PATIENT-CENTRED ACUTE CARE TRAINING

AN ESICM MULTIDISCIPLINARY DISTANCE LEARNING PROGRAMME FOR INTENSIVE CARE TRAINING

Mechanical ventilation

Skills and techniques

Update 2011

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Mechanical ventilation
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LEARNING OBJECTIVES

After studying this module on Mechanical ventilation, you should:

1. Understand the mechanical causes of respiratory failure
2. Have the knowledge to institute mechanical ventilation safely
3. Understand the principles that guide mechanical ventilation
4. Be able to apply these principles in clinical practice

FACULTY DISCLOSURES

The authors of this module have not reported any associated disclosures.

DURATION

9 hours

INTRODUCTION

The mechanical ventilator is an artificial, external organ, which was conceived originally to replace, and later to assist, the inspiratory muscles. The primary function of mechanical ventilators is to promote alveolar ventilation and CO_2 elimination, but they are often also used for correcting impaired oxygenation – which may be a difficult task.

The concept and implementation of ventilation is relatively straightforward in most patients and clinicians starting to work in Intensive Care usually become familiar with the everyday workings of initiating, maintaining and de-escalating/weaning patients from mechanical ventilation using the modes of ventilation commonly used in that particular environment. This module deals with the everyday facets of such care but also addresses in some detail the approach to difficult ventilation problems in patients with severe, complex and evolving lung disease.

Although the mechanical ventilators can be lifesaving, they may at the same time be hazardous machines. In-depth knowledge of mechanical ventilation is of paramount importance for the successful and safe use of ventilators in the full variety of critical care situations and is a core element of critical care practice.

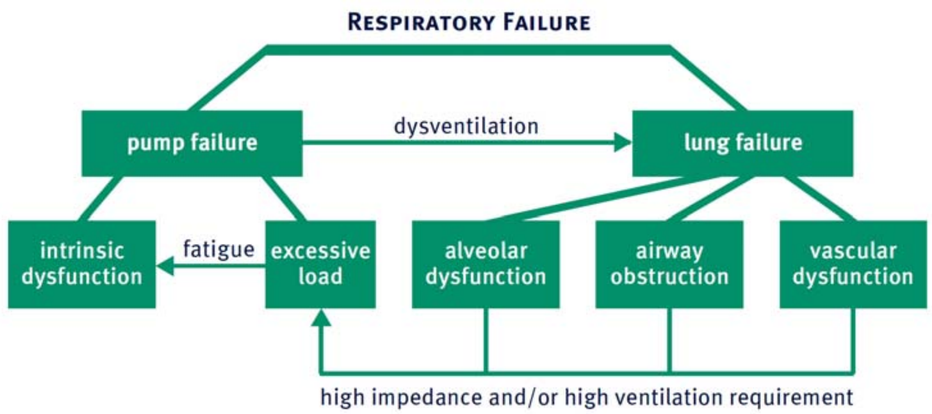
In the online appendix, you will find four original computer-based interactive tools for training in mechanical ventilation. Additional illustrative materials are available online.

1/ THE NATURE OF RESPIRATORY FAILURE

Respiratory failure is usually classified as pump failure (failure of ventilatory function) which is termed type 2 failure or as lung failure (failure of the lung parenchyma), often termed type 1 failure.

Pump failure or lung failure?

The respiratory system can be modelled as a gas exchanger (the lungs) ventilated by a pump. Dysfunction of either, pump or lungs, can cause respiratory failure, defined as an inability to maintain adequate gas exchange while breathing ambient air.



> *Pump failure and lung failure rarely occur in isolation, in intensive care patients. Frequently a patient alternates between prevalent pump failure and prevalent lung failure, during the course of their illness.*

Pump failure

> *Pump failure may cause lung failure due to accumulation of secretions, inadequate ventilation and atelectasis*

Pump failure primarily results in alveolar hypoventilation, hypercapnia and respiratory acidosis. Inadequate alveolar ventilation may result from a number of causes intrinsically affecting one or more components of the complex pathway that begins:

- In the respiratory centres (pump controller)
- Continues with central and peripheral motor nerves
- Ends with the chest wall, including both the respiratory muscles and all the passive elements that couple the muscles with the lungs.

Alveolar hypoventilation may even be seen in the absence of any intrinsic problem of the pump, when a high ventilation load overwhelms the reserve capacity of the pump. Excessive load can be caused by airway obstruction, respiratory system stiffening (low compliance) or a high ventilation requirement culminating in intrinsic pump dysfunction due to respiratory muscle fatigue.

Lung failure

Lung failure may cause pump failure, due to high impedance and increased ventilation requirement

Lung failure results from damage to the gas exchanger units: alveoli, airways and vessels.
See PACT module on Acute respiratory failure for additional information.

Lung failure involves impaired oxygenation and impaired CO_2 elimination depending on a variable combination of

- Ventilation/perfusion mismatch
- True intrapulmonary shunt
- Increased alveolar dead space

Lung injury is also associated with increased ventilation requirements and mechanical dysfunction resulting in high impedance to ventilation. Impedence of the respiratory system is most commonly expressed by the quantifiable elements of respiratory system resistance, respiratory system compliance, and intrinsic PEEP (positive end-expiratory pressure).

Role of mechanical ventilation

*Intensivists have been learning for decades, and are still learning, how to **effectively and safely** use mechanical ventilation in lung failure*

Mechanical ventilation was initially conceived as symptomatic treatment for pump failure. The failing muscular pump is assisted or substituted by an external pump. Because of technological limitations in the early days, substitution was the only choice. Today, technological advances allow mechanical ventilators to be used as sophisticated assistants of the respiratory pump.

Positive pressure ventilation (see Task 4) can also be very effective in primary lung failure. In this context, the safe management of mechanical ventilation requires precise information about altered respiratory mechanics in the individual patient, in order to tailor a strategy that protects the respiratory system from further damage (ventilator-associated lung injury – VALI), and provide an environment that promotes lung healing. In the most severe cases with extreme mechanical derangements, these objectives can be difficult to achieve.

You can find information on applied respiratory physiology and acute respiratory failure in the following links and references.

Charles Gomersall videos on applied respiratory physiology and acute respiratory failure

Hinds CJ, Watson JD. Intensive Care: A Concise Textbook. 3rd edition. Saunders Ltd; 2008. ISBN: 978-0-7020259-6-9. pp. 195–199. Causes of Respiratory failure

Fink MP, Abraham E, Vincent J-L, Kochanek PM, editors. Textbook of Critical Care. 5th edition. Elsevier Saunders, Philadelphia, PA; 2005. p. 571-734
See also the PACT modules on Acute respiratory failure, COPD and asthma.

2/ INITIATING (AND DE-ESCALATING) MECHANICAL VENTILATION

In critical care, the indicaton for mechanical ventilation may be simply for the management of ventilatory (pump) failure e.g. post operatively or for drug intoxication. Often however, it is required for acute respiratory failure due to parenchymal lung disease.

See the PACT module on Acute respiratory failure.

Invasive vs non-invasive techniques

In intensive care, positive pressure ventilators (devices that promote alveolar ventilation by applying positive pressures at the airway opening) are most often used. To transmit positive pressure to the respiratory system, the ventilator must be connected to the patient by means of an interface that guarantees a reasonably effective pneumatic seal. Two kinds of interface are used:
- Tracheal tube (or tracheostomy): the traditional, invasive approach
- Mask: The non-invasive approach.

The invasiveness of endotracheal intubation is the high price paid for maximum safety and flexibility

Tracheal intubation artificially bypasses the upper airway to the lower third of the trachea, with a reliable pneumatic seal. Such tubes have a number of advantages:
- Protecting the lungs from major aspiration
- Protect the upper airway and gastrointestinal tract from positive pressure
- Relieving upper airway obstruction
- Providing easy access to the airway for suction and bronchoscopy
- Reducing dead space
- Enabling a stable and safe connection between the ventilator apparatus and the patient.

If necessary, tracheal intubation enables ventilation modes that provide full control of ventilation.The invasive approach to mechanical ventilation has however a number of disadvantages associated with tracheal intubation including:
- Loss of the protective functions of the upper airway (heating and humidification of inspired gases and protection from infection)
- Decreased effectiveness of cough (risk of sputum retention/atelectatsis)
- Increased airway resistance
- Risk of airway injury
- Loss of the ability to speak.

These disadvantages do not apply to non-invasive mechanical ventilation (NIMV). In carefully selected patients (see below), NIMV is more comfortable and reduces the duration of mechanical ventilation and the incidence of ventilator-associated pneumonia (VAP). For further information about tracheal intubation, read the following reference:

Hinds CJ, Watson JD. Intensive Care: A Concise Textbook. 3rd edition. Saunders Ltd; 2008. ISBN: 978-0-7020259-6-9. pp. 184–186. Tracheal intubation

See also the PACT module on Airway management.

Non-invasive mechanical ventilation (NIMV): When effective, it may be associated with a better outcome but switching to the invasive approach will often be necessary

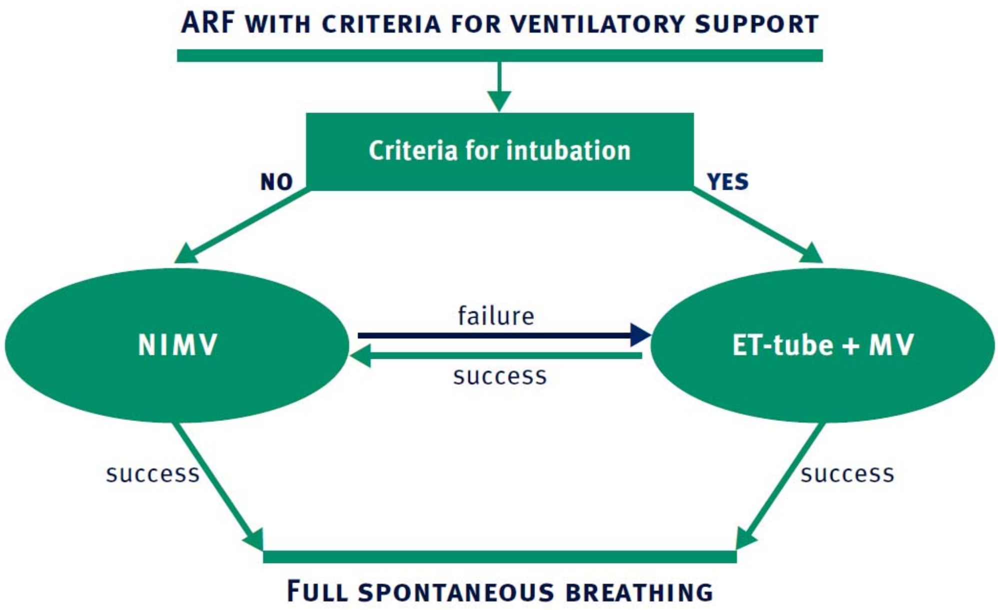
Safe and effective management of mask ventilation requires:
- At least some residual spontaneous breathing (the need for full mechanical support is an absolute contraindication to a non-invasive approach)
- No anticipation that high levels of positive pressure being required
- Ability to tolerate temporary disconnection from the ventilator
- Haemodynamic stability
- Co-operative patient
- The ability of the patient to protect their own airway
- No acute facial trauma, basal skull fracture, or recent digestive tract surgery

When assessing your next ten patients with acute respiratory failure requiring mechanical support, consider the question: **is the need for the tracheal tube merely to be an interface with the mechanical ventilator?**
If the answer is yes, check whether all the requirements for mask ventilation are fulfilled, and discuss with colleagues whether non-invasive ventilation might be better used as the initial approach.

NOTE Mask ventilation is often a reasonable initial approach, as long as the patient's condition is closely monitored and the clinical team is ready to progress to tracheal intubation at any time.

The non-invasive approach, often continuous positive airway pressure (CPAP) initially, will often progress to early initiation of mechanical respiratory support which is most likely to be effective when mechanical support is needed for just a few hours (rapidly reversible cardiogenic lung oedema is a typical example) or when it is applied only intermittently. In other cases, deteriorating lung function will necessitate tracheal intubation. Later, non-invasive ventilation can be reconsidered to assist weaning of an intubated patient, thus allowing earlier extubation. Planned NIMV immediately after extubation, in patients with hypercapnic respiratory disease, has been shown to improve outcome, see reference below.

Ferrer M, Sellarés J, Valencia M, Carrillo A, Gonzalez G, Badia JR, et al. Non-invasive ventilation after extubation in hypercapnic patients with chronic respiratory disorders: randomised controlled trial. Lancet 2009; 374(9695): 1082-1088. PMID 19682735



Decision making between invasive and non-invasive ventilation (NIMV) at different stages of patient's course

For general information about non-invasive ventilation in intensive care, refer to the PACT module on Acute respiratory failure and the first reference below. See the second reference for information about interfaces and ventilators specifically designed for non-invasive ventilation.

Hinds CJ, Watson JD. Intensive Care: A Concise Textbook. 3rd edition. Saunders Ltd; 2008. ISBN: 978-0-7020259-6-9. pp. 176–179. Continuous positive airway pressure

Branson RD, Hess DR, Chatburn RL, editors. Respiratory care equipment. 2nd ed. Philadelphia: Lippincott Williams and Wilkins; 2000. p. 593. ISBN 0781712009

Strategies and timing

The basic concept of initiating mechanical ventilation is not difficult and entails setting the inspired oxygen concentration (FiO2) and positive end-expiratory pressure (PEEP) to control patient oxygenation and attending to the tidal volume (Vt) and respiratory rate/frequency (Fr) as controllers of CO2 elimination.

See underlying physiological principles in Task 3 which starts with management of CO2 elimination.

The choice of the most appropriate ventilation mode and settings may be complex but most centres make regular use of a limited number of modes, familiarity with which is fairly straightforward.

The successful application of the principles (See Tasks 3 and 4) relies on the correct recognition of the clinical context of each patient, described by at least four elements, summarised below.

PHYSIOLOGICAL TASKS TO MANAGE	PRIMARY LUNG DISEASE	TIMING	GENERAL APPROACH
◆ CO_2 elimination ◆ Oxygenation ◆ Assistance of respiratory muscles	◆ No lung disease ◆ Restrictive ◆ Obstructive	◆ Start-up ◆ Escalation and maintenance ◆ De-escalation and weaning	◆ Aggressive (normal blood gases targets) ◆ Conservative (lung protection) ◆ Balanced

In a given clinical context, more than one choice can be clinically acceptable. Consensus is more frequent with regard to what should be avoided, rather than what should be selected. Also, the choice necessarily depends on the equipment usually used in that clinical setting, as well as on the experience of the staff.

Initiating ventilator support

In less severe cases, when there is no independent indication for intubation, the initial support can be performed with pressure-support ventilation (PSV) delivered by mask.

In more severe cases and when mask ventilation fails, intubation is necessary, and support will be initiated with volume-controlled ventilation (VCV) or pressure-controlled ventilation (PCV). The traditional initiation with VCV is not essential.

When oxygenation is severely compromised, ventilation should be started with an FiO_2 of 1, while PEEP, when indicated, should be progressively escalated

Sound principles for management of mechanical ventilation include:
-Appropriate choice between non-invasive and invasive ventilation
-Maintenance of spontaneous respiratory activity if possible
- Adaptation of the ventilatory pattern to the nature of lung disease (restrictive or obstructive)
- Optimisation of alveolar recruitment
- Lung protective strategy

Escalation and maintenance

When mask ventilation is successful, maintenance involves continuous or intermittent PSV by mask. In intubated patients according to the severity of lung disease, associated diseases, the need for sedation, and respiratory muscles status, it may be necessary to either:
- Maintain strict control of ventilation, by using volume-controlled ventilation (VCV), pressure-controlled ventilation (PCV), biphasic positive airway pressure (BIPAP) or synchronised intermittent mandatory ventilation (SIMV) or PC-SIMV (SIMV using pressure-control to determine the Vt) set with relatively high mandatory frequency – see Task 4 for detail of these ventilator modes.

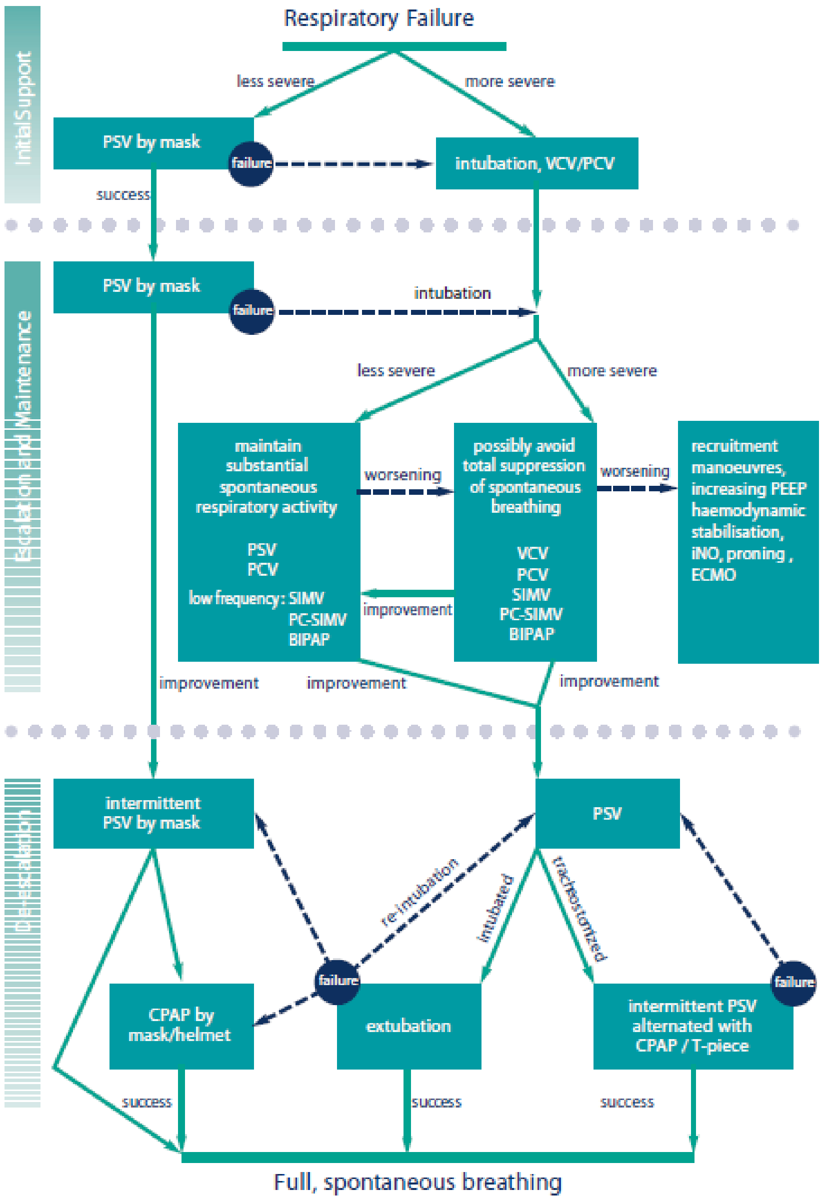
Or, if possible
- Allow a greater degree of patient-ventilator interaction, by using pressure-support ventilation (PSV), BIPAP or alternatively, SIMV/PC-SIMV at low mandatory frequency

Even in the most severe cases, VCV is not always a necessary choice in the modern context. PCV may be a more sensible choice for lung protection. In very severe lung disease, either restrictive or obstructive, the choice of ventilator settings can be more important than the choice between VCV and PCV.

The ventilatory pattern should be selected according to the type of lung disease. Low frequency and low I:E ratio are necessary in severe airway obstruction, while low tidal volumes, relatively high frequency and increased I:E ratios should be selected in severe hypoxaemic, restrictive disease. In very severe lung disease, controlled hypoventilation and permissive hypercapnia should be considered when otherwise not contraindicated.

In patients with refractory hypoxia, supplemental strategies including recruitment manoeuvres, increasing PEEP level, haemodynamic stabilisation, inhaled nitric oxide, proning (prone positioning) and extracorporeal membrane oxygenation should be considered.

A possible strategy for the clinical management of mechanical ventilation



For simplicity, the flowchart considers only the conventional primary modes of ventilation

Sedation is frequently necessary, but total suppression of spontaneous respiratory activity and pharmacological paralysis should be avoided whenever possible. Modes with pressure-controlled management of inspiration (PCV, PC-SIMV, BIPAP, PSV) allow a better matching between the patient's flow demand and ventilator flow delivery when compared to modes such as VCV and SIMV. The inspiratory pressure should be set to achieve a balanced spontaneous respiratory activity, neither too high nor too low.

Q. A patient is assisted by a pressure-support level of 10 cmH_2O. Frequency is 28 b/min, blood gases and haemodynamics are satisfactory. How can you decide whether the spontaneous respiratory load is excessive or not?

A. In addition to observing the respiratory rate and the tidal volume being achieved, asking the patient's opinion and observing respiratory coordination are important additional elements for deciding the adequacy of mechanical assistance.

Although in actively breathing patients, the ventilatory pattern is mainly patient-controlled, the ventilator can powerfully affect the output of the respiratory centre. Therefore, exactly as in (pharmacologically) paralysed patients, you should formulate optimal ventilatory targets, adapted to the type of lung disease, (e.g. restrictive or obstructive). Again, a reduced Vt target should be considered in restrictive lung disease, while in obstructive lung disease it is important to select a low frequency and a low I:E ratio. The ventilator settings should then be adjusted, trying to gently move the patient towards the optimal targets.

In very severe restrictive lung disease, BIPAP ventilation can be useful. BIPAP may allow maintenance of spontaneous respiratory activity, while supporting oxygenation with high but safe pressure levels, prolonged duration of the upper positive pressure phase and even inverse ratio between the upper and lower pressure phases.

Oxygenation is optimised by finding the most appropriate combination of FiO_2 and the various interventions aimed at achieving alveolar recruitment. PEEP normally plays a major role, but we must not forget that several aspects of the management of ventilation may favourably affect oxygenation.

De-escalation and weaning

De-escalation is a process that is started as soon as the patient's respiratory state begins to improve and there is consensus (see Boles JM below) that consideration of de-escalation (and weaning), from the time of initiation of ventilation, is useful.

This and other identified, key aspects of weaning/ de-escalation are well addressed in the consensus publication referenced below.

De-escalation involves adjustments to FiO_2, PEEP, and mechanical support. De-escalation can be started with any ventilation mode, and normally it is continued with PSV, by stepwise reductions in FiO_2, PEEP and pressure-support. Depending on the evolution of the underlying disease, de-escalation may be short (hours) or take a long time (days or even several weeks), and may be interrupted by periods of no progress or re-escalation, when the patient's condition deteriorates.

Link to ESICM Flash Conference: Martin Tobin, Maywood. Prediction of difficult weaning, Vienna, 2009.

Weaning patients from mechanical ventilation is not really a matter of ventilation modes and techniques. Rather, it is based on good clinical practice and constant attention to a timely de-escalation of the different components of ventilatory support, as soon as the patient's condition improves

Boles JM, Bion J, Connors A, Herridge M, Marsh B, Melot C, et al. Weaning from mechanical ventilation. Eur Respir J 2007; 29: 1033–1056. PMID 17470624

In patients with severe lung injury or left ventricular failure, de-escalation of positive pressure, and of PEEP in particular, should be performed particularly carefully and slowly. PEEP de-escalation should be based not only on frequent blood gases, but also on lung mechanics and imaging confirming a real improvement in lung function. When PEEP de-escalation is too fast, oxygenation may dramatically worsen, and recovery may be slow.

Weaning is sometimes confused with de-escalation. It is the final step in de-escalation, involving the patient's complete and lasting freedom from mechanical support and removal of the artificial airway.

Successful weaning depends on a major improvement in lung function and resolution of critical illness, although usually it can be successfully performed before recovery is complete. Several indices have been proposed as predictors of successful weaning, but no index or combination of indices is 100% reliable for predicting either successful or unsuccessful weaning. Successful weaning depends on:

- General and specific care of the patient, leading to the resolution of the indications for mechanical ventilation, and
- A determined approach to de-escalation with a continuous effort to reduce the mechanical support as soon, and as much, as possible

The early measurement of weaning predictors and daily protocolized weaning trials may be useful in the management of weaning. In particular a protocol that pairs spontaneous awakening with spontaneous breathing trials can improve the outcome of mechanically ventilated patients.

Girard TD, Kress JP, Fuchs BD, Thomason JW, Schweickert WD, Pun BT, Taichman DB, Dunn JG, Pohlman AS, Kinniry PA, Jackson JC, Canonico AE, Light RW, Shintani AK, Thompson JL, Gordon SM, Hall JB, Dittus RS, Bernard GR, Ely EW. Efficacy and safety of a paired sedation and ventilator weaning protocol for mechanically ventilated patients in intensive care (Awakening and Breathing Controlled trial): a randomised controlled trial. Lancet. 2008 12;371(9607):126-34

Lellouche F, Mancebo J, Jolliet P, Roeseler J, Schortgen F, Dojat M, Cabello B, Bouadma L, Rodriguez P, Maggiore S, Reynaert M, Mersmann S, Brochard L. A multicenter randomized trial of computer-driven protocolized weaning from mechanical ventilation. Am J Respir Crit Care Med. 2006 15;174:894-900.

In some patients complete weaning is impossible, most often due to failure to recover from the underlying respiratory disease.

In patients receiving mask ventilation, de-escalation involves periods of full spontaneous breathing, with or without CPAP.

In patients with tracheostomy, the last step is normally represented by intermittent ventilation with periods of PSV alternated with periods of spontaneous breathing on CPAP, tracheostomy collar or T-piece. In orally or nasally intubated patients, extubation can be performed directly after a period of PSV at a level of 5 to 8 cmH_2O and a PEEP level of 2 to 5 cmH_2O. If necessary, mechanical support can be continued non-invasively after extubation.

Link to ESICM Flash Conference: Miquel Ferrer, Barcelona. Role of non-invasive ventilation in weaning, Vienna, 2009.

Q. Shortly after extubation, your patient unexpectedly becomes hypoxaemic (PaO_2 54 mmHg [7.2 kPa] with an FiO_2 of 0.6) and dyspnoeic, with hypocapnia, alkalaemia and no sign of airway obstruction. The patient is conscious and co-operative. After clinical assessment, which finds no new pathology, what might be your first choice of intervention?

A. In a conscious patient with refractory hypoxaemia and no difficulty in maintaining alveolar ventilation, CPAP by face mask or helmet should be tried first.

New modes of ventilation like BIPAP and ASV can be used for the entire management of respiratory failure in intubated patients, from initiation of support to weaning

The strategy proposed above is based on several ventilation modes, most of which are conventional. However, single ventilation modes available today are designed for the entire management of complex respiratory failure cases, from initiation to complete weaning. Examples of such modes include:

- Biphasic Positive Airway Pressure (BIPAP). This very open approach to the setting of ventilation parameters allows, in expert hands, safe and effective use in a variety of clinical conditions. The main limits of this mode are the total lack of volumetric control, and the general concept being more difficult to understand than for most of the other modes.
- Advanced breath-to-breath dual-control modes with the capability of automatically switching between full ventilatory support and partial ventilatory support (see Task 2).

3/ UNDERLYING PHYSIOLOGICAL PRINCIPLES GUIDING MECHANICAL VENTILATION

Mechanical ventilators can be used to:

- Control CO_2 elimination
- Improve impaired oxygenation
- Assist ('rest') the respiratory muscles

Mechanical ventilation can be hazardous however as it may have injurious consequences for lung parenchyma and extrapulmonary organs. Accordingly, significant efforts of the critical care, scientific community have been expended to find a lung ventilation strategy to minimise ventilator-associated lung injury (VALI).

NOTE VALI may be caused by delivering excessive airway pressures (barotrauma) or volume (volutrauma); moreover the repetitive opening and closing of lung regions during tidal ventilation may cause shear stresses (atelectrauma); eventually cellular inflammatory response may develop (biotrauma).

At the present time there is wide consensus that tidal volume restriction to 6ml/Kg IBW (ideal body weight) and/or plateau airway pressures limited below 30cmH$_2$O may prevent lung injury. Discussion still exists about the optimal management of positive end-expiratory pressure level and respiratory system recruitment.

Plateau airway pressure is measured at end-inspiration in static conditions of the respiratory system. It may be obtained by performing an end-inspiratory measurement while the patient is sedated and a neuromuscular blocking drug has been administered.

Hinds CJ, Watson JD. Intensive Care: A Concise Textbook. 3rd edition. Saunders Ltd; 2008. ISBN: 978-0-7020259-6-9. pp. 163–166 (Respiratory changes and ventilator associated lung injury); 172–173 (Mechanical ventilation with low tidal volumes); 228–230 (Respiratory support).

The acute respiratory distress syndrome network. Ventilation with Lower Tidal Volumes as Compared with Traditional Tidal Volumes for Acute Lung Injury and the Acute Respiratory Distress Syndrome. NEJM 2000; Vol. 342 No. 18: 1301-1308

Management of CO_2 elimination (alveolar ventilation)

$PaCO_2$ and pH targets

The ideal target for pH is easy to define, corresponding to normal pH in most cases. In some instances a compromise between tidal volume reduction strategy and a lower pH level needs to be achieved.

In general a pH minimum limit of 7.25 is considered safe, but permissive targets for pH and $PaCO_2$ should be individually chosen according to the general state of the patient.

The ideal target for $PaCO_2$ varies, depending on:

- Metabolic side of the acid-base balance, and hence pH
- Usual $PaCO_2$ levels of the patient
- Possible therapeutic need for moderate hypocapnia.

When the standard control of pH and $PaCO_2$ conflicts with mechanical safety criteria, normally priority is given to mechanical safety. If it is considered that the consequent pCO_2/pH is potentially injurious to the specific patient, alternative strategies need consideration

In severe restrictive or obstructive lung disease, aiming at 'normal value' targets for pH and $PaCO_2$ may be incompatible with the mechanical safety of ventilation. In these cases less ambitious targets will likely be required, involving permissive hypercapnia and acidaemia.

Alveolar ventilation and minute ventilation

See Charles Gomersall video on applied respiratory physiology for supplementary information.

Gas exchange between the alveolar spaces and the mixed venous blood flowing through the pulmonary capillaries takes place continuously. The alveolar spaces therefore continuously lose O_2 and collect CO_2. In order to maintain adequate gas exchange, the alveoli are flushed with fresh gas, rich in O_2 and free from CO_2.

This 'alveolar flush' is achieved by the tidal volume (Vt) delivered at a given respiratory frequency (Fr). It is intermittently inhaled and exhaled on top of the functional residual capacity (FRC), the volume of gas remaining in the lung at end expiration. However, only part of the Vt, the alveolar volume (VA) works as alveolar flush. Part of the Vt, the dead space volume (Vd), corresponds to the parts of the respiratory system that are not involved in gas exchange (airways and non-perfused alveoli). Hence, only a proportion of the total minute ventilation (MV = Vt • Fr) is useful for supporting gas exchange. This is the alveolar ventilation (V'A = VA • Fr).

The rate of elimination of CO_2 from the respiratory system is proportional to the V'A. The control of $PaCO_2$, and hence the respiratory control of pH, depends on the balance between the V'A and the metabolic production of CO_2 ($V'CO_2$):

$$PaCO_2 = \frac{k \cdot V'CO_2}{V'A}$$

During mechanical ventilation, we manipulate the V'A to achieve predefined targets for $PaCO_2$ and pH. Since, in clinical practice, we do not know the factor k (that expresses how difficult the CO_2 elimination is) or the $V'CO_2$ of our patients, the manipulation of V'A is necessarily made by repeated attempts, checking the results of any change in settings, in terms of $PaCO_2$, and knowing that an increase in V'A will result in a decrease in $PaCO_2$ and vice versa.

The matter is made more complicated by the fact that we do not directly control the V'A. Rather, we control minute volume (MV) and the way the MV is delivered i.e. the ventilatory pattern defined by Vt, Fr, and I:E ratio.

NOTE It is important to appreciate, for example that reducing apparatus dead space, by e.g. changing from a Heat and Moisture Exchanger (HME) to an active humidifier will increase V'A for the same MV.

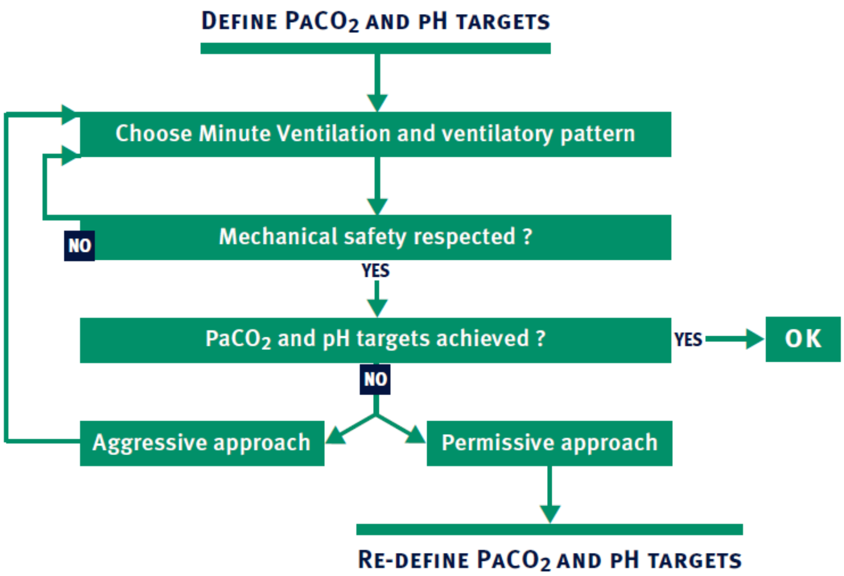
Mechanical safety criteria include:
• Limited tidal volume at 6ml/Kg IBW,
• Limited static end-inspiratory pressure (max plateau pressure at 28-30 cmH2O),
• Limited peak airway pressure,
• Avoiding intrinsic PEEP.

On the one hand the possible choices of ventilatory pattern affect the relationship between MV and V'A: (at constant MV, V'A decreases when Fr increases).

On the other hand the choices are limited by mechanical safety criteria:

• An increase in Vt can be associated with a dangerously high static end-inspiratory pressure (plateau pressure).
• An increase in Vt and/or Fr, and a decrease in I:E ratio can be associated with a dangerous increase in peak airway pressure.
• An increase in Fr and/or I:E can be associated with an undesirable intrinsic PEEP.

In turn, static end-inspiratory pressure, peak airway pressure and intrinsic PEEP depend on respiratory system passive mechanics, namely compliance, resistance and time constants i.e. the product of resistance and compliance.



Basic algorithm for setting mechanical ventilation to control $PaCO_2$ and pH, while maintaining mechanical safety

In adults, a reasonable starting point is an MV setting of 100 ml/kg/min related to the ideal body weight (IBW) of the patient. However, the MV necessary for good control of $PaCO_2$ and pH is often much higher (due to high CO_2 production and impaired lung function), and you will have to choose between:

• An aggressive approach, to be followed as long as the ventilator settings do not conflict with mechanical safety criteria.

• Or a permissive approach involving less ambitious blood gas targets, and in particular accepting a degree of hypercapnia.

Choice of tidal volume and frequency

A given minute ventilation (MV) can be delivered in several possible combinations of Vt and Fr. However, in an individual patient several of the possible combinations may not be very effective, or may even be hazardous. In patients with severe lung disease, selection of the most appropriate Vt and Fr is critical, and should be based on effectiveness and safety.

Minimum effective Vt

When Vt is decreased to a value close to the Vd, then V'A and CO_2 elimination become close to zero, even in the presence of high Fr and maintained MV. If we consider that the in-series Vd (anatomical Vd) is approximately 2.2 ml per kg of IBW, it is not advisable (during conventional convective ventilation) to apply a Vt of less than 4.4 ml/kg, i.e. double the minimum Vd in adult patients.

Maximum safe Vt

The maximum Vt that can be safely delivered is much more difficult to predict: maximal stress (tension developed by lung tissue fibres in response to pressure) and strain (tissue deformation due to volume) can be determined by measuring transpulmonary pressure (i.e. airway pressure minus pleural pressure, AP_L) distending the respiratory system and the functional residual capacity (FRC) of the lung.

In ARDS, a Vt of 6 ml/kg IBW is strongly recommended. However, in the most severe cases of ARDS this low value can still be too high, and the best choice may approach the minimum limit of effective Vt (4.4 ml/kg)

Pleural pressure and FRC determination at the bedside are still not very common in clinical practice. For further reading see:

Chiumello D, Carlesso E, Cadringher P, Caironi P, Valenza F, Polli F, Tallarini F, Cozzi P, Cressoni M, Colombo A, Marini JJ, Gattinoni L. Lung stress and strain during mechanical ventilation for acute respiratory distress syndrome. Am J Resp Crit Care Med 2008; 178: 346-355

At the bedside, plateau pressure (the pressure observed during a relaxed end-inspiratory hold) can be easily measured. A plateau pressure of 25 cmH_2O is always considered safe. A pressure of 30 cmH_20 is probably safe in most cases. Higher values are not recommended.

The static end-inspiratory pressure depends on a number of factors besides the Vt, namely PEEP, intrinsic PEEP and compliance. This means that a relatively high Vt of 12-15 ml/kg is within pressure safety limits when compliance is normal-high and total PEEP is low. On the contrary a Vt as low as 6 ml/kg can produce excessive plateau pressures when the compliance is extremely low and a high PEEP level is applied.

International consensus conference in intensive care medicine. Ventilator associated lung injury in ARDS. American Thoracic Society, European Society of Intensive Care Medicine, Société de Réanimation Langue Française. Intensive Care Med 1999; 25: 1444-1452. PMID 10660857. Full text (pdf)

THINK: Conventionally, we distinguish between lung damage due to high distending pressure (barotrauma) and lung damage due to high lung volume (volutrauma). Think whether this distinction is justified and useful. In particular, reflect on the following points:

- Respiratory physiology tells us that distending pressure and lung volume are just different expressions of the same phenomenon, i.e. respiratory system distension.
- When we reason in terms of pressure, we can evaluate easily and unambiguously the risk of over distension.
- The same evaluation is much more difficult, if we reason in terms of volume.

Maximum acceptable Fr

A low Vt can, to some extent, be compensated by increasing the Fr. However, increasing Fr has an important drawback: the expiratory time (Te) may fall sufficiently to impede complete exhalation to the equilibrium point defined by the applied PEEP. Reaching equilibrium within the end of Te depends on the balance between Te and the respiratory system expiratory time constant (RCe).

RCe corresponds to the product of resistance and compliance, and quantifies the speed of exhalation. With a Te of at least three times the RCe, the equilibrium is at least nearly reached. A Te shorter than twice the RCe generates significant dynamic hyperinflation, and intrinsic PEEP accumulates above the externally applied PEEP. Fortunately most of the patients requiring a low Vt have a low RCe due to reduced compliance, and hence can be safely compensated by increasing Fr. Conversely in asthma/COPD patients, for whom a low Fr is indicated to oppose dynamic hyperinflation, the effect of airways obstruction can be compensated by a relatively high Vt, given that lung compliance is often normal or high.

In severe ARDS compensation for the low Vt by increasing of Fr is usually safe: Exhalation is much faster, due to low compliance combined with nearly normal resistance

In the patient with airway obstruction, Fr should be set low, in order to allow a long Te to avoid dynamic pulmonary hyperinflation

	Depends on	
Minimum Vt	**In-series anatomical Vd**	4.4 ml/kg (IBW)
Maximum Vt	**Static end-inspiratory pressure (plateau pressure)** **Static Vt indexed for IBW**	<25 cmH_2O is safe >30 cmH_2O is potentially hazardous <8 ml/Kg may be safe (it may need to be lower depending on the measured indices of barotrauma above) >8 ml/Kg may be hazardous
Maximum Fr	**Te/RCe**	If >3, PEEPi is absent or irrelevant If <2, relevant PEEPi is generated

Choice of I:E ratio

The normal I:E ratio is between 1:2 and 1:1.5, corresponding to an inspiratory cycle of 33-40%. In obstructed patients, a lower I:E ratio contributes with low Fr to prolong the Te, and hence minimise intrinsic PEEP. In restricted patients with ARDS a higher I:E may improve alveolar recruitment and oxygenation, by increasing the mean pressure applied to the respiratory system. Interestingly, in patients with severe restrictive lung disease, we can even apply a moderately inversed I:E, like 2:1, without generation of relevant intrinsic PEEP, thanks to the low RCe with high exhalation speed, typical of these patients. However, inversed I:E increases the mean intrathoracic pressure and may compromise the circulation.

In the obstructed patient the I:E ratio can be reduced only to a limited extent, because this increases the inspiratory flow and hence the peak airway pressure

NOTE Adjustments to the I:E ratio should be matched with frequency. The choice of both parameters should be guided by the principle that a Te/RCe ratio of at least 3, and never lower than 2, should be achieved.

Try to apply the concepts outlined above with the interactive tool Virtual-MV (Appendix). Start with passive Volume-Controlled Ventilation (VCV). Check the effects of different levels of minute ventilation and selections for Vt, Fr and I:E, while simulating patients with normal lungs, restrictive or obstructive lung disease. Find out the effective and the deleterious settings while trying to prevent:

- Excessive peak airway pressure
- Excessive static end-inspiratory pressure
- Intrinsic PEEP

Q. An ARDS patient with a low compliance (20 ml/cmH_2O) and a normal expiratory resistance (12 cmH_2O/l/s including the circuit) is passively ventilated with PEEP of 12 cmH_2O, Vt of 400 ml and frequency of 22 b/min. If you increase the I:E to 2:1, would you expect significant dynamic hyperinflation, and if so why? How can you check for this?

A. Significant dynamic hyperinflation is not to be expected with an I:E of 2:1, because the expiratory time of 0.9 sec would correspond to more than three times the expected expiratory time constant of 0.24 sec. Actual dynamic hyperinflation can be checked by measuring intrinsic PEEP with an end-expiratory occlusion manoeuvre.

Q. In the case above, knowing that the patient has an IBW of 80 kg, how do you assess and judge the safety of the set Vt of 400 ml?

A. With an IBW of 80 kg and a Vt of 400 ml, the Vt/kg is 5 ml/kg. However, with compliance of 20 ml/cmH_2O, total PEEP of 12 cmH_2O and Vt of 400 ml, the theoretical static end-inspiratory pressure is rather high (32 cmH_2O). If a high plateau pressure is confirmed by an end-inspiratory hold manoeuvre, some further reduction in Vt should be considered.

Management of oxygenation

PaO_2 target

Normoxaemia is the ideal target. In an individual patient, however, the PaO_2 target should be chosen considering the invasiveness and adverse effects of the treatments aimed at improving oxygenation, as well as the general clinical condition of the patient. Although a PaO_2 of 80 mmHg (11 kPa) always remains a desirable goal, the target can be decreased to 60 mmHg (8 kPa), or probably even lower, when hypoxaemia is more refractory to treatment and the risk of ventilation related adverse effect is higher.

Normoxaemia is usually quoted at PaO2 100mmHg (13.5kPa) but this reference point falls progressively with age

Impaired oxygenation is the main problem in lung failure; it may be a consequence of six possible mechanisms:

- Low FiO_2, due for example to altitude
- Hypoventilation, especially when breathing low FiO_2
- Impaired pulmonary diffusion capacity (rarely a cause of hypoxaemia)
- Ventilation-perfusion (V/Q) mismatch
- Shunt, due to perfusion of non-ventilated lung regions
- Desaturation of mixed venous blood (if combined with shunt or V/Q imbalance).

PaO_2 targets are less flexible than targets for pH and $PaCO_2$

Hinds CJ, Watson JD. Intensive Care: A Concise Textbook. 3rd edition. Saunders Ltd; 2008. ISBN: 978-0-7020259-6-9. pp. 199–202. Oxygen therapy

Fink M P, Abraham E, Vincent J.L and Kochanek P M (editors). Textbook of Critical Care, 5th Edn. Elsevier Saunders, Philadelphia USA; 2005. p 454-457

See also the PACT module on Acute respiratory failure

Inhaled oxygen

Hypoxaemia due to V/Q mismatch can be effectively counteracted by increasing the inspired oxygen fraction (FiO_2). The limit to using high FiO_2 is imposed by oxygen toxicity. In general, we should observe the principle of using the lowest FiO_2 that ensures satisfactory oxygenation. An FiO_2 of 0.6 is considered safe, even when administered for long periods. Higher levels of FiO_2 may be toxic for the lungs, but are sometimes used even for long periods, when clinically necessary to avoid serious hypoxaemia.

> **NOTE** Hypoxic pulmonary vasoconstriction (HPV) increases pulmonary vascular resistance in poorly aerated regions of the lung, thus redirecting pulmonary blood flow to better ventilated regions. HPV can be inhibited if the patient is ventilated with high FiO_2 or if alveolar hypoventilated units are recruited (local increase in P_AO_2).

Alveolar recruitment

See Charles Gomersall video on shunt.

Hypoxaemia due to true intrapulmonary shunt is refractory to high FiO_2. In this instance, in order to improve hypoxaemia, non-ventilated lung regions should be re-opened, i.e. recruited to ventilation.

Depending on the aetiology, recruitment can be achieved with a range of manoeuvres. For instance, bronchial suction is effective in atelectasis due to bronchial plugs. Drainage of pleural effusions or pneumothorax is effective when atelectasis is due to lung compression. Also reduction of increased intra-abdominal pressure may have a beneficial effect on alveolar recruitment and oxygenation. In inhomogeneous, diffusely diseased lung (e.g. ALI/ARDS), alveoli may be poorly ventilated or collapsed but unstable. During mechanical ventilation application of PEEP or an intentional transient large increase in transpulmonary pressure (recruitment manoeuvre, RM) or a prolongation of the inspiratory time may all recruit collapsed regions.

Fink M P, Abraham E, Vincent J.L and Kochanek P M (editors). Textbook of Critical Care, 5th Edn. Elsevier Saunders, Philadelphia USA; 2005. p 499-500.

ANECDOTE: *A young lady with severe ARDS secondary to sepsis, developed a left pneumothorax that was successfully drained. On day six, blood gases and chest X-ray showed substantial improvement. Ventilation was switched from PCV to PSV, and PEEP was decreased from 15 to 12 cmH_2O. The following day she was tachypnoeic, tachycardic and in pain. Oxygenation was poor, while the chest X-ray looked unchanged. The left chest tube was still draining a small amount of air during inspiration. The level of sedation was increased and PEEP was re-escalated to 15 cmH_2O, but these manoeuvres resulted in worsening of haemodynamics and no improvement in blood gases. A CT-scan of the chest was then obtained, showing an anterior pneumothorax causing extensive compression of the left lung, and totally separated from the existing pleural drain. A colleague reminded staff that increasing PEEP is not the only treatment for poor oxygenation in ARDS, is not always the most appropriate response and that therapy needs to be targeted to the specifically identified clinical problem.*

PEEP

PEEP is defined as an elevation of transpulmonary pressures at the end of expiration. PEEP contributes to the re-opening of collapsed alveoli and opposes alveolar collapse thus improving V/Q matching. PEEP increases the functional residual capacity (FRC) and, by increasing the number of alveoli that are open to ventilation, improves lung compliance and oxygenation.

In ALI, ARDS, and cardiogenic pulmonary oedema, oxygenation can be greatly improved by applying a PEEP

The application of PEEP is limited by extrapulmonary and pulmonary adverse effects. Ventilation with PEEP increases the transmural pressure applied to the alveoli, which may contribute to re-opening and stabilising of collapsed alveoli. The application of PEEP can be lung protective, since it prevents 'atelectrauma' caused by cyclic collapse and re-opening of unstable alveoli.

An increase in mean intrathoracic pressures may reduce right ventricular filling thus decreasing cardiac output and worsening oxygenation. When testing PEEP effects it is important to assess the adequacy of volume status of the patient.

For information on the 'open lung theory' see these references:

Lachmann B. Open up the lung and keep the lung open. Intensive Care Med 1992; 18(6): 319-321. PMID 1469157

Rouby JJ, Lu Q, Goldstein I. Selecting the right level of positive end-expiratory pressure in patients with acute respiratory distress syndrome. Am J Respir Crit Care Med 2002; 165(8): 1182-1186. No abstract available. PMID 11956065

Unfortunately the application of PEEP can also over-distend other lung regions, promoting barotrauma (with formation of bullae, pneumothorax, and pneumomediastinum) and biotrauma (diffuse lung injury and possible injury to other organs due to release of inflammatory mediators). Intrathoracic pressure variation due to positive pressure ventilation can also affect cardiovascular function and the distribution of perfusion.

See Charles Gomersall video on heart-lung interaction.