting that fewer changes in the settings of the ventilator were required in this group. The authors concluded that a respiratory weaning protocol based on ASV is practicable and may accelerate tracheal extubation and simplify ventilatory management in fast-track patients after cardiac surgery.

A recent study from Belliato and coworkers⁴ also compared the tidal volume-respiratory frequency combination given by the ventilator in patients with obstructive pulmonary disease to patients with restrictive pulmonary disease. This study confirms how much the ventilator is able to

differentiate between these types of respiratory disease, and able to adjust the ventilatory setting according to the patient's mechanics.

The present case report shows clearly how ASV works in COPD patients, and highlights the following very important and specific aspects of ASV in such patients:

1. The ability of ASV to adjust the tidal volume-respiratory frequency combination according to the patient's respiratory mechanics (Figure 4). In this case, the RCe was initially 3.4 seconds, which meant that the patient needed nearly 9 seconds to fully exhale the tidal volume. Consequently, the ventilator's respiratory frequency was relatively low (Figure 4).
2. The ability of ASV to detect the patient's spontaneous respiratory frequency and to adjust the ventilatory support according to the patient's condition. After stopping sedation, the patient's respiratory frequency was taken into account, and the ventilator's respiratory frequency decreased to zero (Figure 4).
3. The ability of ASV to limit PEEPi as much as possible, by taking into consideration the expiratory time constant (Figure 4).
4. The capacity of ASV to reduce weaning time in severe COPD patients. In less than a week, the patient was able to breathe alone, and the pressure given by the ventilator reduced sufficiently to make a T-piece trial practicable.

Conclusion

The present case clearly describes how ASV can be used in COPD patients with severe exacerbation requiring endotracheal intubation. In contrast with other modes of ventilation, ASV is able to track the patient's changes in respiratory mechanics, and to adjust ventilation accordingly.

Automatic detection of spontaneous activity, and automatic adjustment of inspiratory pressure, make ASV a fast-weaning mode of ventilation in such patients.

References

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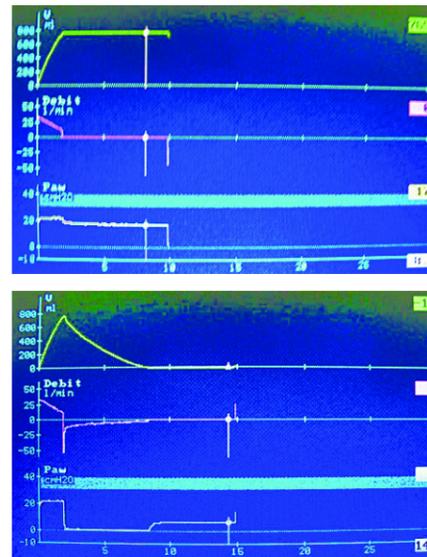


Figure 2: Above: End-inspiratory occlusion to determine the effective alveolar pressure at end-inspiration (Pplat). Below: End-expiratory occlusion to measure static intrinsic PEEP.

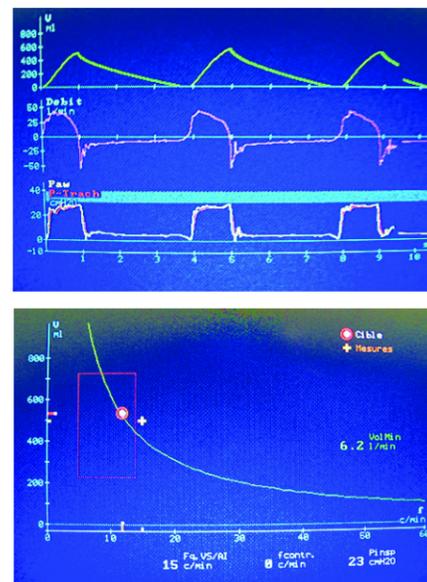
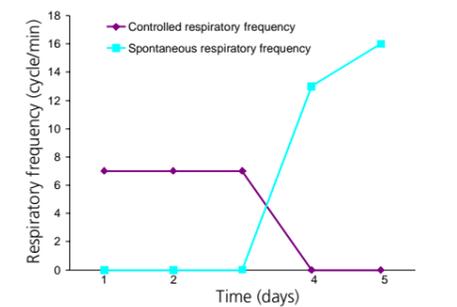
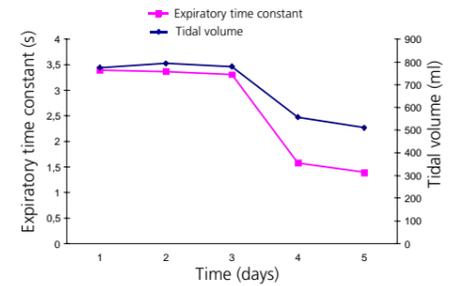


Figure 3: Above: Volume, flow and pressure traces during ASV ventilation on day 3. Below: Day 3 - the special ASV monitoring screen showing (on the tidal volume-respiratory frequency curve) the optimal Vt-FR combination calculated by the ASV algorithm (red circle) and the patient's current tidal volume and respiratory frequency (yellow cross).



Time (days)	D1	D2	D3	D4	D5
Pinsp (cm H ₂ O)	23	22	22	20	13
Pplat (cm H ₂ O)	17	19	18	-	-
PEEPi, stat (cm H ₂ O)	7	7.8	7	-	-
I:E	1:2	1:2	1:2	1:5	1:4
pH	7.39	7.42	7.37	7.40	7.43
PaO ₂ /FIO ₂	232	197	230	214	211
PCO ₂ (kPa)	7.5	7.8	8.5	9.3	8.8

Figure 4: Top: Evolution of the expiratory time constant and tidal volume delivered from day 1 to day 5. Middle: Evolution of controlled-to-spontaneous respiratory frequency from day 1 to day 5. Bottom: Evolution of the pressures, I:E ratio and arterial blood gas from day 1 to day 5.