PATIENT-CENTRED ACUTE CARE TRAINING

AN ESICM MULTIDISCIPLINARY DISTANCE LEARNING PROGRAMME FOR INTENSIVE CARE TRAINING

Mechanical ventilation

Skills and techniques

Update 2011

Module Author (Update 2011)

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EUROPEAN SOCIETY OF INTENSIVE CARE MEDICINE

Mechanical ventilation
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Contents

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.

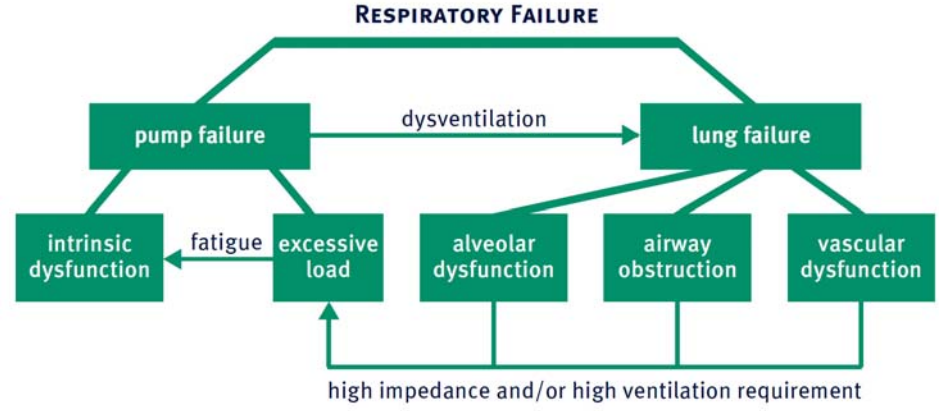
In the online appendix, you will find four original computer-based interactive tools for training in mechanical ventilation. Additional illustrative materials are available online.

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 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.

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

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.

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.

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

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

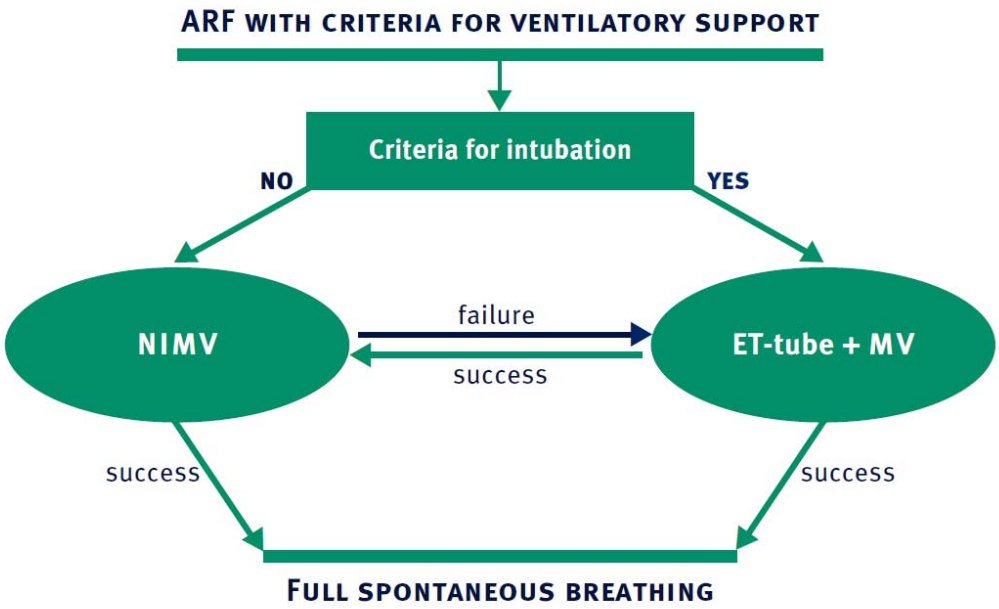
Safe and effective management of mask ventilation requires:
- At least some residual spontaneous breathing (the need for full mechanical support is an absolute contraindication to a non-invasive approach)
- No anticipation that high levels of positive pressure being required
- Ability to tolerate temporary 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

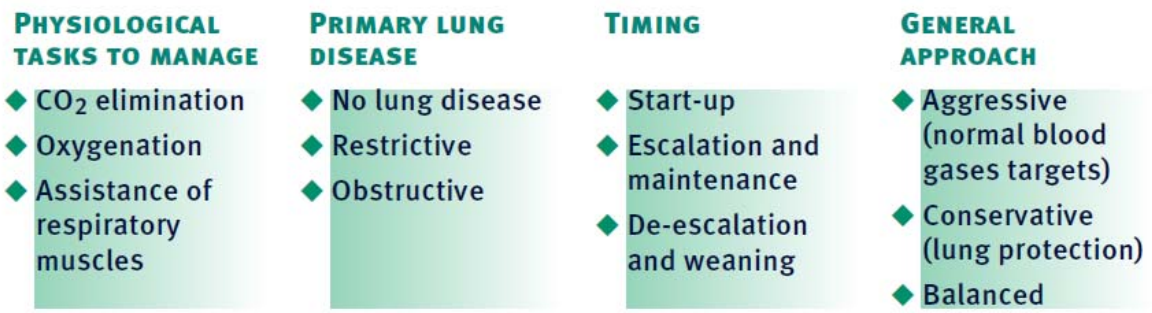
Strategies and timing

The basic concept of initiating mechanical ventilation is not difficult and entails setting the inspired oxygen concentration (FiO_2) 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 CO_2 elimination.

See underlying physiological principles in Task 3 which starts with management of CO_2 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.



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.

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

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

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

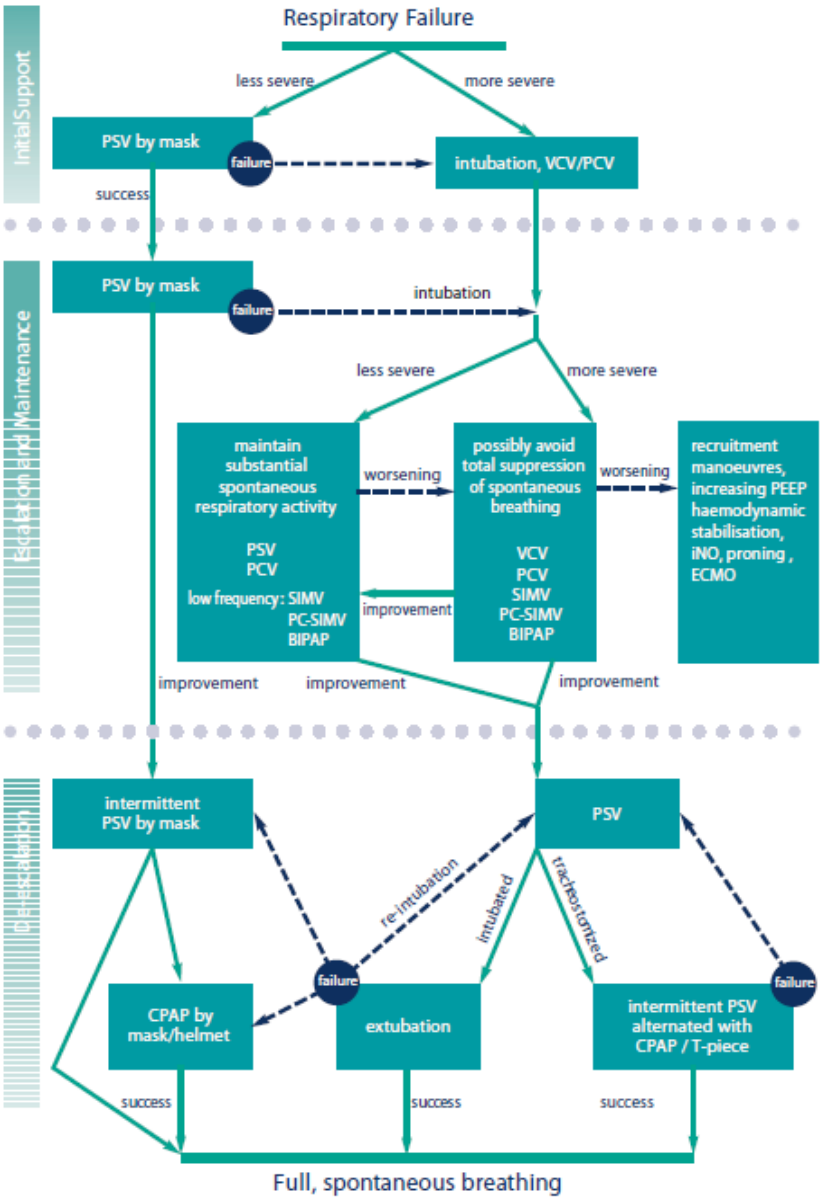
Even in the most severe cases, VCV is not always a necessary choice in the

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

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

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.

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_20 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.

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.

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

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 ($V'CO_2$):

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

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.

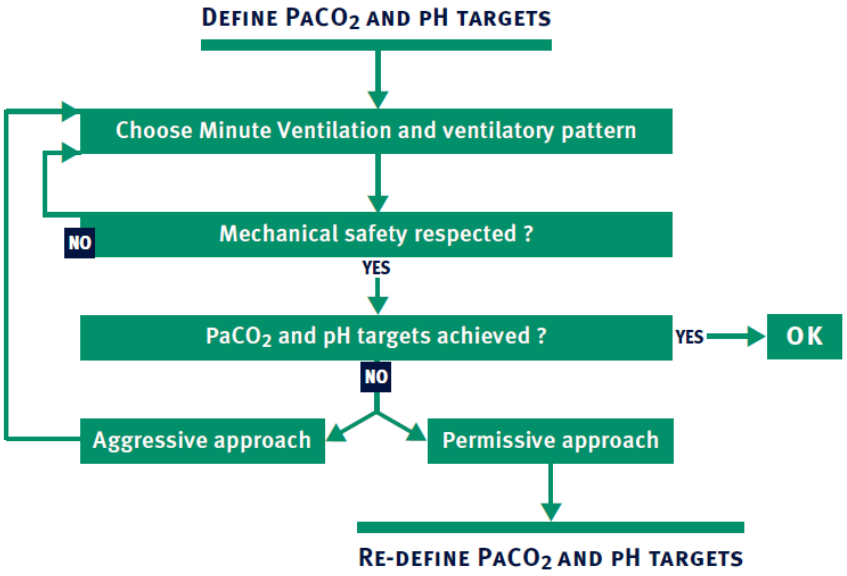
On the one hand the possible choices of ventilatory pattern affect the relationship between MV and V'A: (at constant MV, V'A decreases when Fr increases).

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.

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.*

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_2O 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).

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

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 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)**	<25 cmH_2O is safe >30 cmH_2O is potentially hazardous
	Static Vt indexed for IBW	<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.

Hinds CJ, Watson JD. Intensive Care: A Concise Textbook. 3rd edition. Saunders Ltd; 2008. ISBN: 978-0-7020259-6-9. pp. 171–172. IPPV with PEEP

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 499-501.

A PEEP level of up to 5 cmH_2O has minimal adverse effects, and can be used in most patients. In the majority of ALI-ARDS cases, a PEEP of 10-15 cmH_2O is required. Brain injury with raised intracranial pressure is the most important relative contraindication to this level of PEEP. Attention should be paid to blood volume, haemodynamics, sodium-water retention, and urine output. Tidal volume should be reduced in order to prevent ventilator-associated lung injury (VALI).

Severe lung injury may not result in severe impairment of oxygenation if the pulmonary vessels maintain their capacity to autoregulate (hypoxic pulmonary vasoconstriction)

Very severe ARDS may require a PEEP even higher than 15 cmH_2O. When a high PEEP level is considered, major attention should be paid to monitoring haemodynamics as well as organ perfusion to minimise adverse effects.

***ANECDOTE**: a 31-year-old male with Legionella infection developed ARDS. Worsening hypoxia and respiratory distress necessitated emergency intubation and ICU admission. The ventilator was set to VCV, FiO_2 100% PEEP 8, Tv 6.5ml/Kg IBW, plateau pressure 28 cmH_20. Static compliance of the respiratory system was 24mL/cmH_20, pO_2 70 mmHg (9.3 kPa), pCO_2 44 mmHg (5.8 kPa), pH 7.32. The clinicians decided to set PEEP to 12 cmH_20: plateau pressure and arterial pressure were unchanged while compliance improved to 29 mL/cmH_20. Unexpectedly, pO_2 worsened to 60 mmHg (8.0 kPa). The PEEP trial was repeated after a fluid challenge (500 ml colloids) with a marked improvement in gas exchange (pO_2 123 mmHg (16.3 kPa), pCO_2 42 mmHg (5.5 kPa), pH 7.33).*

For an extensive review of the pulmonary and extrapulmonary adverse effects of PEEP, see also:

Navalesi P, Maggiore SM. Positive end-expiratory pressure. In: Tobin MJ, editor. Principles & Practice of Mechanical Ventilation. 2nd ed. New York: McGraw-Hill; 2006. p. 273-325

Tidal volume reduction to 6ml/Kg and limiting plateau airway pressure to below 30 cmH_20 are widely accepted elements of a 'lung protective strategy'. PEEP titration to prevent inter-tidal alveolar collapse and to keep the lung open throughout the ventilatory cycle is an important aspect of this strategy. PEEP titration to this optimal level is still debated and investigated. A tidal volume reduction strategy can maintain the static end-inspiratory pressure within safe limits, but is likely to involve hypercapnia and even further worsening of hypoxaemia. Moreover the higher the PEEP level, the more likely are both alveolar recruitment and over distension. Three large randomised controlled trials (ALVEOLI, LOV and ExPress studies) comparing low and high PEEP in acute lung injury patients have recently been conducted. Despite a lack of benefit in terms of hospital mortality in an unselected population, higher levels of PEEP may be associated with a lower rate of rescue therapies and lower hospital mortality in the subgroup of severe ARDS patients.

Briel M, Meade M, Mercat A, Brower RG, Talmor D, Walter SD, et al. Higher vs lower positive end-expiratory pressure in patients with acute lung injury and acute respiratory distress syndrome: systematic review and meta-analysis. JAMA 2010; 303: 865–873. PMID 20197533

Brower RG, Lanken PN, MacIntyre N, Matthay MA, Morris A, Ancukiewicz M, et al; National Heart, Lung, and Blood Institute ARDS Clinical Trials Network. Higher versus lower positive end-expiratory pressures in patients with the acute respiratory distress syndrome. N Engl J Med 2004; 351(4): 327–336. PMID 15269312

Meade MO, Cook DJ, Guyatt GH, Slutsky AS, Arabi YM, Cooper DJ, et al; Lung Open Ventilation Study Investigators. Ventilation strategy using low tidal volumes, recruitment maneuvers, and high positive end-expiratory pressure for acute lung injury and acute respiratory distress syndrome: a randomized controlled trial. JAMA 2008; 299(6): 637-645. PMID 18270352

Mercat A, Richard JC, Vielle B, Jaber S, Osman D, Diehl JL, et al; Expiratory Pressure (Express) Study Group. Positive end-expiratory pressure setting in adults with acute lung injury and acute respiratory distress syndrome: a randomized controlled trial.JAMA 2008; 299(6): 646-655. PMID 18270353

Several approaches have been proposed to selecting the most appropriate level of PEEP at the bedside.

In clinical practice selection of the PEEP level is very complex, and should consider benefits and adverse effects, both actual and potential

Gas exchange is the most commonly used guide to the selection of the level of PEEP. PEEP should be increased at least to a level that achieves adequate oxygenation with a safe FiO_2 (≤ 60 %). Besides blood gases, the selection of PEEP can also be based on information about recruitment, assessed by measurement of lung mechanics measurements and/or imaging (standard chest X-ray and CT-scan).

Link to ESICM Flash Conference: Claude Guérin, Lyon. PEEP management in critically ill patients. Peep titration: the pathophysiologic rational, Berlin 2007.

Link to ESICM Flash Conference: Laurent Brochard, Creteil. PEEP management in critically ill patients. High versus low peep strategies in ALI, Berlin 2007.

Studying the respiratory system static pressure-volume relationship at the bedside can provide useful information to guide the setting of both PEEP and tidal volume. In ALI-ARDS the quasi-static P-V curve frequently exhibits a lower and an upper inflection point.

According to the most recent interpretation, the P-V curve corresponds to a curve of recruitment that increases progressively in the lower inflection section, continues steadily in the intermediate linear section, and decreases in the upper inflection section, where over distension becomes prevalent. Since PEEP acts mostly during expiration by preventing alveolar collapse, it is suggested that the expiratory part of the P-V curve may be more informative in terms of PEEP setting. Accordingly, PEEP is titrated by a

decremental PEEP approach. The static end-inspiratory pressure should not exceed the upper inflection point (UIP) or 30 cmH_2O, whichever is lower (except during recruitment manoeuvres).

Other approaches involve analysis of the inspiratory pressure-time curve shape (stress index), measurement of the respiratory system static compliance variations and measurement of end-expiratory lung volume variations (EELV).

Link to ESICM Flash Conference: Hermann Wrigge, Bonn. PEEP management in critically ill patients. Pulmonary imaging and peep titration, Berlin 2007.

The shape of the inspiratory pressure-time curve (linear, curvilinear or concave) can provide information about lung recruitment

A recently published clinical trial introduced the concept of using oesophageal pressure measurement to estimate the transpulmonary pressure as a guide to PEEP selection in ALI/ARDS patients. Despite no difference in outcome between the conventional and the oesophageal pressure guided groups, patients in the latter group had better oxygenation and respiratory system compliance. Though promising, further studies are needed to confirm clinical outcome benefits.

Talmor D, Sarge T, Malhotra A, O'Donnell CR, Ritz R, Lisbon A, et al. Mechanical ventilation guided by esophageal pressure in acute lung injury. N Engl J Med 2008; 359 (20): 2095-2104. PMID 19001507

Increased I:E ratio

Increased I:E ratio can improve recruitment and oxygenation by two mechanisms:

- Increased mean airway pressure, and
- Generating intrinsic PEEP, when the expiratory time is critically shortened.

Both the beneficial and adverse effects of this ventilator-generated intrinsic PEEP are similar to those of externally applied PEEP, although the distribution of intrinsic PEEP may be less homogenous depending on differences in time-constants of different ventilatory units. The major difference between external PEEP and intrinsic PEEP is technical: the former is entirely controlled by the ventilator, while the latter depends on the dynamic balance between ventilator and patient. Intrinsic PEEP can be easily measured in passively ventilated patients, but continuous monitoring is difficult. Therefore, improving recruitment by artificially generating intrinsic PEEP cannot be considered a safe practice.

Additional steps to improve recruitment and oxygenation include:
- *Increased I:E ratio*
- *Spontaneous respiratory activity*
- *Recruitment manoeuvres*
- *Patient positioning*

In ARDS, when oxygenation is severely impaired, a sensible approach includes the setting of an I:E ratio higher than normal, but not so high as to generate intrinsic PEEP. Inverse ratio ventilation (IRV) i.e. ventilation with an I:E ratio

greater than 1:1, requires deep sedation and sometimes even patient (pharmacological) paralysis (unless the Biphasic Positive Airway Pressure - BIPAP mode is used) and periodical verification of the level of intrinsic PEEP.

Maintenance of spontaneous respiratory activity

Hinds CJ, Watson JD. Intensive Care: A Concise Textbook. 3rd edition. Saunders Ltd; 2008. ISBN: 978-0-7020259-6-9. pp. 174–176. Spontaneous modes of respiratory support

Spontaneous respiratory activity:

- Improves ventilation distribution and recruitment in the dependent and basal lung regions, thanks to the tone and pump action of the diaphragm, and
- Reduces the positive intrathoracic pressure associated with mechanical ventilation, thus decreasing the adverse effects of positive pressure on haemodynamics and extrathoracic organs.

Hence, the choice of the ventilation mode and settings should be designed to maintain at least some spontaneous respiratory activity, whenever possible, while avoiding patient discomfort, mechanical stress on the lungs and increased oxygen consumption due to muscular activity. In the most severe ARDS cases options are limited: sedation is necessary, and sometimes muscle relaxants (neuromuscular blocking drugs) cannot be avoided.

Q. In severe ARDS why might Biphasic Positive Airway Pressure (BIPAP) be a sensible choice?

A. BIPAP (or Bi level) allows safe and effective maintenance of spontaneous respiratory activity while exploiting the recruitment effect of an imposed ventilatory pattern with prolonged inspiration and even reversed I:E ratio. A similar ventilator pattern applied by conventional PCV usually requires patient pharmacological paralysis.

Recruitment manoeuvres

The periodic delivery of passive breaths at high pressure and volume may improve alveolar recruitment and oxygenation.

Currently, there is no consensus about the role, safety and best mode for delivering recruitment manoeuvres (RM). Manual bagging can be dangerous in severe lung injury, due to the difficulty of maintaining PEEP and controlling pressure and volume within safe limits. RM can be performed either manually, by temporarily changing the ventilator settings, or automatically, by activating a periodical sigh function. RM can significantly improve oxygenation in the short term with few adverse events (mainly transient and self-limited hypotension and desaturation during the manoeuvre). Clinical outcome benefits of delivering RMs are still unclear so this technique is not recommended as standard treatment in mechanically ventilated patients and should be carefully employed only in selected cases. It is nevertheless useful to recall that recruitment manoeuvres combined with high PEEP could be considered in early severe ARDS patients with life-threatening hypoxemia.

Hinds CJ, Watson JD. Intensive Care: A Concise Textbook. 3rd edition. Saunders Ltd; 2008. ISBN: 978-0-7020259-6-9. pp. 231–232. Body position changes

Patient position

Changes in patient position may improve oxygenation. Periods of ventilation in the lateral position (with the best lung down) are indicated in prevalent one-lung injury, allowing better ventilation and recruitment of the non-dependent lung, and improvement in regional ventilation-perfusion matching.

In diffuse lung injury, periods of ventilation in the prone position may also lead to significant improvement in oxygenation, especially in patients with higher potential for recruitment and marked gravitational distribution of lung densities. Despite a convincing physiological rationale, several recent studies have failed to demonstrate an improvement in overall mortality in acute hypoxemic respiratory failure patients. Although it has been suggested that in the group with the worst hypoxemia (PaO_2/FiO_2 < 100 mmHg or 13.5 kPa) mortality might be improved, prone ventilation is not recommended as standard treatment for ALI/ARDS. See the following reference for further details:

Sud S, Friedrich JO, Taccone P, Polli F, Adhikari NKJ, Latini R, Pesenti A, Guerin C, Mancebo J, Curley MAQ, Fernandez R, Chan M, Beuret P, Voggenreiter G, Sud M, Tognoni G, Gattinoni L. Prone ventilation reduces mortality in patients with acute respiratory failure and severe hypoxemia: systematic review and meta-analysis. Intensive Care Med 2010; 36: 585-599. Full text (pdf)

Extrapulmonary shunt

When impaired oxygenation is caused entirely by extrapulmonary shunt, mechanical ventilation and high FiO_2 will not have any direct benefit on oxygenation. Benefits may only arise indirectly, for instance due to a reduction in oxygen consumption and favourable changes in haemodynamics. In the presence of an intracardiac right-to-left shunt, particular caution should be exercised when applying positive pressure, because any ventilation-induced increase in right heart afterload could increase the shunt and worsen oxygenation.

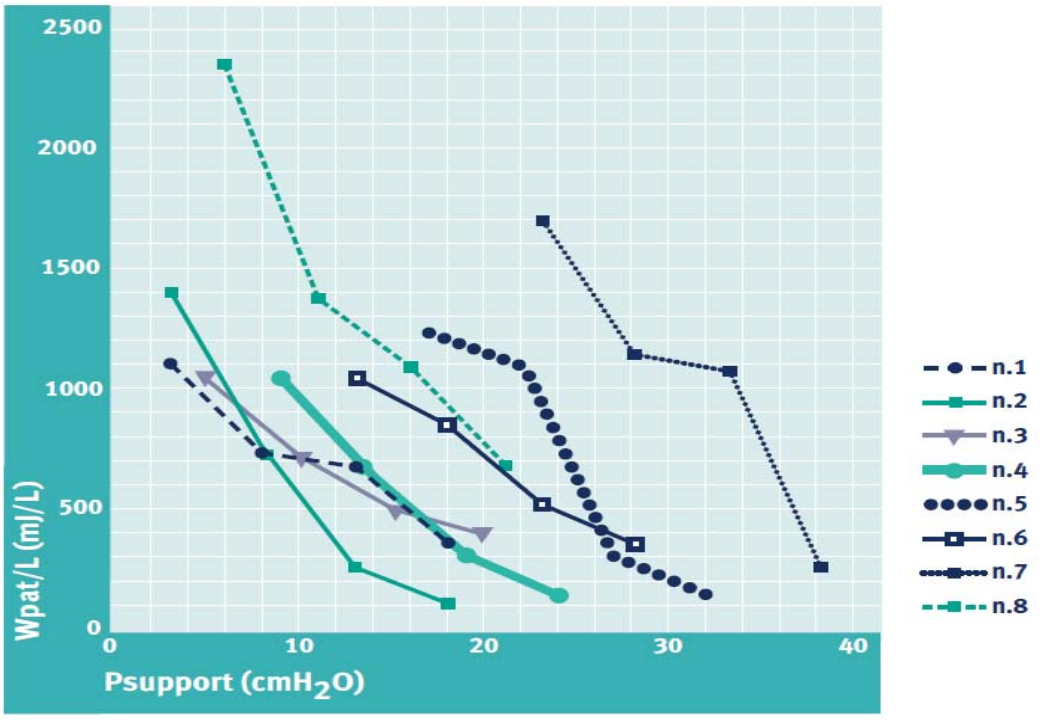
An intracardiac right-to-left shunt should be suspected whenever a paradoxical (oxygenation) response to PEEP is observed

Assist respiratory muscle activity

With appropriate settings, the ventilator can generate an increase in the airway opening positive pressure synchronised with the action of the inspiratory muscles, thus working as an external mechanical assistant of the inspiratory muscles. The physiological response to external assistance is an increase in tidal

volume coupled with a decrease in respiratory drive and activity, resulting in lower respiratory frequency and lower amplitude of the contraction of the inspiratory muscles.

Different modes of assisted mechanical ventilation can be used to support the patient's respiratory effort:
- PSV delivers the designated support (the set level of pressure support) independently from the patient effort.
- Proportional Assist Ventilation (PAV) and Neurally Adjusted Ventilatory Assist (NAVA) adjust, moment to moment, the level of assistance to the patient effort. The greater the patient effort the higher the level of assistance, and vice versa.
- The volume based ventilatory modes, decrease the level of assistance as patient effort increases.



This graph shows how eight different patients with acute respiratory failure responded to stepwise increases in pressure support level. Each one of the patients decreased their spontaneous inspiratory activity, expressed (on the x-axis) as work of breathing.

Q. In the graph above, the same pressure support level of 24 cmH_2O is associated with a totally different spontaneous inspiratory activity in patients 4, 5, 6 and 7, with work of breathing ranging from a below normal value in patient 4 to an extremely high value in patient 7. Give reasons for this.

A. These four patients must have different ventilatory loads, because of differences in compliance and/or airway resistance, and/or intrinsic PEEP, and/or different alveolar ventilation requirements. Ideally, the mechanical support should be individually tailored to each patient.

The patient's response to an increase or decrease in ventilator support is usually quite rapid and a new steady state can be reached within minutes. Setting the

ventilator to assist the respiratory muscles is a process of trial and error requiring:

- Formulation of targets, and
- Assessment of results

Q. A non-sedated neurologically intact patient is assisted with Pressure-Support Ventilation (PSV) and exhibits an irregular breathing pattern: Periods with large Vt are alternated with periods of low Vt and apnoea. What may be the problem?

A. Excessively high levels of pressure support can generate a periodic breathing pattern by lowering the $PaCO_2$ and suppressing the respiratory drive.

Targets are selected on the basis of fundamental principles: Excessive respiratory distress and fatigue should always be avoided, but significant spontaneous activity should be maintained. Total suppression of spontaneous activity should usually be avoided. However, a recent study in severe ARDS a short period of pharmacological muscle paralysis combined fully controlled ventilation was shown to reduce mortality (Papazian reference, below). This general strategy must be adapted to the patient's clinical state, by moving more or less towards full spontaneous breathing according to the phase of ventilation management (de-escalation or escalation, respectively). Maintenance of some degree of patient spontaneous activity may result in recruitment of dependent lung regions, prevention of respiratory muscle atrophy, reduction in the demand for sedative drugs and improvement in haemodynamics. Similarly an increase in oxygen consumption and a reduction in alveolar pressures are to be expected.

Papazian L, Forel JM, Gacouin A, Penot-Ragon C, Perrin G, Loundou A, et al; ACURASYS Study Investigators. Neuromuscular blockers in early acute respiratory distress syndrome. N Engl J Med 2010; 363(12): 1107-1116. PMID 20843245

Link to ESICM Flash Conference: Laurent Papazian, Marseille. Acurasys: neuromuscular blocking agents early in the course of severe ARDS, Vienna 2009.

An excessive level of support may also lead to an increase in patient-ventilator dysynchrony mainly due to ineffective triggering.

Thille AW, Cabello B, Galia F, Lyazidi A, Brochard L. Reduction of patient-ventilator asynchrony by reducing tidal volume during pressure-support ventilation. Intensive Care Med. 2008; 34:1477-86. Full text (pdf)

 With Virtual-MV (Appendix), increase the spontaneous activity of the patient by increasing the Pmus and observe the different results during VCV and PSV.

P0.1 = occlusion pressure at 0.1 sec negative pressure generated by the inspiratory muscles in the first 100 msec of an inspiratory attempt with airway occlusion i.e. in the absence of flow and intrathoracic volume changes.
P0.1 of 1.5 cmH_2O are expected in healthy subject at rest while it may increase to 5 cmH_2O during exercise. Values of P0.1 are affected by the level of sedation, presence of PEEPi and respiratory muscles atrophy.

The assessment of the results is normally based on clinical examination and monitored variables. Ventilatory variables such as Vt, Fr and Fr/Vt are most commonly used. Variables expressing more specifically the degree of muscular activity of the patient, like P0.1, can be of great help for objectively titrating the external mechanical support. As long as adequate alveolar ventilation is maintained and oxygenation is acceptable, blood gases are much less important than mechanical or clinical variables for making decisions about the level of mechanical required.

 In disorders of respiratory control, sepsis and severe hypoxaemia, the physiological response to external mechanical support can be diminished or even lost. In these cases, unless depressant drugs are used, the control of excessive spontaneous respiratory activity is difficult, and the isolated increases in mechanical support usually just result in unnecessary hyperventilation.

Matching the inspiratory flow demand of the patient

In order to effectively unload the inspiratory muscles, the flow demand of the patient must be satisfied during the entire inspiratory period. Since the instantaneous flow demand is difficult to predict and variable, it is more difficult to guarantee effective ventilation with modes based on a pre-set instantaneous flow, such as during VCV and the mandatory breaths of SIMV. Choosing a mode such as PCV, PSV and PC-SIMV is preferable, because the instantaneous inspiratory flow is not limited, and only the inspiratory pressure above PEEP has to be set, i.e. the energy applied by the ventilator to support spontaneous respiratory efforts.

Modern mechanical ventilators may also allow the slope of the inspiratory pressure waveform to be adjusted during PCV, PSV and PC-SIMV.

The optimal assistance of the respiratory muscles involves:
- Appropriate choice of ventilation mode, and
- Fine tuning of ventilator settings

 With Virtual-MV (Appendix), simulate a high spontaneous respiratory activity e.g. a spontaneous frequency of 30 b/min and a peak muscular pressure (Pmus,max) of 20 cmH_2O. With different ventilation modes, try to provide a

substantial level of positive pressure above PEEP throughout inspiration, and find the best settings to avoid intrinsic PEEP.

In **VCV** it may be a challenge: Best results are obtained by increasing the peak flow, i.e. by increasing Vt, decreasing Ti, and using a decelerated flow pattern. If your Ti setting is longer than the patient's Ti, significant intrinsic PEEP may be generated.

In **PCV** it is easier: You can adjust only the controls for inspiratory pressure (Pinsp) and Ti.

In **PSV** it is much easier: You can adjust only the Pinsp control, while the ventilator Ti tends to be automatically matched with the patient's respiratory muscle Ti.

Intrinsic PEEP (PEEPi) and role of PEEP

In ALI and ARDS, if PEEP is successful in achieving alveolar recruitment and improving respiratory system compliance, the mechanical ventilatory load decreases, as long as PEEP is not so high as to push tidal ventilation into the upper section of the pressure-volume relationship, where there will be significant over distension and reduction in compliance.


With the interactive tools CurviLin (Appendix), simulate a restrictive lung disease patient with a lower inflection point (LIP) of 10 cmH_2O, an upper inflection point (UIP) of 30 cmH_2O and a best compliance (Crs) of 25 ml/cmH_2O.

With a Vt of 460 ml, progressively increase PEEP starting from zero and check how tidal ventilation moves along the static pressure-volume curve: The effective compliance (Cqs) improves and hence work of breathing decreases, then Cqs worsens again when tidal ventilation moves beyond the UIP.

During assisted ventilation, intrinsic PEEP (PEEPi) is an additional source of impedance that opposes both the inspiratory muscles and the ventilator throughout the entire inspiration. Any ventilator adjustment that decreases PEEPi will improve the effectiveness of mechanical assistance.

During protective ventilation, assessment of intrinsic PEEP is recommended, since the reduction in tidal volume may trigger an increase in the respiratory rate, which could eventually lead to an increased intrinsic PEEP.

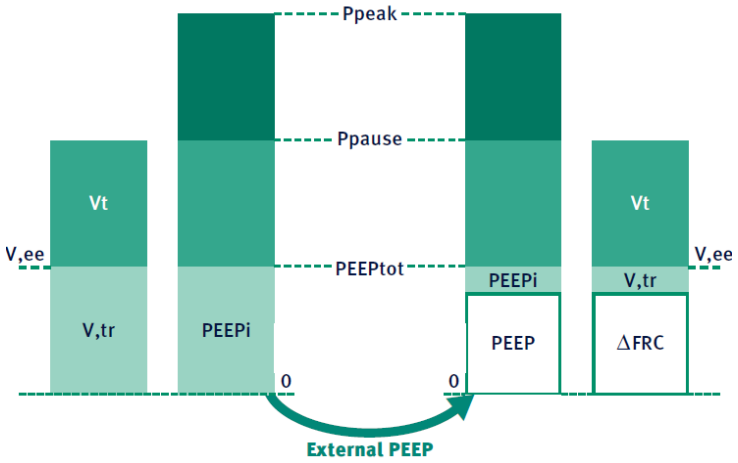
Hough CL, Kallet RH, Ranieri VM, Rubenfeld GD, Luce JM, Hudson LD. Intrinsic positive end-expiratory pressure in Acute Respiratory Distress Syndrome (ARDS) Network subjects. Crit Care Med 2005; 33:527-32

With Virtual-MV (Appendix), you can simulate a COPD patient with Crs of 80 ml/cmH_2O, Rrs of 25 cmH_2O/l/s, a spontaneous frequency of 25 b/min and a Pmus,max of 15 cmH_2O.
If you start PCV with a frequency of 15 b/min, Pinsp of 20 cmH_2O and Ti of 30%, a significant intrinsic PEEP is generated.

Try to decrease the Ti, and check how, with the same patient inspiratory effort and ventilator Pinsp, ventilation becomes more effective: PEEPi decreases, while Vt consequently increases as long as the ventilator Ti is not too short.

In ventilated COPD patients with expiratory bronchial collapse, PEEPi is a common finding. As represented in the schematic drawing below, in this context, moderate levels of external PEEP increase the functional residual capacity (FRC) but at the same time achieve the interesting result of reducing PEEPi and the dynamically trapped volume (V,tr). As a result (up to a point), total PEEP and the end-expiratory lung volume (V,ee) do not increase. Therefore during assisted ventilation of COPD patients, careful adjustment of PEEP can effectively decrease the ventilatory load.

Link to ESICM Flash Conference: Jordi Mancebo, Barcelona. PEEP management in critically ill patients. Peep selection in COPD, Berlin 2007.



A COPD patient with dynamic hyperinflation and air-trapping due to bronchial collapse: Effects of external PEEP on ventilation pressures and lung volumes.

Vt (tidal volume), V,ee (end-expiratory lung volume), V,tr (trapped volume); FRC (functional residual capacity), PEEPi (intrinsic PEEP), PEEPtot (total PEEP)

Check the effects of favourable interaction between PEEP and intrinsic PEEP by the interactive tool B-Collapse (Appendix).

NOTE **Intrinsic PEEP is offset by external PEEP only in patients with expiratory small airway/bronchial collapse.** In other cases of pulmonary hyperinflation, such as severe acute asthma or dynamic hyperinflation primarily due to shortened expiratory times, external PEEP and intrinsic PEEP have largely additive effects, and hence you should be cautious in the use of external PEEP.

With Virtual-MV (Appendix), you can simulate the presence of intrinsic dynamic hyperinflation by increasing the respiratory rate and therefore reducing the expiratory time. Such increased total PEEP (imposed PEEP plus intrinsic PEEP) may be minimised by reducing the imposed PEEP level.

4/ GENERAL WORKING PRINCIPLES OF POSITIVE PRESSURE VENTILATORS

Mechanical ventilators are comprised of four main elements:

- An internal source of pressurised gas including a blender for air and oxygen
- An inspiratory valve, expiratory valve and ventilator circuit
- A control system, including control panel, monitoring and alarms
- A system for ventilator-patient synchronisation

For details about the technology of ventilatory equipment, consult:

Cairo JM, Pilbeam SP. McPherson's respiratory care equipment. 6th ed. St Louis: Mosby International; 1999. ISBN 0815121482

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

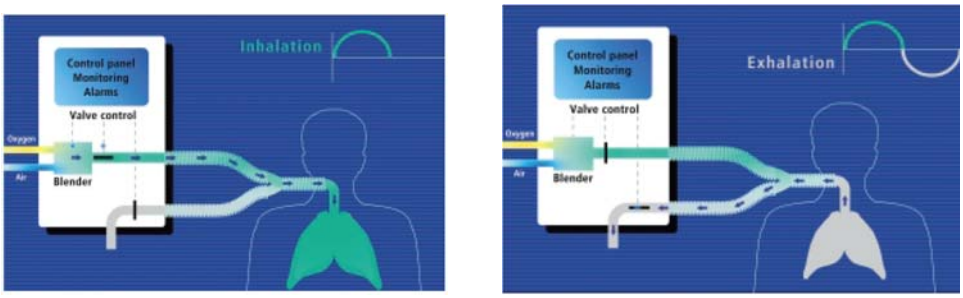
Internal source of pressurised gas

Most commonly, the internal source of pressurised gas makes use of air and oxygen from the hospital central-supply. The two gases are mixed by a blender to achieve the desired oxygen concentration (FiO_2), while the gas pressure (from the 'wall') is appropriately reduced (by pressure-reducing valves). The internal source is thus ready for gas delivery to the patient.

Inspiratory valve, expiratory valve and ventilator circuit

These elements represent the actuators of positive pressure ventilation. In the basic operating mode, the two valves work with a synchronised but opposite phase: while one valve is open, the other is closed and vice versa. The ventilator circuit consists of large bore tubes, mostly external to the ventilator, and includes an inspiratory limb, an expiratory limb, and a connecting Y-piece. Between the Y-piece and the patient interface (tracheal tube or mask), a short flexible tube ('catheter mount') is normally used, representing a common airway through which the gas passes to the patient during inspiration and returns during exhalation.

During the inspiratory phase, the inspiratory valve opens while the expiratory valve is closed, thus generating an increase in the positive pressure applied to the airway opening and delivering gas to the respiratory system.



Ventilator operating valves for inspiration and exhalation

During expiration, the inspiratory valve closes while the expiratory valve opens thus allowing passive exhalation driven by the elastic recoil of the respiratory system.

The degree of opening of both valves is accurately and instantaneously controlled by the control system, to modulate the pressure and flow delivered during inspiration as well as any positive pressure maintained during exhalation.

Control system

This controls the internal source of pressurised gas (including the blender) and of the two main valves, inspiratory and expiratory. The control system works on the basis of the user settings entered by means of the control panel, and on the information continuously provided by sensors for pressure and gas flow. The control system also provides information to the user by means of the monitoring system and alarms.

Synchronisation

Intensive care ventilators are equipped with technology designed to detect both the start and end of the patient's inspiratory efforts, and to synchronise the ventilator inspiratory phase with the patient's inspiratory effort. The synchronisation system is based on sensors for airway pressure and flow, positioned in the ventilator circuit according to the technical choices of ventilator manufacturers, and resulting in an:

- Inspiratory trigger, pressure-based or flow-based, to initiate the ventilator inspiratory phase and
- Expiratory trigger, to stop the inspiratory phase and cycle to the expiratory phase

Ventilatory cycle management

Baseline pressure (PEEP/CPAP)

During exhalation until a new inspiratory cycle is started, the ventilator controls a baseline pressure, which can be set at zero or at positive levels commonly called positive end-expiratory pressure/Continuous Positive Airway Pressure (PEEP/CPAP). In this mode PEEP works on the respiratory system to artificially increase the functional residual capacity (FRC).

The ventilator controls a baseline pressure (zero or positive) at the airway opening

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

In modern intensive care ventilators, PEEP is achieved by appropriate and instantaneous control of the degree of opening of the expiratory valve. In several machines a base-flow of gas runs through the ventilator circuit during the expiratory phase of the cycle, compensating for minor leaks and contributing to effective control of PEEP.

Further pressure applied on top of the baseline promotes inspiration, while the return to the baseline allows passive exhalation

Phases of the ventilatory cycle

Mechanical ventilation breaths can be considered as:
- Controlled
- Assist-controlled
- Assisted-spontaneous
- (Fully) spontaneous breaths.

according to the settings selected for:

- Breath initiation
- Inspiration
- Cycling to exhalation

BREATH TYPE	BREATH INITIATION	INSPIRATION	CYCLING TO EXHALATION
Controlled	Machine *Frequency setting*	Flow/volume control or pressure control	Machine *Inspiratory time setting*
Assist-control	Patient *Inspiratory trigger* Machine *Frequency setting*	Flow/volume control or pressure control	Machine *Inspiratory time setting*
Assisted-spontaneous	Patient *Inspiratory trigger*	Pressure control *Pressure support setting*	Patient *Expiratory trigger*
Spontaneous	Patient *Inspiratory trigger*	Pressure control *PEEP/CPAP setting*	Patient *Expiratory trigger*

Classification of mechanical ventilation breaths

Breath initiation: Machine vs patient

Machine-initiation means that the breath is initiated at a pre-set time, according to the setting for respiratory frequency. Machine-initiation can take place only in the modes that include a control for frequency i.e. in controlled and assist-control breaths – see below.

Patient-initiation means that the breath is initiated by the patient's inspiratory effort, by means of a pressure or flow-based trigger. Patient-initiation can take place during assist-control, assisted-spontaneous and spontaneous breathing modes.

Q. How can machine-initiation and patient-initiation coexist in assist-control breaths?

A. In assist-control breaths, when the patient's respiratory rate overcomes the frequency set in the machine, breaths are patient-initiated i.e. assisted. Should the patient stop breathing, the machine takes over by delivering controlled breaths according to the set frequency.

With pressure-trigger, the ventilator monitors the airway pressure during the expiratory phase. When the patient contracts their inspiratory muscles, the airway opening pressure drops below the baseline. When this drop reaches a pressure threshold defined by the trigger sensitivity control, the machine responds by initiating the inspiratory phase of the respiratory cycle.

With flow-trigger, the ventilator senses the gas flow. When the patient contracts their inspiratory muscles, the airflow reverses from end-expiratory or zero to inspiratory. When the inspiratory flow generated by the patient reaches the trigger sensitivity threshold, the machine responds by initiating inspiration.

Flow-triggering is always associated with an expiratory base-flow in the ventilator circuit. The base-flow allows the patient to generate the initial inspiratory flow and reach the flow-trigger threshold

With the interactive tool EasyTrigger (Appendix) you can explore the differences between pressure-trigger and flow-trigger. Note how the delay in the ventilator response depends on the ventilator settings for trigger type and sensitivity, but also on

- Level of spontaneous activity
- Dynamic pulmonary hyperinflation

Q. What is the main principle for setting the trigger sensitivity whether pressure or flow-triggered?

A. Trigger sensitivity should be set at a high sensitivity (i.e. a low number), to reduce the ventilator response delay. This improves patient–ventilator interaction and patient comfort.
A very high sensitivity can generate ventilator self-cycling (auto-triggering).

Inspiration: Volume-control *vs* pressure-control

Conventional **volume-controlled inspiration** is based on control of the instantaneous inspiratory flow, delivered for the set inspiratory time and according to the set flow pattern, to achieve the set tidal volume.

In conventional volume-control, the constant (square wave) and the decelerated inspiratory flow pattern are the most common choices

At any instant of a volume-controlled inspiration, the pressure developed at the airway opening depends on the balance between the:

- Inspiratory flow and volume change produced by the ventilator, and
Passive impedance of the respiratory system (resistance, compliance and intrinsic PEEP of the lungs) and the patient's chest wall

During **pressure-controlled inspiration**, the ventilator applies a positive pressure above the baseline pressure. This pressure is theoretically a square wave: The ventilator generates a rapid increase in pressure to the user-set level, and then maintains that pressure throughout inspiration.

*The inspiratory pressure controlled by the ventilator above the baseline is referred to as **Control Pressure** in controlled and assist/control breaths, **Pressure Support** in assisted-spontaneous breaths, and **CPAP** in fully spontaneous breaths*

At any instant of a pressure-controlled inspiration, the inspiratory flow and volume depend on the balance between the:

- Inspiratory pressure applied by the ventilator, and
- Passive impedance of the respiratory system and the patient's muscular activity.

Although we can set volume-control and pressure-control to deliver exactly the same tidal volume, the effects of these two approaches are very different when the patient's respiratory mechanics or respiratory muscle activity change.

- Only volume-control can guarantee a minimum set level of minute ventilation.
- Only pressure-control can guarantee that the pressure applied by the ventilator will be limited to the pre-set value.
- Only pressure-control can guarantee a good matching between the flow delivered by the ventilator and the flow demanded by the patient.

Inspiration cannot be managed by simultaneous instantaneous control of volume and pressure. However, modern ventilators can control tidal volume during pressure-controlled ventilation by applying closed-loop control techniques. This kind of management, referred to as **dual-control inspiration**, achieves tidal volume-control:

Dual-control inspiration combines the advantages of pressure-control and volume-control

Either within each breath, by automatically switching from pressure-control to flow-control when the system finds that the set tidal volume cannot be obtained by pure pressure-control.
Or by pure pressure-control, with automatic breath-to-breath adjustments of the inspiratory pressure applied by the ventilator.


With the simulator Virtual-MV (Appendix) you can explore the differences between volume-control and pressure-control, by alternating the view between Volume-Controlled Ventilation (VCV) and Pressure-Controlled Ventilation (PCV). Start with the simulation of a passive patient with normal mechanics, then simulate bronchial obstruction or pulmonary oedema, and finally simulate increasing spontaneous respiratory activity.

Q. What is the inspiratory flow pattern during PCV in a passive patient?

A. During passive PCV the flow pattern is non-linearly decelerating. The instantaneous flow is not pre-set. Deceleration is faster when compliance is lower, and slower when resistance is higher.

Cycling to exhalation: Machine vs patient

When controlled by the machine, cycling is normally time-based. Time-cycling means that the ventilator switches to exhalation as soon as the set inspiratory time (Ti) has elapsed. According to the philosophy of the user interface of the ventilator, the Ti can be set by different means, i.e. directly as a time in seconds, or indirectly depending on the combination of different settings, such as:

- Duty cycle (Ti%) or the Inspiration:Expiration ratio (I:E) combined with frequency.
- Inspiratory peak flow combined with tidal volume and flow pattern.

Time-cycling is used in controlled and assist-control breaths. A limitation of time-cycling is that the machine is unable to adapt to changes in the spontaneous ventilatory pattern: An increase in spontaneous frequency may result in critical shortening of the expiratory time, and hence in dynamic pulmonary hyperinflation, unless the ventilator Ti setting is manually adjusted to match the patient's own Ti.

Patient-controlled cycling is used in assisted-spontaneous and fully spontaneous breaths. Patient-cycling is based on an expiratory trigger that allows the automatic adaptation of the ventilator Ti to the patient's own Ti. The expiratory trigger is based on instantaneous measurement of the inspiratory flow. When the inspiratory flow falls below a threshold value, the ventilator considers that the inspiratory effort should be close to the end, and hence cycles to exhalation. Depending on the ventilator, the threshold (Expiratory Trigger Sensitivity - ETS) can be either an absolute flow value, or a given percentage of the peak inspiratory flow (usually 25%).

Using ETS control you can influence the I:E ratio of assisted-spontaneous or fully spontaneous breaths, while the patient maintains control of cycling to exhalation

In most cases the ETS is fixed at a ventilator-specific value depending on the ventilator design. In a few ventilators a control is available for setting ETS. If we change the sensitivity, for instance by increasing ETS to 50%, or decreasing ETS to 5%, we can respectively advance or delay cycling to exhalation.

 With Virtual-MV you can explore the differences between machine-cycling and patient-cycling. Simulate a patient actively breathing at a frequency of 30 b/min, and then alternate the view between PCV (set with frequency 16 b/min and Ti 33%) and Pressure-Support Ventilation (PSV).

Q. Consider a patient with a large increase in spontaneous breathing rate, for instance due to sepsis. Intrinsic PEEP will probably result with PCV (machine-cycling), but is less likely with PSV (patient-cycling). Why?

A. With machine-cycling, the Ti is pre-set, and hence an increase in the patient's frequency results in significant shortening of the expiratory time. With patient-cycling, the I:E ratio is automatically adapted to the patient's own respiratory cycle, and hence the shortening in expiratory time is less marked.

Exhalation

When cycling to exhalation, the ventilator rapidly drops the airway pressure to the baseline level, which is maintained until the next breath starts. When the next cycle is a controlled breath, the duration of the expiratory phase depends on the ventilator settings for frequency and Ti. Otherwise, the duration depends on the patient. For more details on ventilatory cycle management, consult:

During exhalation the ventilator always works as a pressure controller

Cairo JM, Pilbeam SP. McPherson's respiratory care equipment. 8th ed. St Louis: Mosby International; 2008. pp. 365–387.

Ventilation modes

The ventilation mode represents a specific operating logic (or software program) for the mechanical ventilator, based on one or more approaches to respiratory cycle management. When different forms of management are possible, these can be alternative, alternated in different periods, or even superimposed in the same period.

A modern classification of ventilation modes should distinguish between:

- Conventional primary modes
- Dual-control modes
- Biphasic pressure modes
- Patients effort driven modes

For more on ventilatory support, see:

Ventilatory support; indications. In: Waldmann C, Soni N, Rhodes A, editors. Oxford Desk Reference: Critical Care. Oxford: Oxford University Press; 2008. ISBN 13: 9780199229581. p. 6

Conventional primary modes

This category includes all the classic, conventional ventilation modes:

- **Volume-Controlled Ventilation (VCV)**. In this mode the tidal volume (Vt) is pre-set. Breaths are either controlled or assist-controlled, depending on the lack or presence of spontaneous inspiratory activity, and on whether or not the inspiratory trigger is active. Inspiration takes place according to the principle of the instantaneous control of flow. When the inspiratory trigger is active, VCV is known as Assist-Control Ventilation (ACV).
- **Pressure-Controlled Ventilation (PCV)**. In this mode the tidal volume is delivered by a pre-set pressure. Breaths are either controlled or assist-controlled, depending on the lack or presence of spontaneous inspiratory activity, and on whether or not the inspiratory trigger is active.
- **Pressure-Support Ventilation (PSV)**. In this mode spontaneous breaths are assisted by a pre-set pressure.
- **Spontaneous breathing with CPAP**. All breaths are spontaneous, and the inspiratory pressure is ideally equal to the set PEEP level. Technically, when applied with a mechanical ventilator, spontaneous breathing with CPAP is identical to PSV with a pressure support of zero. Alternatively, this mode can be applied with a freestanding continuous-flow circuit, independent from the ventilator ('wall CPAP').
- **Synchronised Intermittent Mandatory Ventilation (SIMV)**. This mode alternates assist-control volumetric inflations (delivered according to a user-set mandatory frequency) and breaths that can be either assisted-spontaneous (when pressure support is set above zero) or fully spontaneous (when pressure support is set to zero).
- **Pressure-Control SIMV (PC-SIMV)**. This mode is the same as SIMV, except that the mandatory inflations are developed with pressure-control rather than of volume-control.

For more information on freestanding CPAP systems, see the following reference:

Hinds CJ, Watson JD. Intensive Care: A Concise Textbook. 3rd edition. Saunders Ltd; 2008. ISBN: 978-0-7020259-6-9. p. 177, Fig 7.11 CPAP circuit

Cairo JM, Pilbeam SP. McPherson's respiratory care equipment. 8th ed. St Louis: Mosby International; 2008. p. 362-363.

Dual-control modes

This category includes several modes designed for combining the advantages of pressure-control with those of volume-control. The dual-control of inspiratory pressure and tidal volume is made possible by various sophisticated techniques based on closed-loop control applied to mechanical ventilation. In recent years, the number of different dual-control modes offered by mechanical ventilators has overtaken the number of conventional primary modes. The use of a variety of names for these modes by ventilator manufacturers further complicates their classification.

Breath-to-breath dual-control can generate special forms of PCV, PSV, and PC-SIMV, in which the inspiratory pressure is automatically adapted, breath by breath, to achieve a target tidal volume. This principle allows a high degree of freedom for patient-ventilator interaction within each breath, and the simultaneous control of the average tidal volume.

Automatic adaptation to changing needs of the patient is the most recent technical challenge in the field of mechanical ventilation

Q. All breath-to-breath dual-control modes automatically de-escalate the inspiratory pressure when patients increase their spontaneous inspiratory activity. Provide an example of an advantage and a disadvantage of this feature.

A. Automatic de-escalation may result in successful automatic weaning or lead to periods of inadequate assistance of respiratory muscles, depending on the needs of the individual patient for ventilatory support.

Adaptive Support Ventilation (ASV) is a special form of breath-to-breath dual-control ventilation, designed for adaptation of the ventilator to the changes in both passive and active respiratory mechanics of the patient. ASV guarantees a minimum minute ventilation by working on a dual-control PC-SIMV basis. The ASV design allows a non-conventional, simplified user interface, with settings for minimum minute ventilation and patient's ideal body weight (IBW), and no user-setting for mandatory frequency, tidal volume, inspiratory time and pressure support.
All these parameters are automatically adapted on the basis of minimum ventilation setting, the patient's theoretical deadspace, and monitored variables, as well as lung protective, and best energetics criteria. Different models of ventilators have different modes of dual-control ventilation. You should consult the user's manual of your ventilator and see the following reference for information on dual-control modes and ASV.

Iotti G (ed). Closed-loop control mechanical ventilation. Respir Care Clin N Am 2001, 7 (3): 397-408 and 425-440

Biphasic pressure modes

In these modes, the ventilator controls only pressure, which moves between a lower and an upper level, at a given user-set frequency and duty cycle. If the patient is making spontaneous respiratory efforts these are freely superimposed on the variable pressure level, independently from the phase of the ventilator cycle. Depending on the range of frequency of the change from high to low pressure, the biphasic pressure modes can be classified as different forms of **Biphasic Positive Airway Pressure (BIPAP)** and different forms of **High-Frequency Ventilation (HFV).**

With BIPAP, the ventilator applies a dual CPAP level, with a lower and an upper PEEP set by the user. The periodical time-cycling between the two CPAP levels is one source of alveolar ventilation. The second source of alveolar ventilation is based on the spontaneous breaths, superimposed by the patient on the biphasic cycle whenever s/he wants. Contrary to any other ventilation mode, the patient is allowed to exhale even during the high pressure phase delivered by the ventilator, i.e. during the pseudo-inspiratory phase of the biphasic cycle.

The BIPAP mode should not be confused with the BiPAP® ventilator, an apparatus primarily designed for non-invasive pressure support ventilation

Typical BIPAP settings provide a relatively low ventilator frequency, while the patient breathes at a higher rate on top of both the lower and upper CPAP level.

Variants of BIPAP also include:

- Pressure support for assistance of the spontaneous breaths, and
- Patient synchronisation capability for cycling between the two CPAP levels.

Airway Pressure Release Ventilation (APRV) is similar in concept to BIPAP, with particular settings: The patient is maintained at a fairly high CPAP level that is intermittently released to a much lower CPAP level for a short period of time.

The intermittent release and resumption of the upper CPAP level generates alveolar ventilation that augments the ventilation provided by spontaneous breathing. It aims at improving oxygenation without allowing excess hypercarbia.

The reference gives information about BIPAP and APRV.

Kuhlen R, Guttmann J, Rossaint R, editors. New forms of assisted spontaneous breathing. Munich-Jena, Germany: Urban & Fischer; 2001. p 35-65. ISBN 3926762535

Patient effort driven modes

This category includes new modes of assisted-spontaneous ventilation in which the level of pressure above PEEP generated by the ventilator is proportional to the patient's respiratory effort:

During PAV and NAVA, assistance provided by the ventilator is proportional to patient's demand. If patient's demand increases then ventilator assistance increases proportionally, likewise if patient's demand decreases then ventilator assistance decreases.

Proportional Assist Ventilation (PAV). In this mode, the ventilator generates pressure in proportion to patient-generated flow and volume. The operator sets the percentage of flow-assisted and volume-assisted ventilation. This ventilation mode requires the measurement of respiratory system compliance and resistance.

Neurally Adjusted Ventilatory Assist (NAVA). This mode uses the diaphragmatic electrical activity during spontaneous inspirations to trigger and control the delivery of ventilator assistance. The diaphragm electrical activity is recorded by a multiple-array oesophageal electrode positioned in the lower oesophagus. The pressure applied to the airway by the ventilator is moment-by-moment proportional to the diaphragm electrical activity according to a proportionality factor set by the operator.

Fink MP, Abraham E, Vincent JL, Kochanek PM, editors. Textbook of Critical Care. 5th edition. Elsevier Saunders, Philadelphia, PA; 2005. p. 505

Colombo D, Cammarota G, Bergamaschi V, De Lucia M, Corte FD, Navalesi P. Physiologic response to varying levels of pressure support and neurally adjusted ventilatory assist in patients with acute respiratory failure. Intensive Care Med 2008; 34: 2010–2018. PMID 18629471. Full text (pdf)

Gas conditioning

Hinds CJ, Watson JD. Intensive Care: A Concise Textbook. 3rd edition. Saunders Ltd; 2008. ISBN: 978-0-7020259-6-9. p. 161. Humidification

The inspiratory gas delivered to the patient must be adequately heated and humidified, especially when the upper airway is bypassed by an endotracheal tube or tracheostomy. Gas conditioning can be achieved by means of a:

- Heat and Moisture Exchanger (HME) mounted at the Y-piece, or
- Heated humidifier mounted within the inspiratory tubing.

Passive humidification

The performance, dead space and resistance of HMEs depend on the specific product. High performance is usually associated with high dead

HMEs are otherwise known as passive humidifiers or artificial noses. HMEs work by collecting heat and humidity from the expired gas.

HMEs produce an increase in apparatus dead space and resistance. When an antimicrobial filter is coupled to an HME, a further increase in dead space and resistance usually results. Special small-size HMEs are designed for use in infants and children.

HMEs are simple to use and cheap

Advantages of HMEs include low cost and simplified management of humidification and the ventilator circuit.

The performance of HMEs can be inadequate:

- When tidal volume and minute ventilation are very high
- When there are major air leaks
- In severe hypothermia
- When secretions are blood stained or particularly thick

HMEs are not indicated:

- When secretions are very abundant (due to risk of clogging the filter and need for frequent changes)
- In patients with severe impairment in CO_2 elimination (because of additional dead space)
- When tidal volumes need to be limited as in patients with ARDS (Acute Respiratory Distress Syndrome)

Q. Why do major air leaks contraindicate the use of HMEs?

A. With major air leaks, the volumes exhaled by the patient through the HME are much lower than the inspiratory volumes delivered by the ventilator. Therefore the heat and moisture collected by the HME are insufficient for adequate gas conditioning.

NOTE It is advisable to choose an HME with low dead space and low resistance, especially for unsupported spontaneous breathing.

Active humidification

Heated humidifiers can vary in their level of sophistication. Advanced humidifiers are provided with an automatic filling system, inspiratory tube warming with a heated wire, temperature control at the Y-piece, smart algorithms for humidity control, and alarms.
Active humidification:

- Does not increase the apparatus dead space.
- Does not affect the apparatus expiratory resistance and minimally affects the apparatus inspiratory resistance.
- Increases the compressible volume of the ventilator circuit. Special small-size humidifying chambers are designed for use in children and infants.

NOTE The performance of a good active humidifier is superior to that of the best passive humidifier. Unlike any HME, a good active humidifier can deliver a water saturated gas with a temperature of 37° C, at the patient's upper airway. Although HMEs are widely used, heated humidifiers are advisable at least for the most problematic patients.

The optimal targets for setting an active humidifier have not been clearly established. Full saturation with water vapour at a temperature of 37° C at the airway opening seems a sensible approach. Only the gas temperature, and not the levels of humidity, can be easily monitored and controlled. For additional information on gas conditioning, consult:

The assessment of the effectiveness of humidification is based on clinical observations

Branson RD, Hess DR, Chatburn RL, editors. Respiratory care equipment. 2nd ed. Philadelphia: Lippincott Williams and Wilkins; 2000. p 101. ISBN 0781712009

External circuit

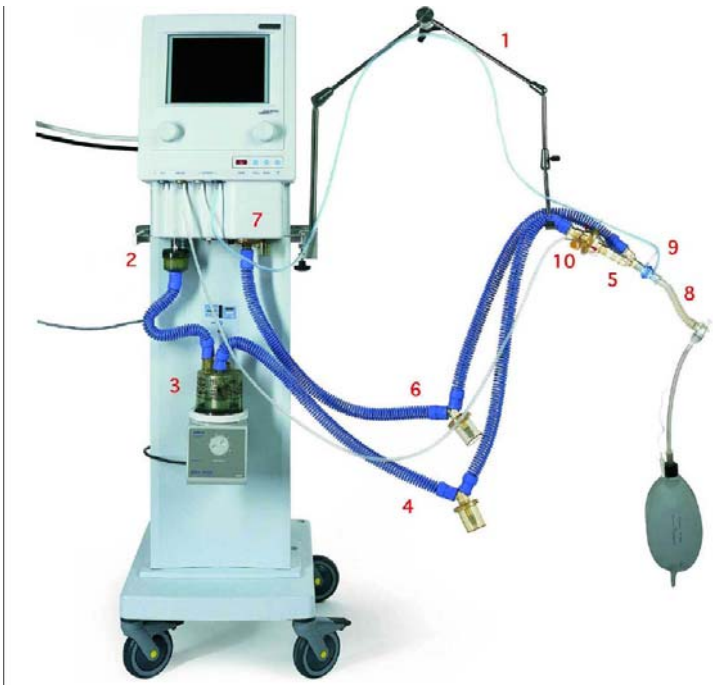
The external circuit is made of flexible tubes and rigid parts.

Parts of the external circuit

The basic design of the external circuit, as represented below in the figure, consists of the inspiratory line, expiratory line, Y-piece and 'catheter mount' for patient connection. In practice, however, the external circuit can vary considerably, depending on the location of the expiratory valve, and on the system used for gas conditioning.

Normally the expiratory valve is located inside the machine, and the expiratory line serves as a return circuit from the Y-piece to the ventilator. Several small-size ventilators use a single tube circuit with the expiratory valve located close to the patient, usually included into a modified Y-piece. The proximal expiratory valve can be either a non-rebreathing system not directly controlled by the ventilator, or a valve pneumatically driven by the ventilator. In the latter case a small bore tube must be used for connecting the valve to the ventilator.

When an active humidifier is used, the circuit requires water traps for collecting condensate, or heated wires in the tubes to avoid condensation. Temperature probes are also necessary, for monitoring the temperature of gas in the airways and for driving the temperature regulator of the advanced humidifiers.



1. Support arm, 2. Inspiratory port with filter, 3. Active humidifier, 4. Inspiratory line with watertrap, 5. Y-piece, 6. Expiratory line with watertrap, 7. Expiratory port, 8. Flexible connector, 9. Proximal flow-pressure sensor, 10. Nebuliser.

A mechanical ventilator for intensive care, with the external circuit

Photo: GALILEO ventilator, Hamilton Medical, Switzerland

Additional parts of the circuit may include:

- Antibacterial filter at the inspiratory port of the ventilator
- Antibacterial filter at the expiratory port of the ventilator (necessary only on a few ventilators)
- Nebuliser in the inspiratory line
- Sensors or sensor ports for pressure, flow and expired gas (CO_2) analysis

Circuit dead space, compliance and resistance

The size of the circuit must be proportional to the patient's size. The inner diameter of the tubes should be:

- 22 mm for adults
- 15 mm for children and infants
- 10 mm for neonates

When choosing an external circuit, you should consider:

- Artificial dead space; depends on the volume of all the elements interposed between the Y-piece and the patient.
- Circuit compliance; depends on the total circuit volume and elasticity of tubes.
- Circuit resistance; depends on the geometry of the tubes and specific resistance of added elements.

The mechanical features of the circuit can be optimised by removing any unnecessary elements, as well as choosing an appropriate length and size of the tubes, especially for the common airway between the Y-piece and patient

High dead space, compliance and resistance of the circuit are all independent elements that decrease the effectiveness of mechanical support. In all ventilators in which monitoring is not based on proximal sensors, high compliance and high resistance also result in poor estimates of the actual values for most of the monitored variables.

Circuit replacement

Before use, a ventilator must be equipped with a complete external circuit with filter(s). The set can be either new single-use, sterile or clean, or re-usable. Re-usable sets must have been cleaned and decontaminated, after the previous use.

NOTE After assembling and attaching each new circuit, a tightness test must be performed. For some ventilators, manufacturers' instructions also prescribe performing other tests and/or calibration procedures after attaching a new circuit. For information on cleaning and disinfection, see the reference below.

Cairo JM, Pilbeam SP. McPherson's respiratory care equipment. 8th ed. St Louis: Mosby International; 2008.

During long term ventilation, the complete circuit with filters is periodically replaced. An unequivocal circuit replacement policy has not been established. Several prospective, randomised trials have demonstrated that the frequency of ventilator circuit change does not affect the incidence of ventilator-associated pneumonia (VAP).

Torres A, Ewig S, Lode H, Carlet J; European HAP working group. Defining, treating and preventing hospital acquired pneumonia: European perspective. Intensive Care Med 2009; 35:9-29. Full text (pdf)

An HME, if present, and the flexible connector for the tracheal tube, are normally replaced every day, or more frequently when macroscopically contaminated.

Ventilator maintenance

The maintenance of modern ventilators is simple. A user's tasks are normally limited to:

- Replacement of the external circuit
- Cleaning and disinfection of some parts of the internal expiratory line
- Replacement of the oxygen cell when exhausted
- Simple calibration procedures and tests

Preventive maintenance must also be performed by trained technicians according to a specific schedule.

Every ventilator is to be set up and maintained according to specific procedures, well described in the user manual.

Have you ever read the users' manual of your ventilators?

THINK: Different models of ventilators have similarities but also important differences. Never forget that safe and effective application of mechanical ventilation implies knowledge about the specific ventilator you are using. You should read the users' manuals of your ventilators, and consult the manuals when in doubt. Studying the manuals is a necessary step, frequently neglected, to maximally exploit the features of your ventilators. Most likely, you will discover functions you did not even know existed.

Ventilator monitor

Airway pressures and flow are the signals on which the entire monitoring system is based. For more information about monitoring of mechanical ventilation, read the PACT module on Respiratory monitoring.

NOTE Optimal monitoring is achieved when ventilators are equipped with pressure and flow sensors at the airway opening. For reliable results, the ventilator must have been properly calibrated.

Integral capnometry is an option provided only by a few ventilators, because expired gas monitoring is usually included in the bedside vital signs monitor.

Modern ventilators for intensive care provide extended monitoring functions, including:

- Graphic monitoring of the important mechanical signals from the ventilated respiratory system (airway pressure, flow and volume), presented as real-time curves and pressure-volume or flow-volume loops
- Numeric monitoring from automatic breath analysis
- Alarms and messages regarding patient and ventilator status

Graphic monitoring is a useful function in intensive care

Carefully choose the alarms you activate and the alarm limits. Too many alarms and too strict limits may result in nearly-continuous alarming, thus making your patient anxious while the attention of the nurses to the alarms may eventually decrease.

Some ventilators also provide functions for special measurements of respiratory mechanics, such as:

- Manual end-inspiratory hold for assessment of static end-inspiratory pressure (plateau pressure)

- Manual end-expiratory hold for assessment of intrinsic PEEP and occlusion pressure at 0.1 sec (P0.1)
- Slow inflation manoeuvre for recording the static pressure-volume curve of the respiratory system

Several of the monitoring functions listed above allow the ventilator to be used as a powerful tool for bedside investigation of respiratory system mechanics:

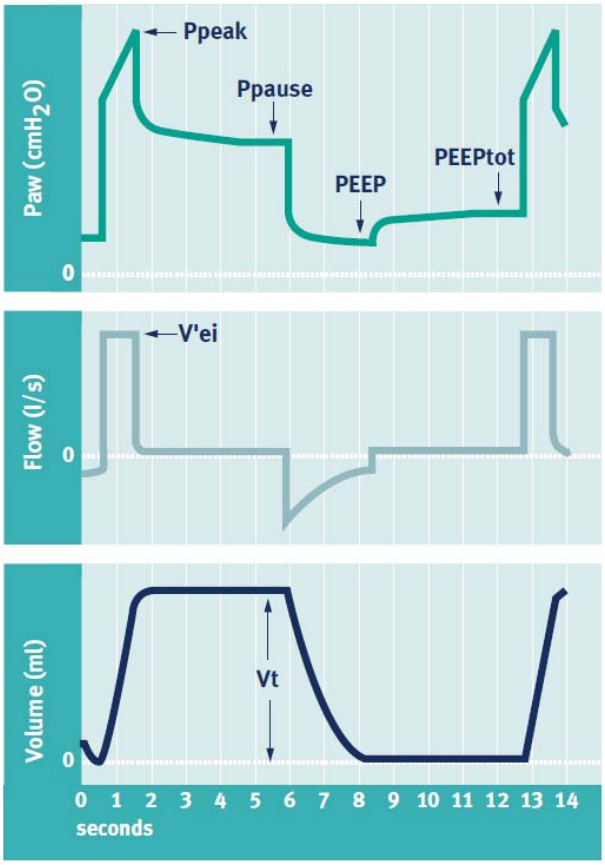
- Passive mechanics: resistance, compliance, time constant, intrinsic PEEP
- Indices of patient spontaneous activity e.g. P0.1
- Spirometric measurements: Volumes, times, frequencies.

Modern mechanical ventilators are powerful tools for bedside investigation of the respiratory function

Data concerning resistance, compliance, time constants and intrinsic PEEP provide a full picture of passive lung mechanics. This is extremely helpful for the safe and effective setting of the ventilator, and allows you to tailor the ventilatory support to the mechanical properties of your patient. Unfortunately, passive mechanics data provided by automatic breath analysis are not always reliable, especially when intrinsic PEEP is present and the measurements are not based on special algorithms such as the least square fitting procedure.

In several instances manual analysis of the curves is necessary. Integrated graphic monitoring with capability to analyse on frozen curves, together with the hold functions, enables easy access to the most important measurements related to passive mechanics: Maximum inspiratory resistance (Ri,max), quasi-static compliance (Cqs), respiratory system time constant (RC) and intrinsic PEEP (PEEPi). The requirements for valid measurements are:

- Patient temporarily relaxed with sedation and neuro-muscular blocking drugs
- Volume-controlled ventilation (VCV) set with a constant inspiratory flow
- An end-inspiratory hold manoeuvre of 4 seconds
- An end-expiratory hold manoeuvre of 4 seconds



In ventilated adults with normal airway resistance, Ri,max is usually 5-8 cmH2O/l/s (including the effect of an unobstructed endotracheal tube of appropriate size)
The clinical interpretation of measurements of respiratory system compliance is easier when referred to the ideal body weight (normal value: 1 –1.2 ml/cmH2O/kg)

Ppeak = peak airway pressure
Ppause = static end-inspiratory pressure
PEEP = positive end-expiratory pressure
PEEPtot = total intrapulmonary PEEP
V'ei = end-inspiratory flow
Vt = tidal volume

$$Ri,max = \frac{Ppeak - Ppause}{V'ei}$$

$$Cqs = \frac{Vt}{Ppause - PEEPtot}$$

RC = Ri,max•Cqs

PEEPi = PEEPtot–PEEP

Frozen curves during passive VCV with constant inspiratory flow and double hold manoeuvre, for manual measurement of passive respiratory system mechanics

	MEASUREMENT TYPE	PATHOPHYSIOLOGICAL MEANING	CLINICAL MEANING
Ppeak	Inspiratory, dynamic	Maximum pressure applied by the ventilator Maximum stress on the large airways	Risk factor of tracheo-bronchial barotrauma
Ppause	End-inspiratory, static	Maximum pressure distending the respiratory system. Maximum stress on the alveoli	It opens collapsed alveoli, but it is also a risk factor of alveolar barotrauma
PEEP	End-expiratory, dynamic	Baseline pressure applied by the ventilator (external PEEP). It artificially increases the end-expiratory lung volume	It keeps the alveoli open, but it may adversely affect haemodynamics
PEEPtot	End-expiratory, static	Minimum pressure distending the respiratory system (intrapulmonary PEEP). It is the true determinant of the end-expiratory lung volume	It keeps the alveoli open, but it may adversely affect haemodynamics

Types of airway pressure

For bedside measurements of respiratory mechanics, consult the reference below.

Iotti GA, Braschi A. Measurements of respiratory mechanics during mechanical ventilation. Rhäzüns, Switzerland: Hamilton Medical Scientific Library; 1999. ISBN 3962186503

Graphic monitoring of expiratory flow provides important qualitative information about airway obstruction and dynamic pulmonary hyperinflation

With the interactive tool Virtual-MV (Appendix), you can simulate different patients and learn to perform manual calculation of passive mechanics data. You can simulate the ventilation conditions necessary for the measurements, activate the hold manoeuvres, read the data necessary for calculation, and calculate the results by the appropriate equations. Check the typical curves and loops associated with obstructive or restrictive lung disease, and with intrinsic PEEP.
After training on Virtual-MV, try to repeat the same measurements and observations on real patients, with the ventilators in your ICU.

CONCLUSION

This module has outlined:

- The nature of respiratory failure.
- How to approach mechanical ventilation and set up a ventilator incorporating a general strategy for ventilatory support which includes non-invasive and invasive approaches.
- An understanding of the underlying physiological principles including an approach to the nature of the underlying respiratory disease and its evolution, the different physiological tasks to be managed and lung-protective ventilation.
- How mechanical ventilators work and how to take full advantage of the information provided by the monitoring system of the ventilator to modify and optimise the ventilator therapy for patient benefit.

APPENDIX

There are four original computer-based interactive tools (Excel files with separate worksheets).

To view the simulators properly (graphics and data together), it is recommended you use a screen resolution of 1024x768 or higher and adapt the zoom accordingly.

Virtual-MV
CurviLin
B-Collapse
EasyTrigger
General instructions (Word File)

Virtual-MV is a general mechanical ventilation simulator. It allows the simulation of volume-controlled ventilation (VCV), pressure-controlled ventilation (PCV) and pressure-support ventilation (PSV), in actively breathing or passive patients.

CurviLin allows the simulation of alveolar recruitment and over distension. CurviLin is based on a curvilinear static pressure-volume curve for the respiratory system, and shows how a passive VCV breath moves along the curve, depending on ventilator settings and patient-ventilator interaction.

B-Collapse allows the simulation of a COPD patient with expiratory bronchial collapse and favourable interaction between external PEEP and intrinsic PEEP.

EasyTrigger is a mechanical ventilation simulator focused on patient-initiation of mechanical breaths. It allows simulation of pressure-trigger and flow-trigger, with selectable trigger sensitivity.

SELF-ASSESSMENT QUESTIONS

EDIC-style Type K

1. Invasive ventilation includes:

A. All positive pressure ventilation
B. Ventilation using an endotracheal tube
C. CPAP using a tracheostomy tube
D. CPAP using mask ventilation

2. Initiation of a breath cycle:

A. Must be started by a patient-initiated inspiratory effort
B. Will always be aborted by a patient-initiated inspiratory effort
C. Machine initiated breath are started on a time cycled basis
D. It is not possible to mix machine initiated and patient-initiated breaths.

3. Usual ways to detect patient-initiated inspiratory efforts include:

A. By a pressure drop in the ventilator circuit
B. By a sudden increase in airway resistance
C. By an inspiratory flow detected in the ventilator circuit
D. By a decrease in airway compliance

4. In volume-controlled ventilation

A. Tidal volume is given according to a pre-set volume target
B. If the inspiratory time is fixed, the peak and mean airway pressure is independent of pulmonary compliance
C. If the minute volume and frequency is set, it is not possible to adjust the tidal volume
D. If tidal volume and minute volume is set, the ventilator frequency must be set between 10 and 20 breaths per minute

5. Which is/are correct statements regarding the inspiratory time (Ti)

A. At the end-inspiratory time, the expiration phase always starts
B. If Ti is set by the Inspiration:Expiration ratio, the Ti is independent of ventilator frequency
C. If Ti is directly set, the expiratory time decreases with increasing ventilator frequency
D. Normal Ti is in the range of 3–4 seconds

6. In biphasic positive airway pressure (BIPAP):

A. The ventilator generates a dual CPAP level with an upper and lower pressure set by the user
B. Patients may freely generate spontaneous breaths in the low pressure phase only
C. Patients are allowed to exhale even during the high pressure phase
D. Airway Pressure Release Ventilation is an extreme concept of BIPAP with a very short low pressure phase

7. When a Heat and Moisture Exchanger (HME) is utilised during IPPV, it

A. Must be mounted in the inspiratory line to be efficient
B. Will increase apparatus resistance
C. Should be avoided/removed in patients with severe impairment of CO_2 elimination
D. Will have no influence on apparatus deadspace

8. Ventilation-induced lung injury may be minimised by the following:

A. Volume-controlled ventilation mode
B. Tidal Volume restriction to 6 ml/kg
C. Limit plateau pressure below 30 cm H_2O
D. Limitation of PEEP below 5 cm/H_2O

9. Regarding the I:E ratio

A. Is normal set between 1:3 and 1:4
B. Should be lowered to decrease intrinsic PEEP
C. Increase I:E ratio may improve alveolar recruitment and oxygenation in ARDS
D. Adjustment of I:E ratio must be matched with respiratory frequency

10. The effects of PEEP on improved oxygenation can be explained by:

A. Re-opening of collapsed alveoli
B. Increased FiO_2
C. Increased functional residual capacity
D. Decreased static compliance

11. Various methods to set optimal PEEP at the bedside include:

A. Arterial PaO_2
B. Analysis of the pressure-volume curve (lower inflection point)
C. Recording of the oesophageal pressure to estimate transpulmonary pressure
D. Measurement of end-expiratory lung volume variations

EDIC-style Type A

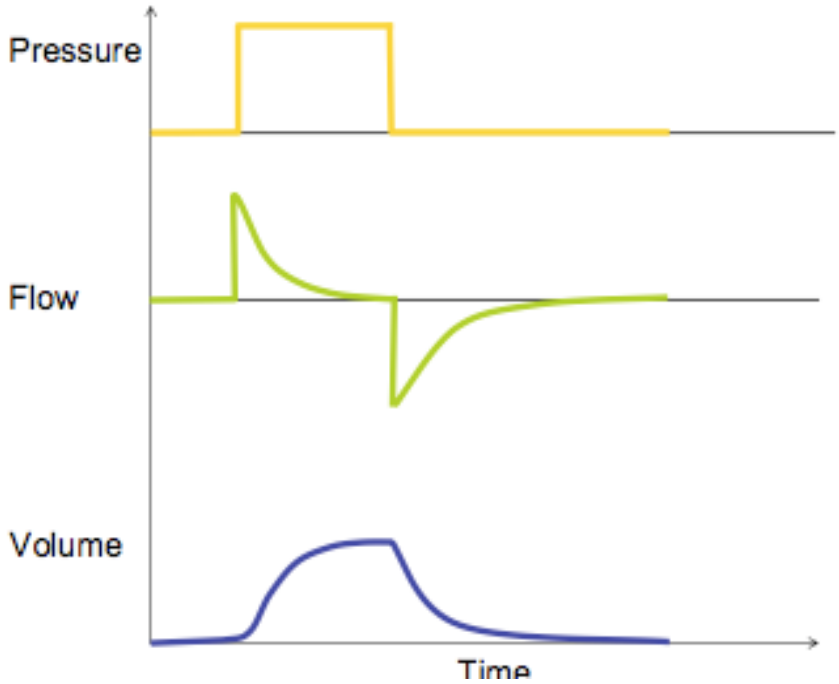
12. Disadvantages of endotracheal intubation includes all of the following, EXCEPT

A. Loss of the protective function of the upper airway
B. Loss of phonation
C. Decreased airway resistance
D. Damage to the subglottic area
E. Need for sedation and or analgesia

13. The figure shows

A. Volume-controlled ventilation
B. Pressure assisted spontaneous breathing
C. Volume-assisted spontaneous breathing
D. Bilevel Positive Airway Pressure Ventilation

E. Pressure-controlled ventilation



14. Effective methods to decrease an elevated PaCO2 may include all of the following EXCEPT:

A. Increase tidal volume
B. Increase frequency
C. Decrease circuit dead space
D. Increase PEEP
E. Increase inspiratory pressure

15. Adverse effects of PEEP include the following EXCEPT:

A. Over distension of normal alveoli
B. Barotrauma
C. Decreased cardiac output
D. Increased intracranial pressure
E. Increased cyclic collapse of unstable alveoli

16. To increase oxygenation during IPPV all of the following are useful EXCEPT:

A. Increase FiO_2
B. Increase PEEP
C. Decrease I:E ratio
D. Increase peak inspiratory pressure
E. Alveolar recruitment

Self-assessment answers

Type K

Q1.	Q2.	Q3.	Q4.	Q5.	Q6.	Q7.	Q8.	Q9.	Q10.	Q.11
A. F B. T C. T D. F	A. F B. F C. T D. F	A. T B. F C. T D. F	A. T B. F C. T D. F	A. F B. F C. T D. F	A. T B. F C. T D. T	A. F B. T C. T D. F	A. F B. T C. T D. F	A. F B. T C. T D. T	A. T B. F C. T D. F	A. T B. T C. T D. T

Type A

12. Answer C is correct
13. Answer E is correct
14. Answer D is correct
15. Answer E is correct
16. Answer C is correct

PATIENT CHALLENGES

A 64-year-old man was admitted to the ICU for severe community-acquired pneumonia.

He has a normal weight (65 kg), with a history of smoking (one pack a day for more than 30 years) but no previous pulmonary disease was reported. On admission to the ICU he was intubated and mechanically ventilated.

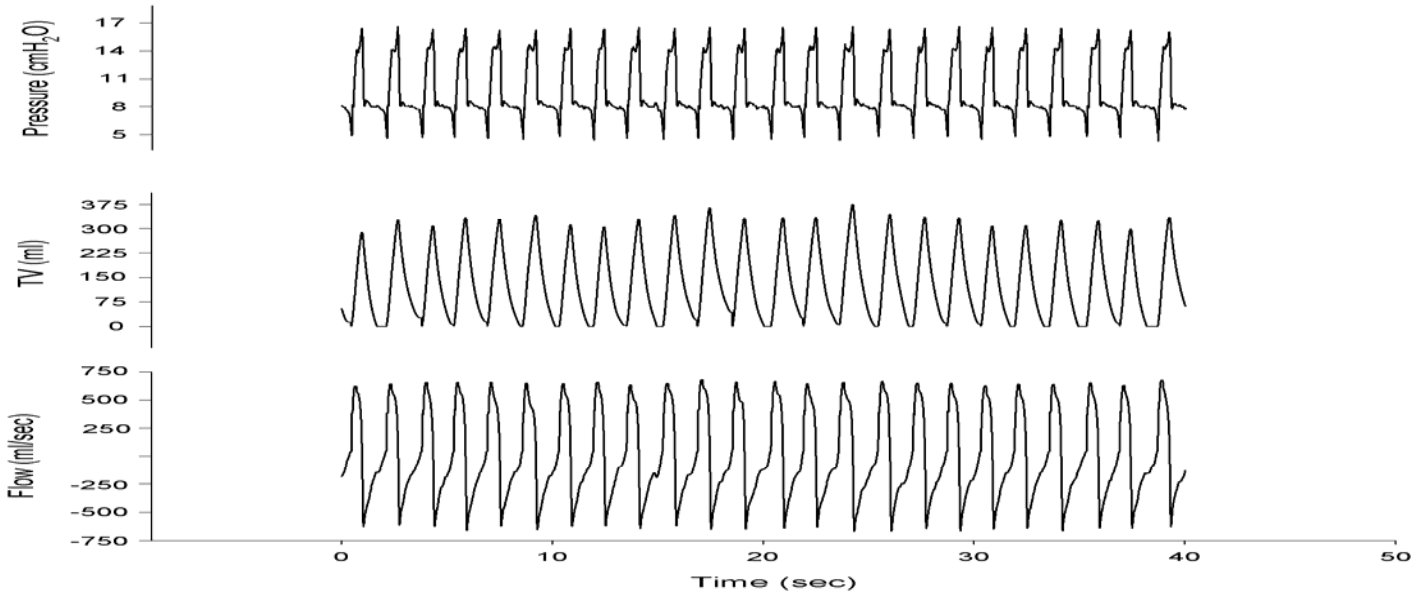
After four days he was receiving pressure-support ventilation. In view of the clinical improvement, sedation was reduced and the level of pressure support was decreased to 6 cmH_2O. After a few hours you find the patient tachypnoeic (respiratory rate 30–35 bpm) with a tidal volume between 300–350 ml, PEEP of 8 cmH_2O and FiO_2 of 0.5.

LEARNING ISSUES
Causes of respiratory failure
Assessment of adequacy of ventilatory support
De-escalation of ventilatory support

See also the PACT module on Respiratory monitoring

You hear on auscultation a few inspiratory crackles and bronchial breathing over the consolidated areas. Accessory inspiratory muscles were clearly being used. ABGs reveal pH 7.38, $PaCO_2$ 42 mmHg (5.5 kPa), PaO_2 89 mmHg (11.7 kPa).
You measure an average P0.1 of 7 cmH_2O.

The ventilator monitor provides the curves shown below.



Q. How do you interpret the respiratory state of this patient?

A. The patient has had to dramatically increase his respiratory effort, which is evident by the elevated P0.1 and the use of accessory inspiratory muscles while attempting to achieve an almost normal level of $PaCO_2$. It seems reasonable to conclude that the level of pressure support was decreased too rapidly.

Q. Is the interaction between patient and ventilator satisfactory?

A. Yes, in spite of the high respiratory rate, there is no dysynchrony between patient and ventilator. From the flow and pressure waveform it appears that all the patient's inspiratory efforts are supported by the ventilator.

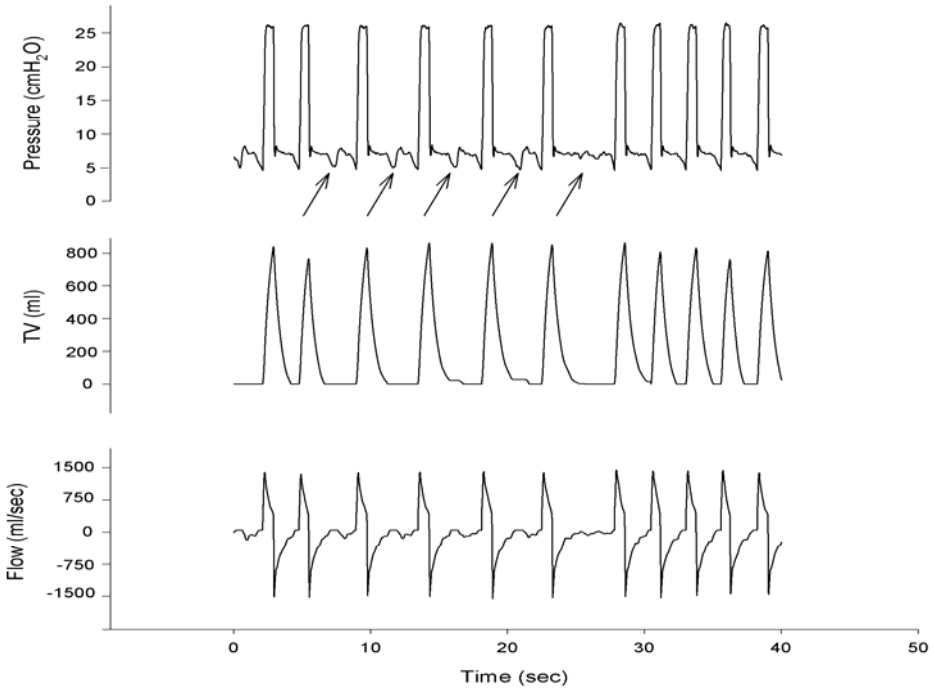
Q. What can you do to relieve the patient's distress?

A. You could simply increase the pressure support level.

LEARNING ISSUES

Assessment of patient-ventilator synchrony
Assessment of patient respiratory work load

You decided to increase the pressure support level to 18. RR (respiratory rate) decreases to 24 bpm, Vt increase to 450 ml while ABGs remain unchanged. P0.1 is now 2 cmH_2O. The following morning the nurse shows you the following ABGs: pH 7.37, $PaCO_2$ 45 mmHg (5.9 kPa), and PaO_2 95 mmHg (12.5 kPa). The ventilator monitor reports a respiratory rate of 17 bpm with tidal volume between 650 and 750 ml. P0.1 is 5 cmH_2O. The ventilator monitor provides the curves shown below.



Though the patient is said not to be uncomfortable, you feel the ventilator settings are not optimum.

Q. Given the respiratory rate (RR) and Vt on the ventilator, what can you say about the measured P0.1?

A. P0.1 of 5 cmH_2O is unexpectedly high and suggests that the patient has a high respiratory drive. On the other hand the RR and Vt on the ventilator seem to indicate a well-supported patient.

Q. How do you interpret the patient-ventilator interaction?

A. There are frequent respiratory efforts that fail to trigger the ventilator – see pointer arrows on pressure trace above. The flow and pressure waveforms show that the patient respiratory rate is higher than the ventilator respiratory rate. There is clearly poor patient–ventilator synchrony. It can be speculated that dynamic hyperinflation was present at the time of attempted triggering. This problem is commonly reported when the level of assistance is too high, leading to large tidal volumes and long insufflation times in patients with chronic obstructive pulmonary disease.

Q. What can you do to improve patient-ventilator synchrony?

A. We interpret the high P0.1 as a sign of patient discomfort due to poor synchronisation between patient and ventilator. Thus, in spite of the P0.1 we decide to decrease the PS level.
A possible alternative in some ventilators is to change the cycling-off criteria for triggering from inspiration to expiration. By decreasing the percentage of inspiratory peak flow at which the ventilator terminates the inspiration (from 25 to 40% for example) you can anticipate expiration. This action may lead to a reduction in the delivered Vt and should decrease the inspiratory time in favour of expiration.

Q. What other reasons to support the need to decrease the PS level?

A. The elevated level of support lead to tidal volumes greater than 10ml/kg, which are higher than the limits recommended in acute lung injury patients to prevent ventilator induced lung injury.

One of your colleagues feels that a high level of support in this patient may help in unloading the respiratory muscle and proposes to increase sedation to decrease the respiratory drive of the patient.

Q. Give some reasons why his choice may not be appropriate.

A. First, at high PS levels decreasing the respiratory drive by sedating the patient may have little effect on the delivered Vt that, as already discussed above, is too high. Second preservation of spontaneous activity may have several advantages in terms of gas exchange, less need of sedation, and faster weaning. Using a high pressure support level reduces the patient respiratory work which may lead to atrophy of respiratory muscles and subsequent difficult weaning. Third, though the patient may not yet be ready for full weaning, he appears able to participate actively in minute ventilation without risk of respiratory muscle exhaustion.

Q. You decrease progressively the PS level while checking the patient's own respiratory rate. During the trial procedure you obtain the following data:
PS 15: ventilator RR 22 bpm, patient RR, 32 bpm, Vt 600 (9.2 ml/kg) MV =13.2 L/min P0.1 3 cmH_2O.
PS 12: ventilator RR 26 bpm, patient RR 27 bpm, Vt 460 (7.1 ml/kg) MV =11.9 L/min P0.1 2 cmH_2O.

PS 8: ventilator RR 32 bpm, patient RR 32 bpm, Vt 420 (6.5 ml/kg) MV =13.5 L/min P0.1 3.5 cmH_2O.
Which level of PS do you think is the most appropriate?

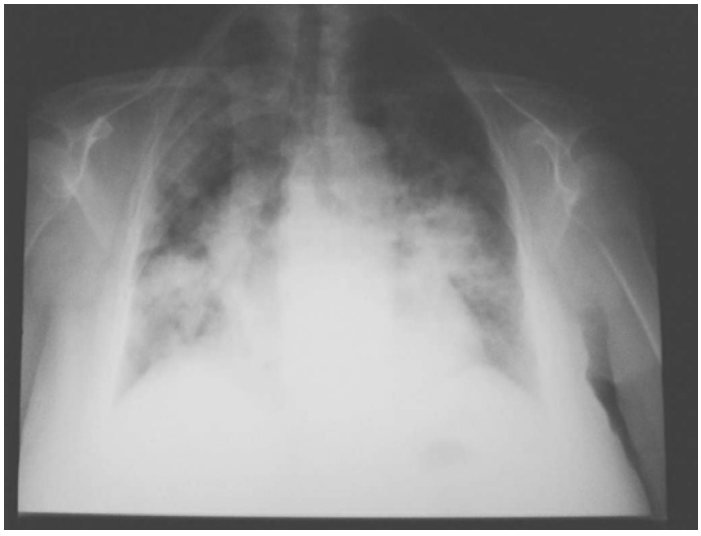
A. A PS level between 8 and 12 seems sufficient to provide adequate support while ensuring that Vt is within the protective range. From PS 18 to 12 P0.1 progressively decreases while patient-ventilator synchrony improves (as indicated by the RR of the ventilator approximating that of the patient). Though a RR of 32 may be easily tolerated in a patient well adapted to the ventilator, at PS 8 the MV and P0.1 are slightly higher than at PS 12. For these reasons, PS 12 may be preferable.

Q. How will you proceed in the forthcoming days?

A. You should progressively decrease the level of PS. Consider that introduction of a protocol that includes daily spontaneous awakening trials, reduction or suspension of sedatives and daily spontaneous breathing trials may improve the outcome of mechanically ventilated patients in intensive care units.

Your colleague on the night shift admitted a 51-year-old man with severe hypoxia due to suspected community-acquired pneumonia from the emergency department**.**

He has a body mass index of 30 (70Kg PBW). The patient had a three-day history of shortness of breath, dyspnoea, cough and fever (max 39°C), before attending the emergency department for worsening dyspnoea. Chest X-ray revealed right middle and lower lobe consolidation and bilateral patchy infiltrates.



In the emergency department the patient had stable haemodynamics (slightly tachycardic) but peripheral saturation of haemoglobin was poor (SpO_2 88%) and deteriorating despite the use of an oxygen mask with a reservoir bag. He was subsequently sedated, intubated as an emergency and admitted to the ICU.

Initial ventilation was with Volume-Controlled Ventilation (VCV) mode, FiO_2 1, PEEP 10 cmH_2O, Vt 700 ml and RR 20. Inspiratory peak and mean airway pressures were 40 and 20 cmH_2O respectively. Haemodynamically he was stable with HR 100 bpm,

BP 110/70 mmHg, CVP 9 mmHg. ABG: PaO_2 70 mmHg (9.2 kPa), $PaCO_2$ 40 mmHg, (5.3 kPa) pH 7.40. Core temperature was 38°C.

LEARNING ISSUES
Pump failure and/or lung failure?
Different reasons for hypoxaemia
Mechanical ventilation

See also the PACT modules on Acute respiratory failure and Respiratory monitoring.

The patient is sedated to tolerate MV, paralysed with repeated boluses of muscle relaxant as needed and breathing 100% oxygen.

Q. Why is the patient so hypoxaemic?

A. Probable reasons for hypoxaemia are: shunt due to perfusion of non-ventilated lung regions (true intrapulmonary shunt). One could in this patient also consider a ventilation–perfusion mismatch (shunt effect) but when breathing 100% oxygen, a ventilation–perfusion mismatch in itself should not cause oxygenation impairment.

Lumb AB, editor. Nunn's Applied Respiratory Physiology. 6th edition. Elsevier/Butterworth Heinemann, Philadelphia; 2005. ISBN 0-7506-8791-6

LEARNING ISSUES
Ventilator monitor

See also the PACT module on Respiratory monitoring

Q. What parameters do you need from the ventilator to obtain a respiratory mechanics profile of the patient (respiratory system compliance, respiratory system resistance, and PEEPi)?

A. It is necessary to perform an inspiratory and expiratory occlusion manoeuvre to obtain the plateau and peak inspiratory airway pressures, Vt, and inspiratory flow during VCV and total level of PEEP.

Q. On the ventilator you measure Vt = 700 ml, Inspiratory Flow = 0.6 L/sec, PEEPi 1 cmH_2O, Peak inspiratory airway pressure=40 cmH_2O, and plateau inspiratory pressure = 34 cmH_2O. Compute respiratory mechanics parameters?

A. Vt is 700 ml, PEEPi 1 cmH_2O. The pressure necessary to counteract the elastic property of the respiratory system is: Plateau pressure – (PEEP+PEEPi) = 37 – (10+1) = 26 cmH_2O. The respiratory system compliance = 700/26 =27 ml/cmH_2O. Inspiratory flow = 0.6 L/sec. The inspiratory flow driving pressure is: Peak pressure – Plateau pressure =3 cmH_2O. Thus, the respiratory system resistance is: 3/0.6 = 5 cmH_2O/L/sec.

LEARNING ISSUES
Choice of tidal volume and frequency
Management of oxygenation

Q. Do the ventilator settings need to be adjusted?

A. The patient is ventilated with 10 ml/Kg PBW with normal pH and pCO_2. Plateau pressure and tidal volume place the patient at risk for ventilator induced lung injury. An attempt should be made to lower tidal volume (<8 ml/kg) and plateau pressure, whilst tolerating hypercarbia (permissive hypercapnia).

LEARNING ISSUES
Management of CO_2 elimination

You lower the tidal volume to 480 ml by keeping the same PEEP level and increase RR to 26 to prevent an excessive increase in $PaCO_2$: indexed tidal volume is now 6.8 ml/Kg PBW, plateau pressure is now 30 cmH_2O, PEEPi 0 cmH_2O, Crs 24 ml/cmH_2O. Mean airway pressure is decreased to 20 cmH_2O. ABG shows: pO_2 55 mmHg (7.2 kPa), pCO_2 47 mmHg (6.2 kPa), pH 7.36.
Haemodynamics are HR 95, AP 120/72 mmHg, CVP 6 mmHg, $SvcO_2$ is low, 65%. Urine output is 70ml/hour.

Q. The new ventilator setting is more protective, though the plateau pressure is still high. However, oxygenation is worse. Can you provide some reasons, related to the new ventilator setting, to explain the decrease in PaO_2?

A. By lowering tidal volume we decreased the plateau pressure and mean airway pressure. The decrease in mean alveolar pressure may have favoured alveolar derecruitment and worsening of intrapulmonary shunt. This possibility is confirmed by the decrease in Crs from 27 to 24 ml/cmH_2O.

LEARNING ISSUES
Management of severe oxygenation impairment
Alveolar recruitment
Recruitment manoeuvres

See also module on Acute respiratory failure

Though a PaO_2 of 55 mmHg (7.2 kPa) could be clinically acceptable in the absence of acidosis or haemodynamic instability, it would be preferable to decrease the FiO_2 which is still set at 1.

Q. What could you do with the ventilator to improve oxygenation?

A. You could try to promote alveolar recruitment by increasing the PEEP level and/or using recruitment manoeuvres.

Some of your colleagues are reluctant to increase PEEP given the already high plateau pressure. Thus, in order to improve oxygenation you perform a 40 seconds-40cmH_2O pressure sustained recruitment manoeuvre (RM). During the manoeuvre arterial

systolic pressure and oxygen saturation drop to 70 mmHg and 80% respectively. Five minutes after the end of the manoeuvre Crs increases to 35 ml/cmH_2O, and PaO_2 increases to 80 mmHg (10.5 kPa). You decide to leave the ventilator settings unchanged. However, after 20 minutes $SatO_2$, PaO_2 and Crs return to the same values as before the RM.

Q. How do you interpret the effect of RM?

A. The increase in Crs and PaO_2 indicate that some alveolar recruitment may have taken place after RM. However this effect was short-lived suggesting the need of a higher PEEP level to better counteract alveolar derecruitment.

The important decrease in arterial pressure and oxygen saturation during the RM suggests some haemodynamic impairment: it seems reasonable to suspect an acute reduction in venous return due to the increased intrathoracic pressure.

LEARNING ISSUES

Management of severe oxygenation impairment
Haemodynamic monitoring
Pulmonary artery catheter
PEEP

See also the PACT module on Haemodynamic monitoring

Now you decide to perform a PEEP trial. However you are worried about haemodynamic side effects. Echocardiography suggests the patient needs some fluid resuscitation and a pulmonary artery catheter is inserted in the left jugular vein to monitor continuously the cardiac output (CO) and mixed venous oxygen saturation (SvO_2) shows: CO 5L/min, pulmonary artery pressure (PAP) 28/15 mmHg, pulmonary artery occlusion pressure (PAOP) 13 mmHg, SvO_2 60%. Fluid challenge with 10ml/Kg crystalloids is then performed: CO 7.7L/min, PAP 27/15, PAOP 12, SvO_2 75%.
Intrapulmonary shunt is obtained by standard calculations:
$Qshunt/Qtotal = (CcO_2 - CaO_2)/(CcO_2-CvO_2) = 47\%$
(where CcO_2 is calculated pulmonary capillary oxygen content, and CaO_2 and CvO_2 are arterial and mixed venous oxygen content, respectively).
Then, you perform a PEEP trial. You set PEEP 12, 14, and 16 cmH_2O for 20 minutes. You check plateau pressure, Crs, ABG, cardiac output and SvO_2 at the end of each step. You obtain the following results:
PEEP 12: Plateau = 29 cmH_2O, Crs = 28 mL/cmH_2O, pO_2 = 80 mmHg, CO =7.8 L/min, SvO_2= 76
PEEP 14: Plateau = 29 cmH_2O, Crs = 32 mL/cmH_2O, pO_2 = 130 mmHg, CO =7.6 L/min, SvO_2= 77
PEEP 16: Plateau = 32 cmH_2O, Crs = 30 mL/cmH_2O, pO_2 = 135 mmHg, CO =6.5 L/min, SvO_2= 72

Q. How do you interpret the results?

A. The increase in PEEP produced an improvement in oxygenation and Crs. Plateau pressure increased by less than the increase in PEEP suggesting alveolar recruitment. However, from PEEP 14 to PEEP 16 Crs slightly decreased, suggesting possible over distension of ventilated alveoli.

Moreover at PEEP 16, CO and SvO_2 decreased. Overall PEEP 14 appears to be the best compromise.

Q. Would you attempt another RM?

A. The increase in Crs during the previous RM was higher than the Crs we obtained with the increase in PEEP. Now that we have a higher PEEP, an RM could have a more sustained effect.

You perform a second 40 seconds-40 cmH_20 sustained recruitment manoeuvre: arterial systolic pressure and oxygen saturation do not significantly change during the manoeuvre. After 20 minutes ABG improve: PaO_2 170 mmHg (22.4 kPa), $PaCO_2$ 46 (6.1 kPa), pH 7.36. Crs increases to 37 ml/cmH_2O, plateau pressure decreases to 27 cmH_2O. Intrapulmonary shunt decreases to 23%. FiO_2 is progressively reduced to 0.6 to maintain a PaO_2 of more than 75 mmHg (9.9 kPa). The patient gradually improves and is de-escalated and weaned from mechanical ventilation after seven days.

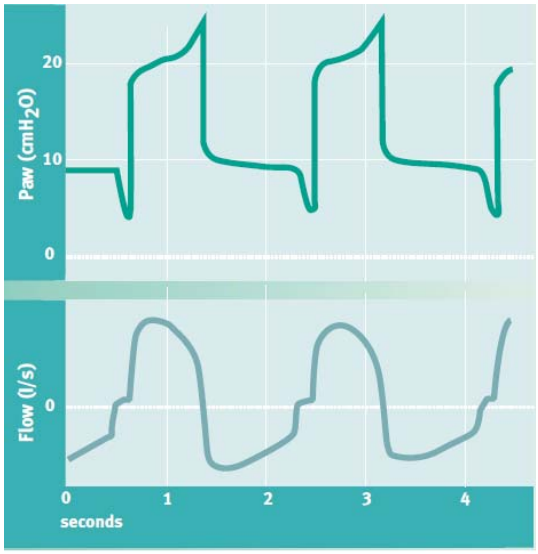
A 73-year-old lady is admitted to your ICU for treatment of pulmonary thromboembolism (PTE) after hip replacement. She is obese (95 kg, with an estimated ideal body weight of 65 kg) and there is no previous history of respiratory disease. She is receiving i.v. heparin. On the third night, due to rapid respiratory deterioration, she is intubated and mechanically ventilated.

The next morning, the patient is lightly sedated and being ventilated with pressure support ventilation (PSV), with an above-PEEP pressure support (PS) level of 15 cmH_2O. The PEEP level is set at 8 cmH_2O and FiO_2 at 0.6 and humidification is with a Heat and Moisture Exchanger (HME).

She has just become severely dyspnoeic, with a respiratory rate of 32 bpm and Vt of 400 ml. There is some wheezing on auscultation but, otherwise, no other evidence of superimposed or complicating pathology.

LEARNING ISSUES
Causes of respiratory failure
Assessment of adequacy of ventilatory support
Choice of humidification device

You hear few pathological sounds (some rhonchi and wheezing) on auscultation. ABGs reveal pH 7.24, $PaCO_2$ 62 mmHg (8.1 kPa), PaO_2 60 mmHg (7.9 kPa), BE -2 mEq/l. Haemodynamics are satisfactory with low level cathecholamine support. The ventilator monitor shows the curves illustrated below. You also note that the end-tidal CO_2 pressure of 40 mmHg (5.3 kPa) is much lower than the arterial $PaCO_2$ indicating an increased alveolar dead space.



LEARNING ISSUES
Tuning of mechanical support

NOTE This clinical case provides an opportunity for diagnostic and therapeutic exercises in a complex case of respiratory failure. It is not to be used as a guide to mechanical ventilation in PTE. No specific ventilatory approach is recommended for PTE.

Q. How do you interpret the respiratory state of this patient?

A. A new embolic episode probably took place. The patient presents with a combination of a high ventilation requirement (due to high alveolar dead space typical of PTE) and increased impedance (possibly due to airway obstruction, as far as you can judge from the expiratory flow curve and the presence of wheezing). The combined action of 15 cmH_2O PS and important inspiratory effort (evident from deep drop of Paw with trigger) is not sufficient to generate the high ventilation needed to achieve a normal $PaCO_2$ and pH.

LEARNING ISSUES
Pump failure or lung failure?
Breath initiation: Machine vs patient
Ventilator monitor
Link to PACT module Respiratory monitoring

Q. What would help you to gain a better picture of the patient's respiratory state?

[Prompt: When a ventilated patient is breathing with difficulty, first rule out any technical problem.]

A. You need to:

- Check for possible obstruction of ET-tube or HME, and other technical problems
- Maximise any possible pharmacological intervention to reverse bronchospasm

- Check for appropriate setting of trigger sensitivity (whether pressure or flow-triggered)
- Obtain a chest X-ray
- Consider assessment/measurements of respiratory mechanics (focusing on PEEPi).

LEARNING ISSUES
Ventilator monitor

Q. An HME humidifier is being used. Explain why this choice of humidifier may be inappropriate.

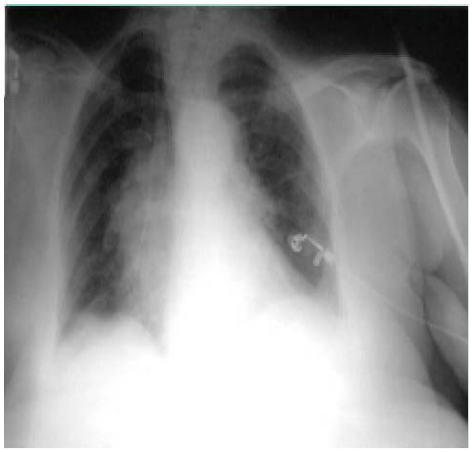
A. HMEs increase the apparatus dead space, and hence are contraindicated in patients with intrinsic difficulties in CO_2 elimination. You replace the HME with a hot-water humidifier.

Q. Does this patient need any change in trigger setting?

A. Increasing the sensitivity of the trigger may reduce the inspiratory work of breathing.

NOTE It is possible that this patient has a high level of intrinsic PEEP (PEEPi) and in order to counteract the PEEPi, she needs to achieve a considerable increase in her inspiratory effort to trigger the ventilator. Increasing trigger sensitivity will not be adequate in such a situation to reduce the excessive inspiratory workload.

LEARNING ISSUES
Gas conditioning
Circuit dead space, compliance and resistance



On chest X-ray, you rule out ET-tube obstruction or malposition and you note a small quantity of thick mucus on suction. A colleague performs an end-expiratory occlusion manoeuvre, and measures a total PEEP (PEEPtot) of 16 cmH_2O, much higher than the applied PEEP of 8 cmH_2O (PEEPi is 8 cmH_2O).

LEARNING ISSUES
Assessment of passive respiratory mechanics, see Ventilator monitor

Q. How do you proceed with the assessment?

[Prompt: During spontaneous or assisted breathing, observation of the flow curve provides a qualitative assessment of dynamic hyperinflation, but quantification of airway obstruction is very imprecise.**]**

A. The clinical information and measured data strongly suggests acute small airway obstruction, which may be further evaluated and quantified by measurement of respiratory mechanics performed during relaxed ventilation.

There is agreement with your suggestion. The patient is sedated, paralysed (with neuromuscular blocker), and ventilated using volume-controlled ventilation (VCV), with constant inspiratory flow, frequency 18 bpm, Vt 520 ml (8ml/kg), I:E 1:2, inspiratory pause 10%, and unchanged PEEP of 8 cmH_2O.

On the ventilator monitor, data generated by automatic breath analysis indicate an inspiratory resistance of 23 $cmH_2O/l/s$ and a quasi-static compliance (Cqs) of 27 ml/cmH_2O. The end-expiratory flow is far from zero. An end-expiratory occlusion manoeuvre indicates a PEEPtot of 13 cmH_2O.

This suggests a high level of intrinsic PEEP (PEEPi of 5 cmH_2O) which optimally requires confirmation of automatic breath analysis data by manual measurements.

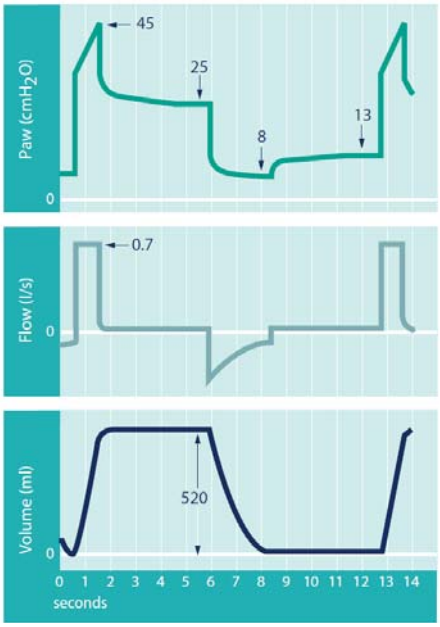
LEARNING ISSUES

Ventilator monitor – automatic analysis of passive mechanics

Q. If a fully comprehensive picture were desired, why are the above data not sufficient and what might you perform in addition?

A. Data confirm small airway obstruction but PEEPi is the only reliable measurement of respiratory mechanics, of those available to date. Manual measurements with a double, prolonged occlusion manoeuvre, at end-inspiration and end-exhalation, will provide further clarification.

Manual measurements on the static screen, with end-inspiratory and end-expiratory pauses of 4 seconds indicate data as follows – see also figure below.
Ppeak=45 cmH_2O
Ppause=25 cmH_2O
PEEP=8 cmH_2O
PEEPtot=13 cmH_2O
Vt=520 ml
End-Insp Flow=0.7 l/s



LEARNING ISSUES

Ventilator monitor – manual measurements of passive mechanics

Q. It is possible from the above data to calculate derived indices of respiratory mechanics. If you opted to do this, what are the values for maximum inspiratory resistance (Rmax), quasi-static compliance (Cqs), respiratory system time constant (RC), and intrinsic PEEP (PEEPi).

A. Rmax = (45 – 25) / 0.7 = 28.5 $cmH_2O/l/s$
Cqs = 520 / (25 – 13) = 43 ml/cmH_2O
RC = 28.5 x 43 / 1000 = 1.23 sec
PEEPi = 13 – 8 = 5 cmH_2O

You repeat the measurements while PEEP is temporarily lowered to zero (ZEEP). Results are similar to those with PEEP viz. the PEEPi is 6 cmH_2O, Rmax 31 $cmH_2O/l/s$, Cqs 40 ml/cmH_2O and RC 1.2 sec.

After resuming the previous PEEP of 8 cmH_2O, with passive VCV, Vt 520 ml, frequency 18 bpm and FiO_2 0.6, the arterial blood gases (ABGs) reveal a pH of 7.23, $PaCO_2$ of 67 mmHg (8.8 kPa) and PaO_2 of 62 mmHg (8.2 kPa).

Q. How do you interpret these values of quasi-static compliance?

[Prompt: Compliance data cannot be interpreted without considering the patient's ideal body weight and body shape, that is considering chest wall compliance**]**

A. The specific Cqs referred to the ideal body weight is 0.66 and 0.62 $ml/cmH_2O/kg$, respectively at PEEP and ZEEP. These values are not much lower than a normal specific Cqs of 1 $ml/cmH_2O/kg$. We must consider that the chest X-ray shows clear lungs in a small-size rib cage surrounded by much fat and heavy breast tissue. The slight reduction in total compliance probably depends on the patient's body shape. Lung elasticity is probably close to normal.

Q. A visiting consultant arrives on the ward. How would you summarise the information derived from the respiratory dynamics.

A. PTE has been complicated by bronchial obstruction, with a clinical picture similar to asthma: airway resistance is high, PEEPi is relevant even at normal respiratory frequency, compliance is just slightly decreased and the expiratory flow curve denotes slow exhalation and confirms dynamic hyperinflation.

Externally applied PEEP has mainly an additive effect on PEEPi in this patient. The reduced PaO_2 is mainly due to ventilation-perfusion mismatch and not to shunt, indicating that externally applied PEEP has minimal effect on improving oxygenation. Moreover it increases the intra-alveolar pressure further, impeding the outflow from the right heart and risks inducing acute cor pulmonale. Bronchial obstruction worsens CO_2 elimination, which was already impaired due to the direct effect of PTE.

LEARNING ISSUES

Pump failure or lung failure?
Role of PEEP and intrinsic PEEP on respiratory mechanics and gas exchange

You decide to try to optimise the ventilator settings.

Q. What is your $PaCO_2$ target, and what minute ventilation may be required? Justify your answer.

A. Due to PTE, significant hyperventilation would be required to achieve a normal $PaCO_2$. The patient showed dynamic hyperinflation due to the presence of bronchial obstruction and an insufficient expiratory time. Therefore respiratory rate should be decreased and hypercapnia should be accepted. In order to avoid excessive hypercapnia, a different ventilation mode could be considered.

LEARNING ISSUES

Principles guiding mechanical ventilation
Management of CO_2 elimination
$PaCO_2$ and pH targets
Alveolar ventilation and minute ventilation

Q. In the present context, what are the alternative choices of ventilation mode?

A. A reasonable choice is to continue passive ventilation, facilitated by sedation and (if necessary) neuromuscular blockade, with either VCV or PCV. As an alternative, you could try BIPAP.

LEARNING ISSUES

Ventilation modes
Timing and strategies
Assistance of respiratory muscles

Q. What are your targets for the ventilatory pattern?

A. You should aim to increase the expiratory time, in order to decrease dynamic hyperinflation and PEEPi. An expiratory time of at least three times the RC is needed, i.e. of 3.6 seconds. Assuming you set an inspiratory time of 1 sec, the total cycle time results in 4.6 sec. The set frequency should not be higher than 60 / 4.6 = 13 bpm. The resulting I:E will be low, equal to 1:3.6 Assuming a minute ventilation of 10 l/min, Vt would be equal to 770 ml.

LEARNING ISSUES
Choice of tidal volume and frequency
Choice of I:E ratio

Q. The lungs are clear. Mechanically, the present PEEP level of 8 cmH_2O does not seem to provide relevant benefits in terms of respiratory mechanics. What is the cause of the PEEPi?

A. PEEPi does not decrease from ZEEP to PEEP 8 cmH_2O suggesting that PEEPi is mainly due to dynamic hyperinflation rather than to flow limitation.

Therefore you consider a reduction in PEEP, especially given the current mode of ventilation is passive. However, oxygenation is greatly impaired, and might worsen further at lower PEEP.

Q. Normally a reduction in PEEP would be expected to be associated with a deterioration in oxygenation. Are there possible mechanisms whereby it might be associated with an improvement in oxygenation in this patient?

A. A PEEP reduction might reduce the right ventricular afterload and improve right and left heart function and thus, by improving mixed venous PO_2, result in benefit to the arterial PaO_2.

Q. What do you do to improve oxygenation?

A. All things considered, you opt to increase the FiO_2.

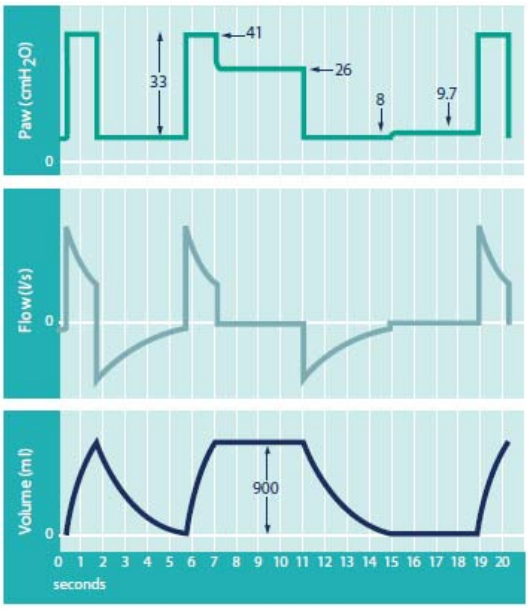
LEARNING ISSUES
Management of oxygenation
De-escalation and weaning

NOTE In severely hypoxaemic patients, a major reduction in PEEP should be avoided.

You maintain sedation and paralysis, and change the mode to PCV, with a frequency of 13 bpm and I:E 1:3.6 You maintain PEEP at 8 cmH_2O, while increasing the FiO_2 to 0.8. You adjust the above-PEEP inspiratory pressure (Pcontr) to 18 cmH_2O, to achieve a Vt of 770 ml. Ppeak is 28 cmH_2O.

NOTE When you choose PCV for passive ventilation, first decide your tidal volume target, and then adjust the inspiratory pressure to achieve this tidal volume. With several dual-control modes of ventilation, this latter task is automatically performed by the ventilator.

A double occlusion manoeuvre shows an inspiratory pause pressure of 26 cmH_2O and a PEEPtot of 9.7 cmH_2O (PEEPi is 1.7 cmH_2O). ABGs reveal pH of 7.30, $PaCO_2$ of 50 mmHg (6.6 kPa) and PaO_2 of 52 mmHg (6.8 kPa). Haemodynamics are stable.



LEARNING ISSUES

Inspiration: Volume-control vs pressure-control

Q. Give arguments as to why you think some of the new ventilator settings are safe and effective and others are not?

A. The new settings have been effective in reducing dynamic hyperinflation and improving $PaCO_2$ and pH.

Ppeak is acceptable, considering that it includes the effect of tracheal tube resistance, but the safety of the observed inspiratory pause pressure is questionable.

Moreover, although FiO_2 has been increased, oxygenation has unexpectedly and critically worsened, with the new settings.

Q. How do you re-adjust the ventilator settings?

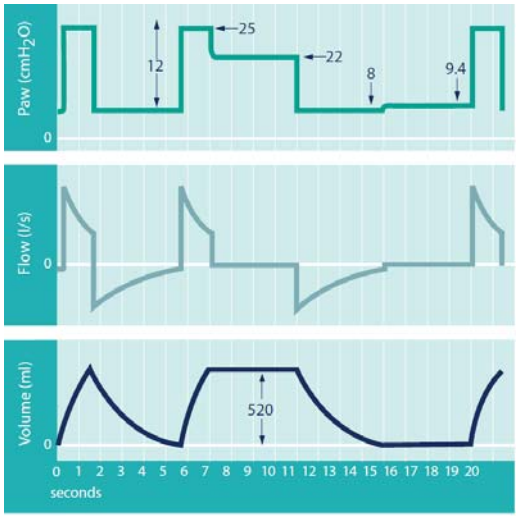
[Prompt: Always remember that static pressures (like the inspiratory pause pressure) depend on volumes.**]**

A. In order to decrease the inspiratory plateau pressure, you should decrease your target for Vt. You cannot compensate for the consequent decrease in ventilation by increasing the frequency, otherwise dynamic hyperinflation would worsen once

again, compromising the effective reduction in pressures. Therefore you must accept a higher $PaCO_2$.

NOTE The choice of Vt depends on the balance between baseline pressure (PEEP), the requirement for adequate alveolar ventilation, and potential damage due to the application of excessive pressures.

You reduce the Vt to 520 ml, by lowering Pcontr to 12 cmH_2O. PEEPtot is 9.4 cmH_2O, with a PEEPi of only 1.4 cmH_2O. Ppeak is 25 cmH_2O, with an inspiratory plateau pressure of 22 cmH_2O. Minute ventilation is 6.8 l/min. ABGs reveal pH of 7.24, $PaCO_2$ of 63 mmHg (8.3 kPa) and PaO_2 of 60 mmHg (7.9 kPa).



The new ventilatory pattern, with low frequency, low I:E, and Vt just slightly higher than the ideal 6ml/kg, combines acceptable inspiratory pressures with near-complete avoidance of dynamic hyperinflation. Ventilation is just slightly higher than normal, while the $PaCO_2$ level is acceptable. You consider partial correction of pH by slow bicarbonate administration, and decide to wait.

NOTE Permissive hypercapnia is not necessarily synonymous with low Vt and absolute hypoventilation.

Haemodynamics are stable, but oxygenation is still considerably impaired. A transoesophageal echocardiograph is suggested. It shows a patent foramen ovale, with right-to-left shunt especially evident during inspiration.

NOTE Intrapulmonary shunt is the most common cause of hypoxaemia, but not the only cause.

Q. How can you try to reduce the extrapulmonary shunt by adjusting the mechanical ventilator?

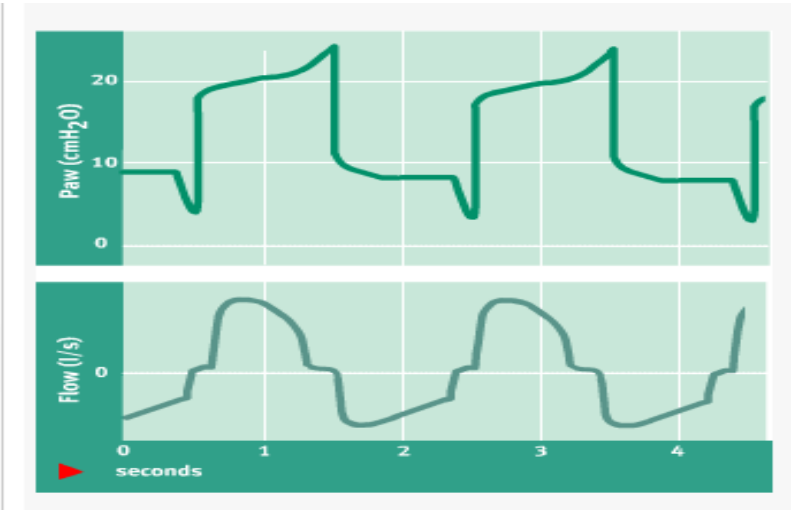
A. You should try a substantial reduction of PEEP, in order to possibly reduce right heart afterload and intracardiac right-to-left shunt.

LEARNING ISSUES

Extrapulmonary shunt
Paradoxical effect of PEEP on oxygenation

You make a trial with PEEP set at zero. With unchanged settings for frequency and I:E, an unchanged Vt of 520 ml is obtained by setting a Pcontr of 12 cmH_2O, that corresponds to an inspiratory pause pressure of 12 cmH_2O. PEEPi is 2 cmH2O. PaO_2 increases to 108 mmHg (14.2 kPa), while $PaCO_2$ decreases to 58 mmHg (7.6 kPa).

An hour later, oxygen saturation on the SpO_2 monitor suddenly drops, while the patient is dyspnoeic and appears to be fighting the ventilator. The ventilator screen shows the curves below.



LEARNING ISSUES

Disadvantage of machine-cycling in actively breathing patients
Cycling to exhalation: Machine vs patient

Q. What is wrong, and how would you intervene?

A. The patient has started spontaneous breathing at high frequency. The fixed inspiratory time of PCV now generates inverse I:E, resulting in a major increase in intrathoracic pressures due to dynamic hyperinflation. You should either:
- Disconnect the patient from ventilator and take control with careful manual ventilation.
- Thoroughly review the ventilator settings, for instance by reducing the inspiratory time or switching to PSV, while re-adjusting the inspiratory pressure level.

Or
- Re-administer neuromuscular blockade to 'paralyse' the patient.

Q. Has anything been forgotten, in the treatment?

[Prompt: Do not forget to combine aetiological treatments, when available.]

A. Bronchodilators. The airway obstruction has not been treated, but just compensated by appropriate settings of the ventilator.

The treatment is continued with sedation, paralysis and passive ventilation with low frequency, low I:E ratio and low PEEP. A β-agonist is added and the dose of i.v. heparin is increased. On the following day sedation is reduced and a trial with PSV is successful. De-escalation of mechanical support is continued slowly with PSV, and the patient is successfully extubated after 12 days of ventilation.

On reflection,
In the first patient, clinical examination coupled with observation of the respiratory waveform trace demonstrated lack of synchrony between the patient and the ventilator and how ventilator adjustments allowed synchrony and improved patient respiratory comfort to be achieved allowing controlled de-escalation of ventilation.

The second case, of type 1 respiratory failure, illustrated the value of a recruitment manoeuvre and optimising the PEEP level to recruit airways and increase functional residual capacity thus minimising intrapulmonary shunt effect and improving oxygenation. Case three was a complex scenario where a combination of pathologies pertained causing a delicate balance between: severe intrapulmonary and extrapulmonary shunt and bronchial (small airway) obstruction.

The key approaches to finding a favourable ventilation strategy were to

- Diagnose the underlying diseases
- Review the ABGs and chest X-ray
- Undertake waveform and data analysis with the patient sedated and ‘relaxed’
- Analyse serial data, including respiratory mechanics and haemodynamics
- Break down the problems and consider the management of the different physiological tasks
- Identify optimal targets
- Monitor the results of your modified ventilator settings and verify their effect/benefit.

This approach, though complex and only fully required in a minority of patients, illustrates a complete approach to the analysis of respiratory dynamics in a ventilated patient and the importance of a patient-centred, serial approach to optimisation of the mechanical respiratory support. However remember that achieving favourable results with such a multifaceted approach is complex and continued attention to detail will be required as things may change rapidly in the clinical setting.

By continuing these processes in this instance, you were able to successfully wean the patient from mechanical ventilation. The underlying disease improved during this time but remember however that recovery can take time!

Finally, always consider that, in most cases, mechanical ventilation is a symptomatic treatment. Never forget the treatment of the underlying condition(s).