Chapter 21 Intensive Care Unit Ventilators Some Aspects in Noninvasive Mechanical Ventilation



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Abbreviations

COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
ICU	Intensive care unit
NIV	Non invasive ventilation
PC-BiPAP	Pressure control, bilevel positive airway pressure
PEEP	Positive end expiratory pressure

21.1 Introduction

The standard management for patients in the ICU suffering from severe acute respiratory failure is becoming increasingly to consider NIV as a first option. In order to decide whether therapy with NIV is indicated, a careful analysis of both the type and the underlying cause of respiratory failure is necessary [1]. The success of NIV in the acute setting, is dependent on proper patient selection, interface, and ventilator capabilities with regard to leak management and auto-triggering. The choice of a ventilator may be crucial for the success of NIV in the acute setting, because intolerance and air leaks are significantly correlated with NIV failure especially in acute respiratory failure [2, 3].

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Supplementary Information The online version contains supplementary material available at [https://doi.org/10.1007/978-3-030-71298-3_21].

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A. M. Esquinas (ed.), *Teaching Pearls in Noninvasive Mechanical Ventilation*, https://doi.org/10.1007/978-3-030-71298-3_21

In this section two acute respiratory failure patients (First COPD exacerbation, and second acute de novo respiratory failure because of pneumonia and multiple malign metastases) will be discussed and recommendations for NIV with ICU ventilators will be written.

Case 1

A 66-year-old man with a history of end-stage COPD, having been intubated within the last month for a similar exacerbation, arrived by ambulance to our Emergency Department. The chief complaint was severe difficulty breathing which came on gradually. Initial assessment noted tachypnea with nasal flaring and purse lipped breathing, as well as bilateral wheezing and wet cough.

This patient is well known to our staff with multiple prior admissions. He has home mechanical ventilation as BiPAP–ST and is having long term oxygen therapy. Based on history, it was anticipated that this patient would be intubated and admitted to the ICU.

He had a smoking history of 1 pack per day for the past 47 years. On examination he appears cachectic and in severe respiratory distress. His neck veins are mildly distended. Lung examination reveals a barrel chest and poor air entry bilaterally, with severe inspiratory and expiratory wheezing. Heart and abdominal examination are within normal limits. Lower extremities exhibit scant pitting edema. He has beard. Arterial blood gas examination reveals respiratory acidosis (PH: 7.25, PaCO₂: 86 mmHg, PaO₂: 55 mmHg, SaO₂: 85%) although he was using his home ventilator for 24 h before admission (In acute illness CO₂ rebreathing occurred).

In ICU NIV began with ICU ventilator. The setting of NIV was as below:

On NIV mode spontan-CPAP was began with Δ Psupp: 18 mmHg PEEP: 5 mmHg, FiO₂: 60%, ramp: 0.40 ms.

On this settings, patient became confused and apneas occurred. He could not trigger the ventilator. Arterial blood gases became more acidotic (PH: 7.21 PaCO₂: 90 mmHg, PaO₂: 70 mmHg, SaO₂: 92%). We thought leak occurred because of his beard. Although the ICU ventilators have leak compensation, if the leak reaches above 80% this can be problem for triggering the ventilator and apnea occurred. Both severe respiratory acidosis and leak from mask patient became more sick. His mouth and nose dried. We shaved the beard and changed the ventilator mode to PC-BiPAP, added heated humidifier and settings was made as below:

PEEP: 5 mmHg, FiO₂: 60%, ramp: 0.10 ms, Pressure support: 18 mmHg, respiratory rate: 15/min, I/E ratio: 1/2, Trigger sensitivity: 2 L/min.

Arterial blood gases improved (PH: 7.37 PaCO₂: 55 mmHg, PaO₂: 78 mmHg, SaO₂: 94%).

Case 2

A 30-year-old man with acute respiratory failure due to pneumonia admitted to ICU. He has multiple pulmonary metastasis because of malign mesenchymal cancer. On examination he appears in severe respiratory distress. He was tachypneic.

His arterial blood gases reveals hypoxic respiratory failure (PH: 7.48, PaCO₂: 28 mmHg, PaO₂: 55 mmHg, SaO₂: 85%). NIV started with ICU ventilator.

The initial setting was as below:

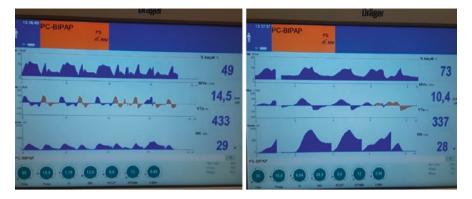


Fig. 21.1 Pressure controlled ventilation as; PEEP: 5 mmHg, FiO_2 : 55%, ramp: 0.40 ms, Pressure support: 12 mmHg, respiratory rate: 12/min, İnspirium time: 1.7 s, Trigger sensitivity: 2 L/min. On this settings autotriggering was seen on waves (first). We increased the respiratory rate to 29/min. Patient ventilator synchrony occurred (Second) although 73% leak

On NIV mode spontan-CPAP was began with Δ Psupp: 16 mmHg PEEP: 5 mmHg, FiO₂: 60%, ramp: 0.10 ms, Trigger sensitivity: 2 L/min.

On this settings he became tachypneic and ventilator waves reveal double triggering. Although leak compensation of ICU ventilator, we thought that it is due to leak (64%) from mask. We increased trigger sensitivity to 4 L/min and tighten the mask. On this settings apnea occurred. Patients NIV settings were revised. Mode changed to pressure controlled ventilation as; PEEP: 5 mmHg, FiO₂: 55%, ramp: 0.40 ms, Pressure support: 12 mmHg, respiratory rate: 12/min, İnspirium time: 1.7 s, Trigger sensitivity: 2 L/min. On this settings autotriggering was seen on waves. We increased the respiratory rate to 29/min. Patient ventilator sencrony occured (Fig. 21.1) although 73% leak. Because leak compensation of ICU ventilator is good enough.

21.2 Discussion

Here the cases with acute respiratory failure due to COPD exacerbation and *de novo* respiratory failure treated with ICU ventilators in NIV modes were described.

Even if any ventilator can be theoretically used to start NIV both in acute and chronic respiratory failure, success is more likely if the ventilator is able to (a) adequately compensate for leaks; (b) let clinician continuously monitor patient-ventilator synchrony and ventilator parameters due to a display of pressure-flow-volume waveforms and a double-limb circuit; (c) adjust the fraction of inspired oxygen (FiO₂) to assure stable oxygenation; and (d) adjust inspiratory trigger sensitivity and expiratory cycling as an aid to manage patient-ventilator asynchronies [3, 4].

In first case we used ICU ventilator for NIV in COPD exacerbation for decreasing CO_2 levels and avoiding CO_2 rebreathing. Patient had hypercapnia although using home ventilator. Because he had high respiratory rate and one limb circuit of home NIV, he rebreated CO_2 . Using two limb circuit ventilator can avoid CO_2 rebreathing especially in patients with tachypnea. It is important to use two limb circuit in patients with high arterial CO_2 levels.

Types of ventilators have been commonly used for NIV in acute settings are regular ICU ventilators (with no NIV capabilities or algorithm), ICU ventilators with NIV algorithm, and dedicated NIV ventilators. ICU ventilators are more powerful and have more adjustable features (trigger type and sensitivity, slope of pressurization, cycling criteria) and monitoring capabilities [4, 5].

New generation ICU ventilators has good leak compensation, which allows a partial or total correction of air leak-induced patient-ventilator asynchrony, even with large amount of leaks. If leak flow reaches the trigger threshold, auto-triggering occurs. On the other hand, if the leak is large enough, the ventilator may not detect respiratory efforts, or patient can not start the respiration leading to miss-triggering or apnea as in our first patient. Additionally, leaks can lead to aerophagia, odynophagia, dry mouth, eye irritation, and nasal symptoms, and noise may result, all of which reduce therapeutic compliance [5]. In acute settings both in *de novo* respiratory failure and acute on chronic respiratory failure, using ICU ventilators for NIV can be more successful because of patient–ventilator synchrony (leak compensation), monitoring patient, avoiding CO_2 rebreathing (two limb circuit).

The ventilators may automatically decrease trigger sensitivity according to the level of leak to avoid auto-triggering, but as the leak decreases, the trigger sensitivity increases. This can lead to miss-triggering, particularly if the change is larger than the inspiratory effort. If the change in leak is smaller than the inspiratory effort, miss-triggering is unlikely, though higher patient effort is required to reach this threshold [6]. In acute respiratory failure with air hunger as in our second patient because of patients high respiratory rate autotriggering can be occurred and increasing ventilator respiratory rate can solve the problem.

In acute respiratory failure, NIV algorithms provided by ICU ventilators can reduce the incidence of asynchronies because of leaks, thus confirming bench test results, but some of these algorithms can generate premature cycling.

Key Teaching Points

- In acute respiratory failure especially patients with high CO₂ levels and high respiratory rate NIV with ICU ventilators with an O₂ blender and dual limb circuit is more appropriate than only NIV ventilators
- ICU ventilators have dual limb circuit for avoiding CO₂ rebreathing. In patients with COPD exacerbation dual limb circuit has an advantage in lowering CO₂.
- NIV with ICU ventilators can avoid patient ventilator asynchrony in acute settings by leak compensation but there is a wide range of heterogeneity among ICU ventilators in the leak compensation algorithms.

- Too sensitive trigger, especially if flow based, may induce auto triggering during NIV with substantial air leaks and, consequently, ventilator dys-synchrony due to unwanted efforts.
- NIV algorithms mostly improve ICU ventilator performance in NIV, however, modifications still have to be carried out to prevent triggering and cycling asynchrony.

References

- 1. Nava S, Hill N. Non-invasive ventilation in acute respiratory failure. Lancet. 2009;374(9685):250–9.
- Ozsancak A, D'Ambrosio C, Hill NS. Nocturnal noninvasive ventilation. Chest. 2008;133(5):1275–86.
- Vignaux L, Vargas F, Roeseler J, et al. Patient-ventilator asynchrony during non-invasive ventilation for acute respiratory failure: a multicenter study. Intensive Care Med. 2009;35(5):840–6.
- Di Marco F, Centanni S, Bellone A, et al. Optimization of ventilator setting by flow and pressure waveforms analysis during noninvasive ventilation for acute exacerbations of COPD: a multicentric randomized controlled trial. Crit Care. 2011;15(6):R283.
- 5. Hess DR. Patient-ventilator interaction during noninvasive ventilation. Respir Care. 2011;56(2):153-65.
- Carteaux G, Lyazidi A, Cordoba-Izquierdo A, Vignaux L, Jolliet P, Thille AW, et al. Patientventilator asynchrony during noninvasive ventilation: a bench and clinical study. Chest. 2012;142(2):367–76.