Mechanical Ventilation in Specific Cases

A summary of highlights from presentations at the 1st ERS – Respiratory Failure and Mechanical Ventilation Conference 2020 by Lise Piquilloud (Lausanne, Switzerland), Martin Dres (Paris, France) and Nicholas Hart (London, United Kingdom)

There are several primary indications for initiating mechanical ventilation (MV). The first presentation by Dr Piquilloud focussed on MV in obstructive lung disease. The term obstructive respiratory disease refers to two distinct disease entities – chronic obstructive pulmonary disease (COPD) and asthma – with one common feature: airway obstruction causing increased resistance to airflow and dynamic hyperinflation. In an obstructed lung, air becomes trapped in the lung at the end of expiration, and therefore lung volume exceeds functional residual capacity. Pressure in the alveoli is higher than atmospheric pressure, and there is intrinsic positive end-expiratory pressure (PEEP).

This hyperinflation can worsen when we ventilate obstructive patients. It increases the work of breathing, decreases compliance, and leads to abnormal blood gases and decreased venous return, leading to increased respiratory rates and haemodynamic instability. When a patient is ventilated, the blood pressure and cardiac output is low. In an emergency situation, the ventilator can be disconnected, and the settings changed when reconnected.[1] Monitoring hyperinflation is mandatory; it is evident when the expiratory flow does not come back to zero, but to quantify this, we need to perform an exploratory occlusion.[2]

Dr Piquilloud reported that there is no definitive optimal ventilation mode in patients with obstructive respiratory disease but volume assist controlled (VAC) ventilation has advantages. Minute ventilation is set and is not influenced by variations in obstruction severity. Airway pressure can be monitored and safety limits can be set. In addition, it is easy to monitor respiratory mechanics and air trapping monitoring. However, VAC is not mandatory and other modes can be used.

It is important to optimise the ventilator settings in patients with obstructive respiratory disease. During controlled ventilation, to reduce hyperinflation we should reduce the minute ventilation to lower the volume to exhale. We can achieve this by keeping tidal volume (VT) as low as possible (6–8 ml/kg), and reducing respiratory rate (RR), keeping the minute ventilation below 10L/min, and controlling plateau pressure (below 30cm H_2O).[3] A very low RR (<10/min) is often not needed if minute ventilation is low, except in very severe patients (Pplat >30) or if hyperinflation related complications have already occurred.[4] We should also increase inspiratory flow (min 60 l/min), change I:E ratios (minimum 1:3 to 1:4) and maintain low PEEP (usually ≤5 cmH_2O).[3] One consequence of reducing hyperinflation is that we should accept controlled

hypoventilation, hypercapnia and a low pH when we ventilate obstructive patients.[5,6] However, we must be careful to maintain good oxygenation. Contraindications for this approach are brain injury and right ventricular failure.[6,7]

Another important consideration is how to set PEEP; however, little data exists to inform this decision. Setting PEEP at zero is seldom required. Applying PEEP during controlled ventilation in an obstructive patient can increase resistance and hyperinflation. However, applying PEEP can improve oxygenation and prevent atelectasis.[8] In MV for severe asthma, it is important to keep PEEP less than 80% of intrinsic PEEP.[9]

Mechanical ventilation also presents challenges in patients with interstitial lung disease (ILD), and this was the focus of the second presentation by Dr Dress. It is not commonly used (less 10% of ILD patients receive MV),[10] and is associated with a poor prognosis (in hospital mortality is over 50%).[10–12] Risk factors for mortality include elevated Acute Physiology and Chronic Health Evaluation (APACHE) score, hypoxaemia, and MV use.[11,12] One study showed that only 25% of patients with fibrosing interstitial pneumonia receiving MV were alive at 30 days.[13] In the intensive care unit (ICU), ILD under MV most closely resembles acute respiratory distress syndrome (ARDS).[14] The major differences are poor lung compliance, low potential for lung recruitment, and a high risk for ventilator induced injury.

It is essential to optimise the ventilator settings in patients with ILD. One study showed that PEEP is an important determinant of mortality: PEEP of less than 5 cmH_2O was associated with better survival.[15] It is also important to investigate the trigger that resulted in the patient being in the ICU and exclude infections, cardiac dysfunction and drug toxicity before considering acute exacerbation.[16] Clinical data does

not support the use of corticosteroids in ARDS,[17] and it is reasonable to assume that this is also the case in ILD. Lung transplant should be considered in patients with fibrosing interstitial pneumonia but is uncommon.[13]

Extracorporeal membrane oxygenation (ECMO) may also be considered but ethical issues should be taken into account when recovery seems out of reach.[18]

The third presentation from Prof Hart discussed noninvasive ventilation (NIV) strategies during weaning from intensive ventilatory support. Weaning centres are growing as patients are liberated from ventilators earlier and undergo pulmonary rehabilitation. Patients should be categorised into three groups based on the difficulty and duration of the weaning process:[19]

- Simple – can be extubated after the first spontaneous breathing trial (SBT)
- Difficult – needing up to 3 SBTs and up to 7 days from the first SBT
- Prolonged – more that 3 SBTs and more than 7 days from the first SBT

Both extubation delay and the need for reintubation are associated with poor outcomes.[20]

Strategies to improve weaning rate involve systematically approaching the patient and assessing their respiratory and cognitive responses to reducing support. An SBT is the major diagnostic test to determine whether patients can be successfully extubated.[19] However, some patients experience respiratory failure after extubation.[21] Noninvasive ventilation has been shown to reduces weaning time, shorten the time in ICU, and improve survival rates.[22,23] However, most studies to date have involved participants with COPD.[23]

The Breathe randomised controlled trial investigated the effectiveness of NIV in a general intensive care patient population.[24] The majority of patients had pneumonia or post- surgical respiratory failure. Patients were randomised to receive either protocolized weaning via early extubation to NIV or protocolized standard weaning (continued invasive ventilation until successful spontaneous breathing trial, followed by extubation). Results showed no difference between the two groups in terms of the primary outcome, time to liberation from ventilation. However, the findings in secondary outcomes were positive, including reduced invasive ventilator days, reduced risk of ventilator assisted pneumonia as shown by the lower use of respiratory antibiotics, reduction in sedation days, reduced ICU days, and a higher rate of extubation. Early extubation to NIV increased the re-intubation rate but there was no effect on adverse event rate, tracheostomy rate or survival.

The investigators concluded that early extubation to NIV is a safe and effective intermediate step in the weaning process in a general ICU population who have failed a SBT. Early extubation to NIV could encourage early mobilisation as the risk of accidental extubation is removed, enhancing staff confidence.

In summary, ventilatory support can be a lifesaving procedure in a number of respiratory conditions, improving gas exchange and relieving respiratory distress. However, it can cause considerable increase in morbidity and mortality if not used properly. Therefore, it is necessary to have a good understanding of pathophysiology, mechanics and pattern of flow obstruction to provide the most suitable ventilation to individual patients. Strategies for weaning from intensive ventilatory support are also important. Evidence has been presented that supports early use of NIV during weaning.

References

1. Pepe PE, Marini JJ, Occult positive end-expiratory pressure in mechanically ventilated patients with airflow obstruction: the auto-PEEP effect, Am Rev Respir Dis, 1982;126:166-70;10.1164/arrd.1982.126.1.166;7046541.
2. Blanch L, Bernabe F, Lucangelo U, Measurement of air trapping, intrinsic positive end-expiratory pressure, and dynamic hyperinflation in mechanically ventilated patients, Respir Care, 2005;50:110-23; discussion 23-4;15636649.
3. Tuxen DV, Lane S, The effects of ventilatory pattern on hyperinflation, airway pressures, and circulation in mechanical ventilation of patients with severe air-flow obstruction, Am Rev Respir Dis, 1987;136:872-9;10.1164/ajrccm/136.4.872;3662241.
4. Leatherman JW, McArthur C, Shapiro RS, Effect of prolongation of expiratory time on dynamic hyperinflation in mechanically ventilated patients with severe asthma, Crit Care Med, 2004;32:1542-5;10.1097/01.ccm.0000130993.43076.20;15241099.
5. Darioli R, Perret C, Mechanical controlled hypoventilation in status asthmaticus, Am Rev Respir Dis, 1984;129:385-7;10.1164/arrd.1984.129.3.385;6703497.
6. Feihl F, Perret C, Permissive hypercapnia. How permissive should we be?, Am J Respir Crit Care Med, 1994;150:1722-37;10.1164/ajrccm.150.6.7952641;7952641.
7. Peigang Y, Marini JJ, Ventilation of patients with asthma and chronic obstructive pulmonary disease, Curr Opin Crit Care, 2002;8:70-6;10.1097/00075198-200202000-00011;12205409.
8. Tuxen DV, Detrimental effects of positive end-expiratory pressure during controlled mechanical ventilation of patients with severe airflow obstruction, Am Rev Respir Dis, 1989;140:5-9;10.1164/ajrccm/140.1.5;2665589.
9. Leatherman J, Mechanical ventilation for severe asthma, Chest, 2015;147:1671-80;10.1378/chest.14-1733;26033128.
10. Rush B, Wiskar K, Berger L, et al., The use of mechanical ventilation in patients with idiopathic pulmonary fibrosis in the United States: A nationwide retrospective cohort analysis, Respir Med, 2016;111:72-6;10.1016/j.rmed.2015.12.005;26733227.
11. Huapaya JA, Wilfong EM, Harden CT, et al., Risk factors for mortality and mortality rates in interstitial lung disease patients in the intensive care unit, Eur Respir Rev, 2018;27:10.1183/16000617.0061-2018;30463873.
12. Durheim MT, Judy J, Bender S, et al., In-Hospital Mortality in Patients with Idiopathic Pulmonary Fibrosis: A US Cohort Study, Lung, 2019;197:699-707;10.1007/s00408-019-00270-z;31541276.
13. Gaudry S, Vincent F, Rabbat A, et al., Invasive mechanical ventilation in patients with fibrosing interstitial pneumonia, J Thorac Cardiovasc Surg, 2014;147:47-53;10.1016/j.jtcvs.2013.06.039;23968871.
14. Collard HR, Ryerson CJ, Corte TJ, et al., Acute Exacerbation of Idiopathic Pulmonary Fibrosis. An International Working Group Report, Am J Respir Crit Care Med, 2016;194:265-75;10.1164/rccm.201604-0801CI;27299520.
15. Fernandez-Perez ER, Yilmaz M, Jenad H, et al., Ventilator settings and outcome of respiratory failure in chronic interstitial lung disease, Chest, 2008;133:1113-9;10.1378/chest.07-1481;17989156.
16. Suh GY, Kang EH, Chung MP, et al., Early intervention can improve clinical outcome of acute interstitial pneumonia, Chest, 2006;129:753-61;10.1378/chest.129.3.753;16537878.
17. Steinberg KP, Hudson LD, Goodman RB, et al., Efficacy and safety of corticosteroids for persistent acute respiratory distress syndrome, N Engl J Med, 2006;354:1671-84;10.1056/NEJMoa051693;16625008.
18. Rozencwajg S, Schmidt M, Extracorporeal membrane oxygenation for interstitial lung disease: what is on the other side of the bridge?, J Thorac Dis, 2016;8:1918-20;10.21037/jtd.2016.07.50;27619972.
19. Boles JM, Bion J, Connors A, et al., Weaning from mechanical ventilation, Eur Respir J, 2007;29:1033-56;10.1183/09031936.00010206;17470624.
20. Thille AW, Cortes-Puch I, Esteban A, Weaning from the ventilator and extubation in ICU, Curr Opin Crit Care, 2013;19:57-64;10.1097/MCC.0b013e32835c5095;23235542.
21. Esteban A, Frutos-Vivar F, Ferguson ND, et al., Noninvasive positive-pressure ventilation for respiratory failure after extubation, N Engl J Med, 2004;350:2452-60;10.1056/NEJMoa032736;15190137.
22. Nava S, Ambrosino N, Clini E, et al., Noninvasive mechanical ventilation in the weaning of patients with respiratory failure due to chronic obstructive

pulmonary disease. A randomized, controlled trial, Ann Intern Med, 1998;128:721-8;10.7326/0003-4819-128-9-199805010-00004;9556465.
23. Burns KE, Adhikari NK, Keenan SP, et al., Use of non-invasive ventilation to wean critically ill adults off invasive ventilation: meta-analysis and systematic review, BMJ, 2009;338:b1574;10.1136/bmj.b1574;19460803.
24. Perkins GD, Mistry D, Gates S, et al., Effect of Protocolized Weaning With Early Extubation to Noninvasive Ventilation vs Invasive Weaning on Time to Liberation From Mechanical Ventilation Among Patients With Respiratory Failure: The Breathe Randomized Clinical Trial, JAMA, 2018;320:1881-8;10.1001/jama.2018.13763;30347090.