

Ventilator Settings for Different Disease States

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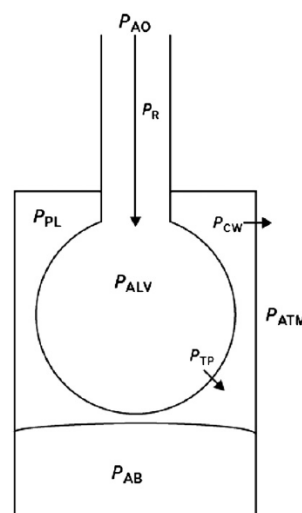
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Understanding the need for special ventilator settings for different disease states requires that we first understand the basics of how ventilators support respiratory function, and also how ventilatory parameters can damage the lungs.

Why do patients need ventilator treatment?

Short-term ventilator support may be required for patients with relatively normal lungs either during or after anaesthesia. Indications for mechanical ventilation in the ICU may be grouped under the following main headings:

- i. Failure to protect the airway
- ii. Failure to ventilate due to neurological, muscular or mechanical factors (eg. Flail chest, pneumothorax, bronchospasm, pleural effusion, abdominal compartment syndrome and so on)
- iii. Airway obstruction
- iv. Failure to oxygenate due to V/Q mismatch (shunting, dead space ventilation), or diffusion defect.
- v. Failure to utilize oxygen: sepsis, cyanide, carbon monoxide poisoning



The figure shows the various pressures determining air flow in the respiratory system. The volume of each alveolus depends on the balance between the inflating pressure or P_{ALV} and the pleural pressure or P_{PL} , which is influenced by the compliance of chest wall as well as abdominal pressure,

P_{AB} . Air flow at any instant during inspiration or expiration is determined by the pressure gradient between P_{ALV} and P_{AO} as well as resistance of the airways.

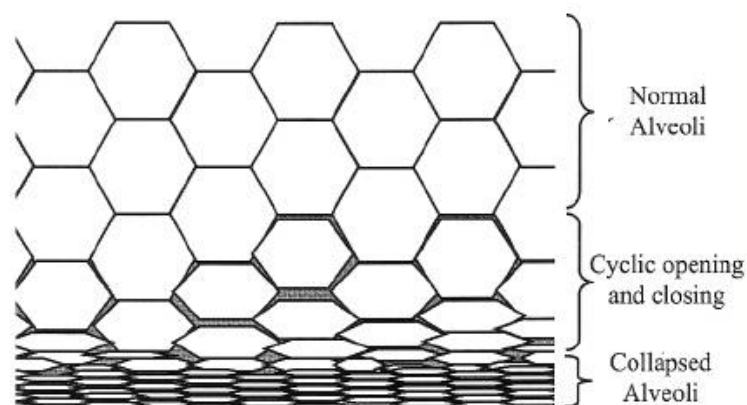
Adverse effects of mechanical ventilation:

- **Barotrauma:** Lung injury associated with high alveolar pressures ($>35-40$ cm H_2O), presentation ranges from asymptomatic pulmonary interstitial emphysema (PIE), through subcutaneous emphysema, pneumomediastinum; to tension pneumothorax.
- **Volutrauma:** This term refers to lung injury due to overstretching of alveoli because of excessive tidal volumes, which leads to release of inflammatory mediators that have both pulmonary and systemic effects, increasing morbidity and mortality. When non-homogenous pulmonary pathology is present, tidal volume delivered by the ventilator is preferentially delivered to the more compliant, relatively normal areas of the lung. Thus normal alveoli are overstretched, and the remaining normal lung is damaged, further increasing lung injury and damage. Repeated closing and re-opening of alveoli (recruitment and collapse) is another factor which causes damage due to shear stress and loss of surfactant. The release of inflammatory mediators can cause damage to remote organs such as the liver and kidney.

Barotrauma is due to high alveolar pressures while volutrauma is related to high trans-alveolar pressure gradient.

- **Auto-PEEP:** This is the positive pressure which develops in the alveoli at the end of expiration due to incomplete emptying of alveoli, either due to airway obstruction due to secretions, bronchospasm or airway closure; or else if expiratory time is too short. This cannot be detected at the ventilator end of the tube but causes increased work of breathing and progressive overdistension of the lungs, if not corrected.
- **Oxygen toxicity:** High inspired oxygen concentrations ($FiO_2 > 0.5$) cause cellular damage due to free radical formation. In addition, high FiO_2 s are also associated with absorption atelectasis. Hence it is important to set the lowest FiO_2 possible, aiming for $SaO_2 > 90\%$
- **Cardiovascular effects** of mechanical ventilation include decreased venous return, RV dysfunction and altered LV compliance.

Shear Forces



Initial ventilator settings:

Mode of ventilation

The choice depends on whether or not the patient is conscious, sedated or has received neuromuscular blockers. SIMV or A/CMV modes are appropriate for initial settings. Patients with a good respiratory drive can be placed on PSV mode.

Tidal volume:

Currently, lower tidal volumes are recommended than in the past, and 5-8 ml/Kg of ideal body weight should be set, adjusting the value so that P_{plat} is less than 35 cm H₂O.

Respiratory rate:

This should be set at 8-10 breaths per minute, unless hyperventilation is required for intracranial pathology or metabolic acidosis. Higher rates may result in air trapping and intrinsic PEEP.

Inspired oxygen level:

Though an initial FiO₂ of 1 is usually recommended, it is advisable to reduce the FiO₂ as rapidly as possible, aiming for an oxygen saturation >90% and PaO₂ >60 mmHg.

Inspiration/expiration ratio:

Normally the I/E ratio is set at 1:2. Expiratory time may be increased if airway obstruction is present so as to avoid air trapping and auto-PEEP. Inspiratory time may need to be prolonged in the setting of severe ARDS so as to equal or even exceed expiratory time (inverse ratio ventilation).

Inspiratory flow rate:

Inspiratory peak flow rate may need to be set in some ventilators when volume control ventilation (VCV) is being used. It should be set at 4 times the desired minute volume so that the patient's inspiratory flow requirement is met. If the flow is too low, the patient needs to make an extra effort, and too high an inspiratory flow causes the peak pressure to be too high. It is usually set at 60 L/min to start with.

In newer ventilators, the peak flow is set automatically, depending on the tidal volume, I/E ratio, respiratory rates and inspiratory pause, if present.

During pressure modes of ventilation, the flow is determined by the inspiratory pressure and the resistance and compliance of the respiratory system.

Positive end-expiratory pressure

Even in patients with normal lungs, PEEP of 3-5 cm H₂O should be set during mechanical ventilation, in order to prevent decreases in FRC and dynamic airway collapse. Higher levels of PEEP may be necessary in patients with diseased lungs, so that acceptable oxygenation may be achieved with lower inspired oxygen concentrations.

Several methods have been described for selecting the optimal PEEP, such as setting the PEEP at 1 or 2 cm H₂O above the lower inflexion point of a pressure/volume loop. Alternatively, a high PEEP can be set initially (eg. 20 cm H₂O) and then progressively decreased till desaturation occurs. At this point, a recruitment manoeuvre is done and the PEEP is set about 2 cm H₂O higher.

Sensitivity:

If pressure triggering is used, the trigger sensitivity is set at -2 cm H₂O initially, keeping in mind that the presence of intrinsic PEEP increases the effort required by the patient if an adequate level of extrinsic PEEP is not set. Modern ventilators provide the option of flow-triggering which needs less effort from the patient.

Specific disease states

Obstructive airway disease

The main physiological abnormality in these conditions is an increase in airway resistance, especially during expiration. This has a large reversible component in bronchial asthma, unlike chronic obstructive pulmonary disease (COPD). In COPD, there is an abnormal and amplified inflammatory response to various noxious substances, which affects the airways, parenchyma as well as the pulmonary vasculature. The airway obstruction has only a small reversible component, and is due to the following factors:

- i. Oedema, smooth muscle hypertrophy peri-bronchial fibrosis
- ii. Excessive mucus secretion and plugging
- iii. Destruction of alveoli and respiratory bronchioles with reduced recoil. This predisposes to collapse of conducting airways.
- iv. Hyper-responsive airway reflexes – bronchospasm

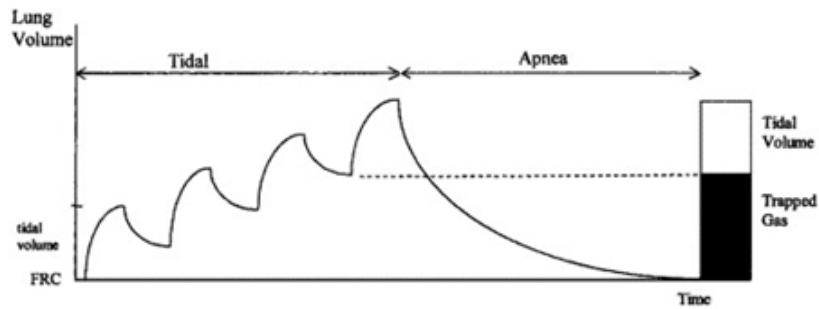
The increased airway resistance leads to expiratory flow limitation (Intra-thoracic airway diameter is always smaller during expiration, during spontaneous respiratory efforts). This, along with inadequate time for expiration due to tachypnoea, causes dynamic hyperinflation leading to respiratory embarrassment, increased work of breathing and hemodynamic compromise. The goals of ventilator support are to minimize intrinsic PEEP, rest the respiratory muscles and maintain acceptable blood gases.

Initial settings in these patients consist of a low tidal volume (6-8 ml/Kg), high inspiratory peak flow (80-100 ml/min), low PEEP (0-5 cmH₂O), low respiratory rate and I:E ratio of 1:4 to 1:5. Traditionally, assist/control mode is chosen, but there is no consensus on which mode of ventilation should be set initially in obstructive airway disease. Volume ventilation ensures delivery of the set tidal volume, but the distribution of that tidal volume may be patchy, with reduction of flow through constricted airways causing an increased inspiratory pressure and resultant dilatation of non-constricted airways elsewhere in the lungs. Changing to pressure controlled ventilation has been shown to improve gas exchange in pediatric patients. A simulation study using an integrative network model of the lung showed PCV produced a more uniform distribution of tidal volume, but the delivered tidal volume was highly sensitive to small changes in inspiratory pressure setting. Dual modes such as PCRV or autoflow may be helpful in this situation; these are essentially PCV with the inspiratory pressure continually adjusted to deliver the set tidal volume.

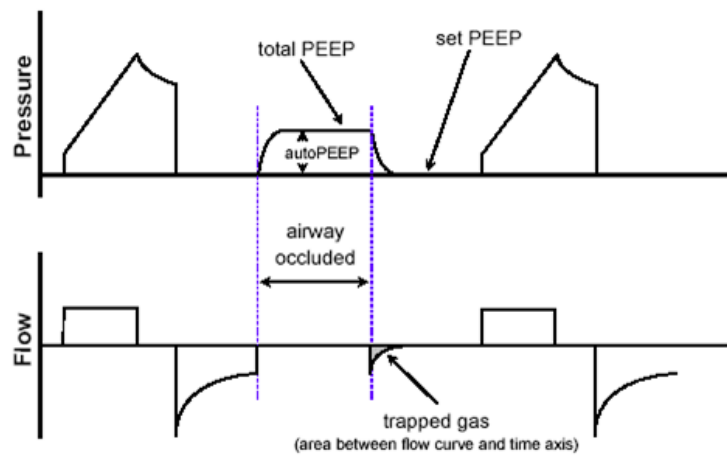
Dynamic hyperinflation may be estimated by measuring the end-inspiratory volume above FRC, by collecting the total exhaled volume over 60 sec of apnoea. Other estimates of dynamic hyperinflation are the plateau pressure and intrinsic PEEP which can be measured during an

expiratory hold manoeuvre. The ventilator flow-time tracing shows continued expiratory flow at the beginning of inspiration, showing that the lung has not fully emptied, i.e. air trapping is taking place.

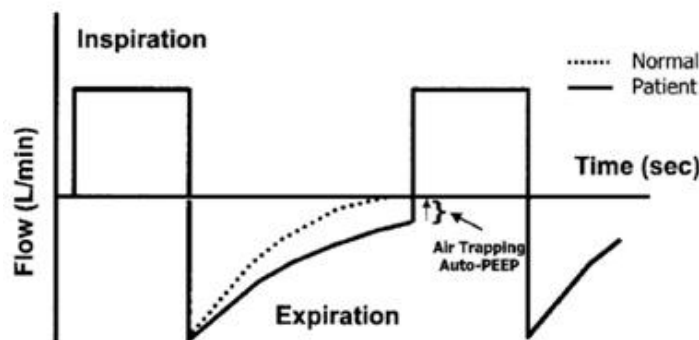
These ventilator settings with a low minute ventilation lead to hypercapnia and respiratory acidosis. Permissive hypercapnia is tolerated down to a pH of 7.15, below which sodium bicarbonate or THAM infusions are recommended. Adequate sedation and sometimes, neuromuscular blockade is required in these patients to achieve adequate ventilation.



Dynamic hyperinflation



Measuring autoPEEP by expiratory hold



**Expiratory flow limitation
Auto-PEEP**

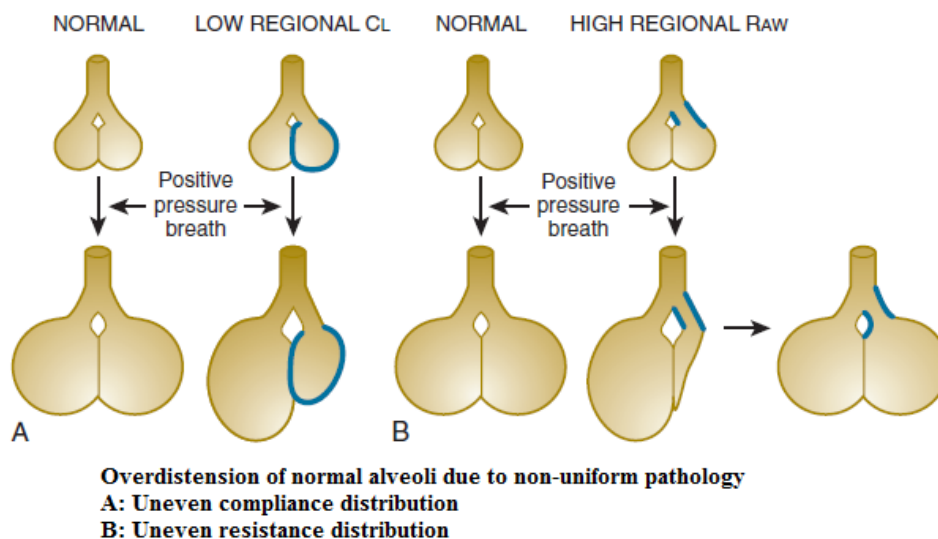
Acute Respiratory Distress Syndrome / Acute Lung Injury

Lung injury due to a variety of pulmonary and extra-pulmonary causes presents with patchy infiltrates and is included under the heading of ARDS/ALI. There is an oxygenation defect due to intrapulmonary shunting, which can be estimated by the $\text{PaO}_2/\text{FiO}_2$ ratio; with a ratio < 200 mmHg designated as ARDS and ratio 200-300 mmHg defined as ALI. Depending on the severity, these patients may require ventilator support till the lungs recover from the primary insult which caused alveolar damage. It is important to ensure that this ventilatory support does not itself produce further lung injury by damaging the remaining normal alveoli.

The pathophysiology of these conditions involves increased alveolar permeability with patchy atelectatic areas as well as more normal air filled alveoli. On the macro scale, the lung appears stiff with a low compliance, but there is considerable heterogeneity with different alveoli having a range of compliance values and time constants. It is as if there is an atelectatic lung and a normal lung connected in parallel. Thus, a relatively normal tidal volume is forced into the normal alveoli which add up to much less than a normal lung – the much smaller or so-called “baby lung”, overdistending the alveoli and causing volutrauma.

Thus the ventilator settings for ARDS include a low tidal volume (6 ml/IBW) keeping the $P_{\text{plat}} < 30$ cmH₂O; and FiO_2 –PEEP combinations as per the ARDS network protocol, aiming for a PaO_2 of 55-80 mmHg and SaO₂ of 88-95%. Pressure targeted modes of ventilation give better distribution of tidal volume compared to volume targeted modes but there is no conclusive evidence that these give better results as long as the tidal volumes and plateau pressures are kept low. I:E ratio is kept at 1:2 to 1:3 to start with, but severe oxygenation defect may benefit from I:E ratio of 1:1 or even higher – inverse ratio ventilation.

Ventilator modes which allow spontaneous breathing such as APRV and Bilevel PAP have been shown to improve oxygenation and hemodynamics, help lung recruitment and also reduce the need for sedation. Recruitment manoeuvres, which involve the application of sustained positive pressure of 40-50 cm H₂O followed by PEEP, have been shown to improve oxygenation. This approach is referred to as “open the lung and keep it open!”



Abdominal compartment syndrome

The elevated intra-abdominal pressure and cephalad displacement of the diaphragm reduce the FRC. The compliance of the respiratory system is greatly reduced, though lung compliance may be normal. Alveolar volume is determined by transmural pressure and not the absolute alveolar pressure. These factors influence ventilator settings in these patients.

Intra-abdominal pressure raises pleural pressure but since the pleural pressure is not routinely measured, transmural pressure across the alveolar wall may be estimated by the following formula:

$$P_{tm} = P_{plat} - IAP/2$$

Volutrauma is mainly related to alveolar over-distension, and hence, the inspiratory pressure may be safely increased as long as the transmural pressure is kept below 35 cm H₂O. Moderate PEEP (10 cm H₂O) has been shown to increase LV afterload produced by IAH. Recruitment manoeuvres in these patients need higher inflation pressures (40 + IAP/2 cm H₂O).

Hemodynamically, filling pressures (CVP and PCWP) are elevated in IAH, hence other techniques should be used for preload assessment, such as esophageal Doppler, echocardiography and pulse contour analysis. Monitoring the extravascular lung water with techniques such as PICCO is also helpful.

Heart failure / Pulmonary oedema

Many patients with congestive heart failure respond to noninvasive ventilation, either simple CPAP or BiPAP. These modes help by increasing FRC and opening the alveoli. Fluid is shifted out of the alveoli and ventilation/perfusion balance is improved. Oxygen cost of breathing is reduced by moving the patient onto a more favourable position on the compliance curve and by unloading the respiratory muscles. PEEP is transmitted to the left ventricle, reducing transmural pressure and afterload, and thus improving performance.

For patients who need intubation, ventilator settings are similar to those used for ARDS, namely a low tidal volume (6 ml/IBW) keeping the P_{plat} <30 cmH₂O; and FiO₂-PEEP combinations aiming for a PaO₂ of 55-80 mmHg and SaO₂ of 88-95% and relatively high respiratory rates. Hypotension often follows intubation and ventilation; this should be managed by a combination of fluid therapy and manipulation of cardiac contractility and peripheral resistance.

Intracranial pathology

Mechanical ventilation affects intracranial dynamics in many ways. Raised intrathoracic pressure can raise ICP by direct transmission and by lowering CPP due to fall in venous return and cardiac output. However, this effect is relatively modest, and most patients with intracranial pathology tolerate IPPV and PEEP well. In patients with ARDS as well as intracranial hypertension, recruitment manoeuvres which would be expected to increase ICP actually produced the opposite effect because of improved gas exchange and CO₂ elimination.

CO₂ has a powerful effect increasing cerebral blood flow, but the effect is transient and wears off over a period of hours. Hyperventilation is now recommended in the context of acute intracranial hypertension, only for temporary reduction of ICP while other measures become effective. At other times, normocapnia should be maintained, since excessive hypocapnia predisposes to cerebral ischaemia due to vasoconstriction.

Traditionally, ventilatory strategy in the presence of intracranial pathology focused on airway protection, optimizing oxygen delivery to the brain, careful control of CO₂ and maintaining the lowest possible intrathoracic pressure. This method used large tidal volumes, high FiO₂, low or zero PEEP, fluids and vasopressors to maintain CPP.

However, it is now recognised that volutrauma and atelectrauma caused by this type of settings predispose to ALI/ARDS as well as extrapulmonary adverse effects on the kidney, liver, gut and other organs; especially if they have been “primed” by the initial brain injury. The concern about using lung-protective ventilation is that it may result in hypercapnia, which is deleterious in the presence of intracranial hypertension. But it is possible to apply a lung protective strategy without causing hypercapnia, and low tidal volumes (6 ml/IBW), moderate PEEP and relatively high respiratory rates should be used in these patients.

Neuromuscular Respiratory Failure

Patients who need ventilator support for neuromuscular disease have relatively normal lungs to begin with. Tidal volumes of 8-10 ml may be given initially, with PEEP of 3-5 cm H₂O, keeping the P_{plat} below 30 cm H₂O. Volume or pressure targeted modes may be used, but pressure targeted modes may be more comfortable for the patient. Dual modes such as PRVC or autoflow are helpful in ensuring adequate ventilation even in pressure targeted modes.

Non-invasive ventilation

This term refers to providing ventilatory support without intubation, via a nasal mask, full face mask or a helmet. It requires an alert, cooperative patient with airway protective reflexes. Other contraindications are GI bleeding, severe hemodynamic instability and airway obstruction (other than obstructive sleep apnoea).

There are several ways of providing NIV. Simple CPAP or continuous positive airway pressure can be given with a high flow of oxygen, a well fitting mask and a PEEP valve, without a ventilator. Dedicated NIV ventilators are available which can give CPAP or BiPAP (bi-level positive airway pressure). BiPAP is essentially pressure support ventilation in which EPAP represents PEEP and IPAP is the level of pressure support. These specialty ventilators have fewer options but are more tolerant of leaks in the circuit.

Most of the critical care ventilators nowadays have a NIV option and thus can be used for NIV. The most common mode employed is CPAP with pressure support. Some of these ventilators can also provide proportional assist ventilation or PAV. Though volume modes can be used for NIV, pressure support and PAV are better tolerated by patients and are more leak-tolerant than volume modes.

Initial ventilator settings are 5 cm H₂O PEEP or EPAP and 10 cm H₂O pressure support or IPAP. The aim is to achieve tidal volumes of 5-7 ml/Kg, respiratory rate below 25/min and SaO₂ above 90%. Serial blood gas analysis and close clinical monitoring are essential for success with NIV. There should be a low threshold for moving to invasive ventilation, should the patient deteriorate.

Exacerbation of COPD and cardiogenic pulmonary edema are the two conditions well suited for NIV and improvement may be seen within 1-2 hours of starting treatment. Reduction in intubation rates and mortality with NIV has been seen in both these conditions. NIV can also act as a bridge support

after early extubation, since the rates of extubation failure may be as high as 10-20%. This is especially so in patients with underlying COPD.

Other conditions where NIV is beneficial are:

- Immuno-compromised patients – solid organ transplants, febrile neutropenia
- Asthma
- Postoperative patients
- Rib fractures, lung contusion
- Advanced malignancy (do not intubate status)
- ARDS – in less severely ill patients
- SARS – able to avoid intubation in 70% patients. Useful in hypoxemic patients with low APACHE II scores

Weaning from Ventilatory Support

Planning for separating the patient from the ventilator should start right from the time of institution of mechanical ventilation. To this end, the modes used should generally maintain spontaneous respiratory activity, as well as optimize other factors such as fluid balance, nutrition, electrolyte and metabolic status. The two major weaning strategies are either using a so-called weaning mode and step-wise reduction of ventilator support, or alternatively daily assessment of possibility of ventilator withdrawal. An evidence based task force has recommended the latter approach.

- i. The patient is assessed daily for readiness for a spontaneous breathing trial. This also involves daily interruption of sedation. The primary indication for ventilation should have resolved and patient should be alert. Pulmonary gas exchange should be adequate, with $\text{PaO}_2/\text{FiO}_2$ ratio >200 , $\text{FiO}_2 <0.5$ and $\text{PEEP} <5-7$ cm H_2O . Hemodynamics should be stable without high levels of inotropic support. The patient should be able to initiate spontaneous breaths.
- ii. The spontaneous breathing trial is performed (using T-piece, CPAP or pressure support of 5 cm H_2O) for 30 -120 min, monitoring ventilator pattern, gas exchange, hemodynamics, subjective comfort.
- iii. Patients who pass this trial may be extubated. Those who fail the trial should be put back on a stable, non-fatiguing mode should be instituted till the next SBT. Modes of partial support should provide adequate muscle unloading by using sensitive and responsive triggering systems and an unrestricted flow pattern which meets the patient's demands.

Conclusion

Ventilatory settings have profound effects on the outcome; and the interaction of each condition with the effects of the different modes of ventilation should be kept in mind so as to avoid further iatrogenic damage to the lungs and other organs,

Abbreviations:

Auto-PEEP	Intrinsic pressure
A/CMV	Assist control mode
ALI	Acute lung injury
ARDS	Acute respiratory distress syndrome
BiPAP	Bi-level positive airway pressure
COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
CPP	Cerebral perfusion pressure
CVP	Central venous pressure
EPAP	Expiratory positive airway pressure
FiO ₂	Inspired oxygen concentrations
FRC	Functional residual capacity
IAH	Intra-abdominal hypertension
IAP	Intra-abdominal pressure
IBW	Ideal body weight
ICP	Intracranial pressure
I:E ratio	Inspiration to expiration ratio
IAP	Intra-abdominal pressure
IPAP	Inspiratory positive airway pressure
NIV	Non-invasive ventilation
P _{AB}	Abdominal pressure
P _{ALV}	Alveolar pressure
P _{AO}	Airway pressure
PaO ₂	Arterial partial pressure of oxygen
PAV	Proportional assist ventilation
PCV	Pressure controlled ventilation
PCWP	Pulmonary capillary wedge pressure
PEEP	Positive end-expiratory pressure
PiCCO	Pulse induced contour cardiac output
PIE	Pulmonary interstitial emphysema
P _{PL}	Pleural pressure
PRVC	Pressure regulated volume control
PSV	Pressure support ventilation
P _{tm}	Transmural pressure
SARS	Severe acute respiratory syndrome
SaO ₂	Oxygen saturation
SBT	Spontaneous breathing trial
SIMV	Synchronised intermittent mandatory ventilation
VCV	Volume control ventilation
V/Q:	Ventilation/perfusion

