Principles of artificial ventilation

Tim Gould
J M A de Beer

Abstract

Ventilators are commonly used in the operating theatre and in the ICU to deliver mechanical ventilation to the lungs. In the operating theatre, ventilation is given to anaesthetized and often pharmacologically paralysed patients with predominantly normal lungs. These ventilators are relatively simple and are designed to deliver varying concentrations of oxygen, air, nitrous oxide and volatile agents to patients through an anaesthetic circuit. In the ICU ventilators are more sophisticated and provide respiratory support to patients with respiratory failure. Respiratory failure is a state in which the pulmonary oxygen uptake is so severely disturbed that the supply of oxygen to and/or removal of carbon dioxide from the tissues is inadequate. Respiratory failure can be caused by relative hypoventilation, characterized by an increase in arterial carbon dioxide tension, increased work of breathing or failure of diffusion at the alveolar–capillary membrane, characterized by decreased arterial oxygen tension. Patients with respiratory failure may be difficult to ventilate and the intensivist must use his or her knowledge of normal respiratory physiology, the pathophysiology of acute respiratory distress syndrome (ARDS), asthma and chronic obstructive pulmonary disease (COPD), and the results of clinical trials to meet this challenge.

Keywords ARDS; COPD; mechanical ventilation; respiratory failure; respiratory physiology

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Tim Gould, FRCP, FRCA, is Consultant in Intensive Care at the Bristol Royal Infirmary. He qualified from the University of Bristol and trained in general medicine, anaesthetics and intensive care. His research interests include splanchic blood flow in intensive care patients and weaning from ventilation.

J M A de Beer, FRCA, is Consultant in Intensive Care at the Royal Cornwall Hospital in Truro, Cornwall. He qualified from the University of the Orange Free State, South Africa and trained in anaesthesia, medicine and intensive care in South Africa, and the South West of England. His main interests are ventilatory support in ARDS, difficult airway management, and simulation technology.

In the ICU, ventilators are more sophisticated and provide respiratory support to patients with respiratory failure. Respiratory failure is a state in which the pulmonary oxygen uptake is so severely disturbed that the supply of oxygen to and/or the removal of carbon dioxide from the tissues is inadequate. Respiratory failure can be caused by relative hypoventilation, characterized by an increase in arterial carbon dioxide tension, or failure of diffusion at the alveolar–capillary membrane, characterized by decreased arterial oxygen tension.

Classification of ventilators

Numerous classifications have been suggested and include classifications by Ward and Mapleson. Classifications refer to the following elements.

Control describes how the ventilator delivers flow to the patient. In volume-controlled ventilators the rate of flow delivered is constant, the tidal volume is targeted, with a variable pressure delivered relative to compliance of the lung. In pressure-controlled ventilators the rate of flow delivered decreases through the breath to maintain the targeted pressure at the peak inspiratory pressure. The tidal volume delivered is determined by the compliance of the ventilated lung.

Cycling determines how the ventilator switches from inspiration to expiration. Time cycling is used in pressure-controlled ventilation. Flow-cycling is used in pressure-support ventilation, where a reduction of the peak inspiratory flow cycles the ventilator into expiration. Volume cycling is used in volume-controlled ventilation. The ventilator cycles to expiration when a set tidal volume has been delivered. If an inspiratory pause is added the breath will be classified as both volume and time cycled.

Triggering is how inspiration is initiated in association with patient breaths. Ventilators may be triggered by changes in pressure, flow or by a preset time interval having elapsed.

• Pressure: the ventilator delivers a breath when the baseline pressure decreases during the patient’s inspiratory effort.
• Flow: some ICU ventilators deliver a constant background flow throughout the respiratory cycle (flow by). A change in this constant flow, caused by patient inspiration is detected at the flow sensor in the expiratory limb. This triggers the ventilator to increase the flow and a breath is delivered to the patient. Flow triggering reduces the work of breathing when compared with pressure triggering because there is always some background gas flow from the patient and no delay in inspiratory valve opening.
• Time: the ventilator cycles at a frequency determined by the respiratory rate or the ratio of inspiration to expiration (I:E).

Volume-controlled ventilation and pressure preset ventilation

Volume-controlled mechanical ventilation is delivered with a constant inspiratory flow, resulting in an increasing airflow pressure through inspiration. To maintain this fixed rate of gas flow the pressure must rise through inspiration. The actual preset tidal volume remains constant whilst lung compliance and resistance change. The inspiratory flow rate alters the velocity with which gas flow is delivered (Figure 1). Ventilation with a high inspiratory flow delivers the pre-selected tidal volume more quickly. If the ventilator is time-cycled between inspiration and expiration and the tidal volume has been delivered before all the time allowed for inspiration has elapsed, an inspiratory pause will occur and the pressure will drop below the

Figure 1

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peak inspiratory pressure. There is no fresh gas flow during this inspiratory pause. High inspiratory flow during volume-controlled ventilation has a few detrimental effects on lung ventilation (Table 1). Therefore, low inspiratory flow rates should be used when possible to keep the peak ventilatory pressure as low as possible. This will ensure more homogeneous ventilation. The risk of lung injury can be reduced by using pressure-limited ventilation (Figure 2).

In old ventilators, pressure limitation completely stops the inspiratory flow, resulting in a reduction in target tidal volume. In more modern ventilators, when the pressure limit is reached the flow decelerates to maintain the peak pressure at the pressure limit for the rest of the breath. This ensures the tidal volume delivered is as close to the target tidal volume as possible for the set pressure limit. A pressure limit of 30–35 cm H$_2$O is appropriate in adults.

Pressure-controlled ventilation rapidly achieves a fixed pressure throughout the breath by the delivery of a decelerating inspiratory flow pattern (Figure 3). The result is a tidal volume that will vary with lung compliance and resistance. For example, if there is an increase in airways resistance, or reduction in lung compliance, the delivered tidal volume will decrease and hypoventilation will result. Pressure-controlled ventilation is usually closely monitored with alarms set for a minimal acceptable tidal, minute volume or both.

**Interaction between ventilated breaths and the patient’s inspiratory efforts** – various modes exist and only the more common modes are described below.

- Controlled mandatory ventilation (CMV) with no allowance for spontaneous breathing. This mode is the most commonly used in the operating theatre during routine anaesthetics.
- In synchronized intermittent mandatory ventilation (SIMV), controlled breaths are delivered to a preset respiratory rate separate from the spontaneous breaths. This can be a volume- or pressure-controlled breath.
- In assist-controlled ventilation mode, triggered spontaneous breaths are assisted identically to the controlled breaths.
- In pressure-support ventilation, spontaneous patient breaths trigger a set amount of pressure to assist the breath.
• Biphasic positive airway pressure (BIPAP) is a mixture of spontaneous breathing and time-driven, pressure-controlled ventilation (Figure 4). This system alternates between two adjustable pressure levels of continuous positive airway pressure (CPAP). Spontaneous breaths are possible at both pressure levels at all times. Cycling between the two levels produces gas flow and a resulting mechanical breath.

Normal respiratory physiology and the effects of mechanical ventilation
Oxygenation occurs by diffusion of oxygen across the alveolar capillary membrane. Its prime determinants are the inspired oxygen concentration and the mean airway pressure. The mean airway pressure is modified by changes in peak airway pressure, positive end-expiratory pressure (PEEP) and the I:E ratio. Removal of carbon dioxide requires movement of air in and out of the lung. Its prime determinant is minute volume, whatever changes are made to the ventilator. Partial pressure of arterial carbon dioxide (PaCO$_2$) is regulated at about 5.3 kPa. A normal tidal volume is between 350–500 ml or 5–7 ml/kg in an adult. The normal respiratory rate is 10–15 breaths per minute and depends on the requirement for carbon dioxide elimination.

The total volume of the respiratory system or total lung capacity when expanded by voluntary effort is 5–6 litres in the average person.
adult. The total alveolar volume can be divided into that which can be measured at the lips (vital capacity) and that which remains in the lungs after maximal expiration (residual volume).

The functional residual capacity (FRC) is the combination of expiratory reserve volume and residual volume (Figure 5).

Resistance
Resistance is defined as the pressure difference between the beginning and end of a tube divided by the flow of gas volume per unit time. In the lung it is the difference between atmospheric pressure and alveolar pressure. The tracheobronchial tree is a dynamic system of cartilaginous structures that can distend or narrow depending on the forces exerted on them. The smaller, more distal airways are kept open by the balance of the intrapleural pressures and the elastic recoil of the lung. During inspiration, elongation of the elastic pulmonary fibres increases the elastic retraction pressure. As the bronchioles are stretched by the stronger radial pull, the bronchial resistance decreases. With expiration the elastic recoil of the lung increases, the bronchiolas become narrower and the flow resistance increases. These changes in the flow characteristics during the respiratory phases explain why the expiratory phase is slightly longer than the inspiratory phase. Dynamic compression of the small airways occurs when the intrapleural pressures increase to about 40 cm H₂O during forced expiration. When the intrapleural pressures are considerably greater than the intraluminal pressures, narrowing or closure of the bronchioles occurs (Figure 6). If airway closure is premature the closing volume will encroach on FRC.

Closing volume
Closing volume is the volume of lung inflated when small airways in the dependent parts of the lung begin to collapse during expiration. In health, closing volume is normally less than FRC and accounts for the residual volume (RV) at the end of maximal expiration. If closing volume encroaches on FRC, airway closure may occur during normal expiration. Ventilation to areas distal to the closure therefore decreases, with the ventilation/perfusion relationship (V/Q) worsening. Closing capacity is defined as the sum of closing volume and residual volume. FRC usually remains greater than closing volume, but closing volume slowly increases with age. Closing volume exceeds FRC in an individual aged 45 years in the supine position and in an individual aged 60 years in the erect position. The reduction in FRC during general anaesthesia often reduces FRC below closing volume in young individuals. Therefore, young patients may have increased V/Q mismatch and a reduction in arterial oxygenation.

Compliance
Compliance describes the elastic properties of various parts of the respiratory system. Compliance represents a volume change per unit change in pressure (200 ml/cm H₂O in the normal lung). The total respiratory compliance consists of combined lung and chest wall compliance and is normally 70–80 ml/cm H₂O. It can be divided into two components.

- Static compliance (alveolar stretchability) is measured when there is no flow activity at the end of inspiration (Figure 7).
- Dynamic compliance describes the change in volume as pressure changes during actual gas flow through the respiratory cycle (Figure 8).

The static compliance curve can be used to select the ideal level of PEEP for a patient in the ICU (Figure 7). This level of PEEP corresponds to a point on the favourable part of the pressure–volume curve for alveoli, maximizing oxygenation and minimizing overdistension. PEEP should be increased to the critical opening pressure for most of the alveoli (the lower inflection point). At this point most of the collapsed alveoli will open and the lung will become more compliant. Over-distension of alveoli will occur at the flatter, top part of the curve (upper inflection point) and the lung will become less compliant. The advantages and disadvantages of using PEEP are shown in Table 2.

Time constants
The time constant of a ‘lung compartment’ is a function of its resistance and compliance. The time constant (τ) expresses how quickly a compartment can react to an alteration of pressure
and gives an indication of the filling or emptying velocity of a lung compartment. The lung consists of a large number of compartments with widely variable time constants. This heterogeneity is often exaggerated in lung disease (e.g. pneumonia, pulmonary fibrosis). The more inhomogeneous the lung ventilation, the wider the spectrum of regional time constants. This causes variation in the filling and emptying periods as well as the filling volumes for individual compartments. At a given pressure, a compartment with a high resistance and good compliance fills slowly, resulting in a large volume (e.g. asthma). Conversely, a compartment with a poor compliance and low resistance will fill quickly, resulting in a small volume (e.g. pulmonary fibrosis).

During volume-controlled ventilation with an end-expiratory pause, *pendelluft* arises between compartments with different time constants. The greatest part of the inspired volume is taken in by the compartment with the quickest time constant. During the end-inspiratory pause the lung redistributes its volume into various compartments, depending on the different time constants of the alveolar units. Inhaled gas that has undergone gas exchange now flows from the quicker to the slower compartments. This is not important in the normal lung, but it may play a role in reducing overall lung compliance and oxygenation in chronic obstructive pulmonary disease (COPD).

In acute respiratory distress syndrome (ARDS) it may be difficult for inflated lung units to equilibrate with stiff lung units during an inspiratory pause (Laplace equation $P = 4\pi T/r$, where $P$ is the pressure, $T$ is the surface tension and $r$ is the radius of the unit). The effects of surface tension will become important when surfactant is depleted by disease and small lung units empty into larger ones.

**Work of breathing**

Work of breathing (Figure 8) is the work required to move the chest wall and lungs during inspiration and expiration. In this context, it is most convenient to measure work as the product of pressure and volume. This can be explained by considering the dynamic pressure–volume relationship in quiet inspiration and expiration. In spontaneous respiration, the work of breathing involves two components, namely the elastic work needed to overcome elastic forces and the work needed to overcome the non-elastic forces or the flow resistance of the airways.

The area on the right of the gradient line in Figure 8 represents the work expended to overcome the airway resistance during inspiration. The area to the left of the gradient line represents the work expended to overcome elastic forces during passive expiration in the normal lung. It is important to note that more work is necessary to overcome the flow resistance in obstructive
ventilation disorders, particularly if positive intrapleural pressures are generated in expiration. Conversely, in restrictive disorders more elastic respiratory work is required during inspiration. In the intensive care setting respiratory work is further increased in intubated patients because of the increased flow resistance of the endotracheal tube and ventilator tubing.

Ventilation/perfusion relationship
The V/Q relationship varies throughout the lungs. During spontaneous ventilation the distribution of ventilation and perfusion is not uniform throughout the lungs. In the normal setting ventilation and perfusion are matched adequately throughout the lung with the bases receiving substantially more of both than the apices. The distribution of perfusion throughout the lung is partly due to the effects of gravity and partly due to anatomical differences with improved flow to the bases. In the upright position, the perfusion pressure at the base of the lung is equal to the mean pulmonary arterial pressure plus the hydrostatic pressure between the pulmonary artery and lung base. At the apices the hydrostatic pressure difference is subtracted from the pulmonary artery pressure with a resulting low pressure, which may fall below the alveolar pressures, leading to vessel compression and intermittent cessation of blood flow. This gravity-induced distribution pattern is explained by the West three zone model shown in Figure 9.

The distribution of ventilation across the lung is related to the position of each area on the compliance curve at the point of FRC. Because the bases are on a more favourable part of the compliance curve than the apices, they gain more volume change for the pressure change applied, and thus receive a greater degree of ventilation. Although the inequality between bases and apices is less marked for ventilation than perfusion, overall there is still good V/Q matching and efficient oxygenation of blood passing through the lungs. Disturbance of this distribution can lead to V/Q mismatching (Figure 10). For an area of low V/Q ratio the blood flowing through it will be incompletely oxygenated, resulting in a decreased level of arterial oxygenation. Providing some ventilation is occurring in an area of low V/Q, the hypoxaemia can normally be corrected by increasing the fraction of inspired oxygen (FiO₂).

V/Q mismatch occurs very commonly during anaesthesia because the FRC falls, leading to a change in the position of the lungs on the compliance curve. The apices therefore move to the most favourable part of the curve, whilst the bases are located on a less favourable part of the curve (at the bottom).

Alveolar dead-space
Dead-space is an area of lung where no significant gas exchange occurs. The physiological dead-space equals anatomical plus alveolar dead space. Anatomical dead-space includes the conducting airways not lined with respiratory epithelium. Alveolar dead-space describes ventilated lung normally contributing to gas exchange, but not doing so because of impaired perfusion. The ratio (Vd/Vt) of dead-space volume (Vd) and the volume of ventilation to perfused alveoli (Vt) is normally 0.3. The Vd/Vt ratio increases in conditions such as COPD. This is because of increased anatomical dead-space from lung parenchymal destruction. Beyond a Vd/Vt ratio of 0.7–0.8, spontaneous breathing is no longer possible because the increased work of breathing gives rise to more carbon dioxide than can be exhaled.

The respiratory effects of mechanical ventilation
Lung volume increases above end-expiratory volume on inspiration. During spontaneous inspiration intrathoracic pressure decreases, but on positive pressure ventilation inspiration occurs by an increase in intrathoracic pressure. The distribution of ventilation and perfusion in the lung varies with these pressure and volume changes.

Alterations in the distribution of ventilation – after induction of anaesthesia in the supine position non-independent parts of the lung are more compliant than the dependent zones. Positive-pressure ventilation will preferentially ventilate these non-dependent zones. This phenomenon becomes further accentuated if the lungs have increased density, as in ARDS or left ventricular failure.

Alterations in distribution of perfusion – below a mean intrathoracic pressure of 30 cm H₂O, distribution of perfusion remains normal during mechanical ventilation. In non-dependent areas of the lung with relatively lower capillary pressure, blood

**West three zone model: the gravity-induced distribution pattern of blood flow in the lung**

[Figure 9]
vessels are particularly susceptible to compression, as mean intrathoracic pressure increases above 30 cm H₂O. Compression of the alveolar capillaries in these preferentially ventilated zones reduces blood flow, worsens V/Q mismatch and Vd/Vt ratios. This can be further exaggerated by PEEP.

The systemic effects of mechanical ventilation

Heart–lung interactions
Heart–lung interactions result from changes in intrathoracic pressure and lung volume that affect preload, contractility and afterload.

Intrathoracic pressure: the heart is a pressure chamber within a pressure chamber. There is a pressure gradient for venous return between the venous reservoirs and right atrial pressure. An increase in mean intrathoracic pressure (transmitted through the compliant right atrial wall) increases right atrial pressure and impedes venous return.

Preload and contractility – right ventricle (RV) output and left ventricle (LV) output are in series; thus, changes in RV preload will alter LV preload and therefore end-diastolic volumes. Left ventricular end-diastolic volume (LVEDV) is the main determinant of the position of the LV on the Frank–Starling curves. If end-diastolic volume falls, cardiac output also falls. Some of these changes can be modified by infusion of fluid (to increase central venous pressure) at the start of mechanical ventilation. In spontaneous ventilation, the decrease in mean intrathoracic pressure on inspiration increases venous return, LVEDV and stroke volume.

Lung volumes: changes in lung volume affect autonomic tone and pulmonary vascular resistance (PVR). In the normal range (Vt < 10 ml/kg) heart rate increases as vagal tone decreases (sinus arrhythmia). PVR increases if lung volume decreases below normal end-expiratory levels (e.g. ARDS, pneumonia). As lung volume decreases, lung interstitial radial tone decreases, small airways collapse, alveolar hypoxia follows, and hypoxic pulmonary vasoconstriction (HPV) increases (alveolar recruitment that increases FRC may reverse this process). When lung volume increases there will be a point at which increased alveolar pressure relative to capillary pressure collapses small vessels and also increases PVR (e.g. emphysema, hyperinflation, gas trapping).

Contractility and afterload – as PVR increases, changes in pulmonary hypertension and RV ejection result in a fall in cardiac output. If the right ventricular end-diastolic volume (RVEDV) becomes significantly raised, bulging of the intraventricular septum into the left ventricular cavity can occur, and left ventricular compliance is reduced (ventricular interdependence: changes in RVEDV inversely change LV compliance). LVEDV and stroke volume will fall in a manner analogous to cardiac tamponade. Increased right ventricular wall tension can compromise right ventricular coronary perfusion and leads to a further reduction in cardiac output.

Very large tidal volumes can restrict absolute cardiac volume by direct compression of the heart (biventricular free wall can collapse into the septum) and limit end diastolic volumes.

Patients with gas trapping: hyperinflation can occur because of premature airway closure on expiration (e.g. emphysema, acute COPD, asthma) or when expiratory time is too short to allow complete exhalation-gas trapping (inverse ratio ventilation). Cardiac output may fall because of an exaggeration of the effects described above. The immediate manoeuvre if cardiac output is falling acutely is to allow a longer expiratory time and to consider the use of bronchodilator therapy, reduced tidal volumes, and the judicious use of PEEP or CPAP to splint airways open in expiration.

Patients with heart failure: in patients with existing heart failure, the increase in intrathoracic pressure and reduction of preload can increase cardiac output. If RVEDV is increased (e.g. associated with COPD, cor pulmonale or excessive negative pressures on inspiration in spontaneous respiration) the reduction in preload by intermittent positive-pressure ventilation can reduce RVEDV, and hence through ventricular interdependence an improvement in LV compliance and LVEDV will follow.
Afterload reduction can also occur because raised intrathoracic pressure is transmitted through the walls of the ventricle and thoracic aorta, causing a reduction in the work of the LV to maintain a specific systolic arterial pressure (i.e. pressure in the ventricle will be supplemented by the actual intrathoracic pressure).

The ultimate cardiovascular response to acute respiratory failure is a balance of the basal cardiovascular function of the patient, the respiratory pathophysiology and the ventilatory pattern.

**Kidney**
Mechanical ventilation with positive airway pressure leads to a reduction of renal water and sodium excretion. The increase of intrathoracic pressures results in a decrease in cardiac output and mean arterial pressure. Low pressure baroreceptor discharge leads to increased sympathetic activity and a rise in plasma vasopressin concentration (low urine output). The reduction in renal perfusion and increase in renal sympathetic activity stimulate the renin–angiotensin system. Angiotensin II formation stimulates aldosterone production, with a resultant increase in reabsorption of water and sodium. Reduced venous return and low stretch of the right atrium decrease atrial natriuretic peptide (ANP) release, contributing towards increased sodium reabsorption and increased diuresis. The higher mean intrathoracic pressure increases venous pressure and causes some kidney congestion, an additional problem when associated with low renal perfusion pressure from reduced cardiac output. These effects are not significant in healthy individuals but they may exacerbate the situation if associated with other comorbidities.

**Liver**
Hepatic blood flow depends on a balance of flow through the hepatic artery and portal circulation. The reduction in cardiac output associated with intermittent positive-pressure ventilation leads to a proportional reduction in hepatic blood flow. In addition, raised mean intrathoracic pressure leads to increased hepatic venous congestion, which will have a deleterious effect on portal vein blood flow (a relatively low pressure system). Hepatic cellular function may be compromised, especially if associated with other comorbidities.

**Cerebrovascular side effects**
An increase in intrathoracic pressure and use of PEEP is accompanied by an increase in intracranial pressure. The raised mean intrathoracic pressure obstructs venous return from the jugular veins. This, combined with the fall in cardiac output, leads to a fall in cerebral perfusion pressure. In the healthy brain, autoregulation ensures adequate cerebral perfusion despite changes in arterial pressure and intracranial pressures. This autoregulation may be disrupted by cerebral pathologies with a risk of hypoperfusion. Therefore, PEEP should be used with caution in these patients.

**Ventilatory strategies for special conditions**

**Asthma and COPD**
Asthma is an acute generalized airway obstruction syndrome associated with bronchospasm caused by the hyper-reactivity of airways and the release of inflammatory mediators such as histamine.

COPD is a slow, progressive disorder characterized by airway obstruction with a reduced forced expiratory volume in 1 second/forced vital capacity (FEV1/FVC). There is a gradual deterioration over time, with lung parenchymal destruction, an increased residual volume and hyperinflated chest. The diaphragm is flattened and intercostal muscles elevated to a mechanically disadvantageous position. Generally, patients breathe with small tidal volumes. A high Vd/Vt ratio (high PaCO2) and a significantly reduced inspiratory reserve volume (hinders coughing) necessitates an increase of minute volume by a faster respiratory rate. As the condition progresses, expiration no longer becomes passive, but requires positive pressure to overcome bronchial resistance. This positive pressure at the end of expiration causes premature small airway collapse and incomplete emptying of the lung. Intrinsic PEEP (PEEPI) develops. This is a measure of the adequacy of expiration, and relates to the amount of air trapping during the respiratory cycle. Before inspiratory flow begins, intrapleural pressure needs to be reduced by an amount equivalent to PEEPI. Increased inspiratory and expiratory muscular effort is needed to overcome PEEPI. Acute exacerbations of COPD can increase bronchial resistance further and accentuate the problem. Lung impairment is often progressive, but reversibility of the condition should be assessed with bronchodilator therapy or steroids in all patients.

Ventilatory strategies in asthma and COPD: the ventilator used should have graphical real-time displays of flow and pressure waveforms. The elimination of hypoxaemia can almost always be achieved by the short-term use of a high inspired oxygen concentration titrated to a partial pressure of oxygen in arterial blood (PaO2) of 8–10 kPa. The minute volume should be adjusted to pH and not by the PaCO2, to avoid over-ventilation with consequent alkali loss and reduced renal compensation. At the start of controlled ventilation in people with asthma, tidal volumes may need to be reduced to avoid high airway pressures and barotrauma. Enough time for expiration needs to be allowed to reduce gas trapping. Increased respiratory rate leads to a significant increase in PEEPI as a result of a reduced expiratory time. Therefore, a low respiratory rate and a I:E ratio of 1:1.5 or 1:2 should be chosen to achieve a flow pattern as seen in Figure 11.

Whether non-invasive positive-pressure ventilation (reduces mortality and incidence of intubation in management of COPD) or conventional ventilation is used, triggered spontaneous breathing (pressure support, assisted spontaneous breathing) is the optimal mode. The use of PEEP or CPAP requires particular care but can reduce inspiratory and expiratory work. On inspiration, PEEP reduces the negative inspiratory effort needed by the patient to overcome PEEPi before gas begins to flow. On expiration PEEP helps to splint open the airway and allow more complete exhalation (improved CO2 clearance). Optimally, PEEP or CPAP should be set below PEEPi (Figure 12).

Qualitative measurement of PEEPi during positive-pressure ventilation can be obtained by the end-expiratory hold control on the ventilator. This allows for equilibration of pressures between the alveoli and the ventilator, allowing an indication of
the total PEEP. The PEEPi can then be calculated by subtracting the dialled PEEPe from the total PEEP (Figure 13).

ARDS
ARDS is characterized by generalized pulmonary infiltration secondary to increased permeability, and results in interstitial and alveolar oedema. The ARDS is defined by: a PaO$_2$/FiO$_2$ of less than 200 mm Hg; bilateral interstitial infiltrates on a chest radiograph; no evidence of left ventricular failure with a pulmonary artery wedge pressure of less than 18 mm Hg; and a recognized cause of ARDS. Hypoxia is secondary to decreased compliance, increased pulmonary shunt and pulmonary hypertension. It is this pulmonary hypertension that causes an increase in microvascular pressure, resulting in increased capillary leak and interstitial oedema. It may also precipitate right ventricular failure. Poor lung compliance and attempts to ventilate with normal tidal volumes may lead to high airway pressures and possible volutrauma.

Ventilatory strategies in ARDS: the ventilator should have graphical, real-time displays of flow and pressure waveforms. Protective ventilation is key to a safe and effective management plan of a patient with ARDS. Ventilation strategies are aimed at preventing the detrimental effects of volutrauma. Strategies include pressure-controlled forms of ventilation with small tidal volumes (5–6 ml/kg) and high respiratory rates (>25 breaths/min) to prevent over-distension. When volume-controlled ventilation is used, gas is preferentially delivered to more compliant lung units, with a risk of over-distension. The application of pressure-controlled ventilation results in better gas distribution and less distension of non-compliant lung units (Figure 14).

Figure 11

Figure 12
Mean airway pressure (which corresponds with alveolar recruitment and improved PaO₂) should be increased. The mean airway pressure correlates with the area under the pressure curve and can be increased by several manoeuvres.

A higher mean airway pressure for a given peak airway pressure can be achieved with pressure-controlled ventilation. The normal alveoli are more susceptible to the effects of peak airway pressure (Figure 1). High airway pressures result in volutrauma and should be limited to less than 35 cm H₂O. If volume-controlled ventilation is used, it can be difficult to keep the peak airway pressure below this value in patients with non-compliant lungs.

With pressure-control modes it is possible to safely increase the mean airway pressure by prolonging inspiratory time, without a need to increase the peak pressure. A reduction in expiratory time can induce auto-PEEP (PEEPi) where a new breath is delivered before expiratory flow is complete. The remedy for this is a reduction in the respiratory rate followed by a reduction in the I:E ratio, until gas trapping disappears on the flow waveform. This results in improved oxygenation within the pressure limit without overstretched the alveoli (Figure 11). If gas trapping develops with pressure-controlled ventilation, a reduction in tidal volume with each subsequent breath follows. When volume-controlled ventilation is used for inverse ratio ventilation, the tidal volume is sustained with each breath, and peak airway pressure rises. This progressively worsens gas trapping. Volume-controlled ventilation should not be used in inverse ratio mode.

The optimal PEEP level should be set from the lower inflection point on a plot of the static pressure–volume curve (Figure 7). In ARDS secondary to diffuse, homogeneous lung injury, the PEEP level may be more than 12–15 cm H₂O. The static curve can be constructed with pressure-controlled ventilation using the peak pressure value, provided the gas flow ceases before the end of expiration, or using volume-controlled ventilation and plotting the plateau pressure (no flow) against the respective tidal volumes.
volumes. By randomly changing the size of the tidal volume every five breaths, a curve can be safely plotted in a compromised patient. Some commercially available ventilators can specifically construct a static compliance curve in addition to conventional dynamic loops. Higher levels of PEEP will ensure an FRC greater than the closing capacity, and reduce the tendency towards alveolar collapse, V/Q mismatch and hypoxaemia.

In ARDS associated with focal consolidation (secondary to pneumonia) new evidence suggests that high PEEP levels may be less beneficial and lead to over-distension of healthy lung units.

The principles of protective ventilation described above may lead to permissive hypercapnia (a higher than normal PaCO₂), particularly as an increased Vd/Vt ratio is associated with ARDS. If renal function is preserved HCO₃⁻ will normalize pH. The benefits of protective ventilation strategies may outweigh the detrimental effects of a higher PaCO₂ in adult critical care.

**Prone ventilation** can improve oxygenation in 50–75% of patients with ARDS. In the supine position ventilation is preferentially directed to the anterior part of the lung, but the optimal perfusion will be in the lung bases close to the diaphragm. In addition, the weight of the mediastinum and heart, together with heavy oedematous lung, collapses the lung posteriorly. When prone, the compliance of the anterior chest wall is increased, the posterior chest wall is already relatively non-compliant, and ventilation is preferentially directed to the bases where perfusion is optimal. The mediastinum and heart are also dependent, and lung previously compressed posterior to this can expand. The benefits are essentially from improved V/Q matching, decreased pulmonary dead-space fraction, and increased drainage of secretions. To date, prone ventilation has failed to improve outcome in adult patients with ARDS. However, a reduction in PaCO₂ may translate into an improved outcome in patients with ARDS ventilated in the prone position.

**High-frequency oscillatory ventilation (HFOV)** is a relatively new mode in adult practice. HFOV generates sub-dead-space tidal volumes at high rates (4–5/s) at a constant high mean airway pressure. Lung recruitment follows with reasonable clearance of CO₂. It is commonly used in neonates but there are few good adult studies.

**Other treatment modalities** include: inhaled nitric oxide; inhaled prostacyclin; continuous rotation; partial liquid ventilation; extracorporeal membrane; extracorporeal membrane oxygenation; and interventional lung-assist ventilation.

**Summary**

It is essential for both the anaesthetist and the intensivist to have a good understanding of the pathophysiology of the lung and knowledge of ventilator/lung interactions. Some important basic principles of ventilation are listed below.

- The pathophysiology of the lung varies with time, so ventilator settings need to be regularly reviewed and adjusted.
- A healthy lung can be damaged with inappropriate ventilator settings (a diseased lung may be more susceptible). Therefore, peak airway pressure should be below 35 cm H₂O, where possible.
- To minimize side effects, the physiological targets (PaO₂ and PaCO₂) do not need to be in the normal range.
- In the past PEEP has gone unnoticed. When possible flow patterns of ventilation should be monitored to detect the abnormality.

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**FURTHER READING**


