Positive end-expiratory pressure (PEEP) can be defined as artificial maintenance of supra-atmospheric pressure at the end of passive exhalation during controlled ventilation. Continuous positive airways pressure (CPAP) implies application of a preset positive pressure throughout the respiratory cycle (i.e. inspiratory and expiratory phases) in a spontaneously breathing patient.

Intrinsic PEEP (PEEPi; also called air trapping, dynamic hyperinflation or auto PEEP) results from premature termination of passive exhalation before the lung volume reaches functional residual capacity (FRC). This can be due to a short expiratory phase (inverse ratio ventilation), a slow expiratory flow (e.g. asthma) or both.

**Physiological basis for the use of PEEP and CPAP**

Alveoli and small airways have a tendency to collapse (Laplace’s law). Their integrity is maintained by structural and physiological mechanisms. Structural mechanisms include the elastic recoil of the thoracic wall, radial traction of elastic tissue of lung, alveolar interdependence and the presence of surfactant. Physiological mechanisms include coughing and intermittent sighing.

**Table 1 Common causes of relatively reduced functional residual capacity**

<table>
<thead>
<tr>
<th>Physiological</th>
<th>Peri-operative</th>
<th>Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children &lt; 4 years – supine</td>
<td>Induction of anaesthesia</td>
<td>Obesity</td>
</tr>
<tr>
<td>Adults &gt; 40 years – supine</td>
<td>Insertion of endotracheal tube</td>
<td>Restrictive chest wall disorder</td>
</tr>
<tr>
<td>Supine position: FRC is 33% less compared with sitting position</td>
<td>Breathing 100% oxygen: promotes atelectasis</td>
<td>ARDS</td>
</tr>
<tr>
<td></td>
<td>Abdominal surgery and/or packs</td>
<td>Cardiogenic pulmonary oedema</td>
</tr>
<tr>
<td></td>
<td>Thoracic surgery</td>
<td>Pulmonary fibrosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chest/abdominal trauma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Atelectasis due to retained secretions</td>
</tr>
</tbody>
</table>

**Functional residual capacity**

A number of factors can reduce FRC, sometimes even below the closing capacity (Table 1). This can result in airway closure, atelectasis, shunting and hypoxaemia. At appropriate levels, both PEEP and CPAP may increase the FRC above the closing capacity and reverse hypoxaemia.

**Pressure–volume relationship**

Figure 1 shows the typical pressure–volume relationship of a normal lung and the effect of change in compliance. This relationship produces an ‘S’ shaped curve with a lower and an upper inflection point; the gradient at any point on the curve reflects compliance. At lower lung volumes, compliance is lower because a critical pressure is required for opening the closed alveoli. Once this is achieved (i.e. at the lower inflection point [LIP], typically around 5 cmH₂O), an improvement in compliance is seen with a subsequent increase in the gradient of the curve. Normally, tidal volume breathing occurs at the steep part of the curve and is associated with optimal gas exchange with minimal work of breathing.

**Key points:**

- PEEP and CPAP are most effective when hypoxia is due to alveolar collapse
- CPAP significantly improves outcome in acute cardiogenic pulmonary oedema
- PEEP should be adjusted to ensure adequate oxygen delivery
- Watch for signs of barotrauma
- Cardiovascular support may be required

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Fig. 1 Diagrammatic representation of the relationship between unit lung volume and transpulmonary pressure (compliance curve). The decrease in compliance shifts the lower inflection point to the right and reduces the gradient of the curve. The solid lines show the pressure–volume excursion during inspiration.
The position of LIP and gradient of the pressure–volume curve may be changed in disease. For example, in ARDS LIP is shifted to the right and the gradient of the pressure-volume curve is reduced (Fig. 1). This suggests significant alveolar closure at the onset of inspiration during a tidal breath and is associated with poorer gas exchange and increased work of breathing. PEEP or CPAP, at a level just above LIP, would recruit alveoli and shift tidal breathing to the steeper portion of the pressure–volume curve resulting in improvement in gas exchange and reduction in the work of breathing. Thus, LIP on the expiratory limb of the pressure-volume curve can be used as a guide to the level of PEEP required in order to recruit alveoli.

**Indications for the use of PEEP and CPAP**

A well-documented indication for PEEP is augmentation of the partial pressure of oxygen in arterial blood ($P_{aO_2}$) in hypoxic patients ($P_{aO_2} < 7.5$ kPa on $60\%$ inspired oxygen), especially if hypoxia is due to alveolar collapse and refractory to an increase in inspired concentration of oxygen.

**Anaesthesia**

In patients who are at extremes of age (<4 years, >40 years), obese or have raised abdominal pressure, FRC often falls resulting in shunt and hypoxaemia. Upper abdominal surgery causes a postoperative restrictive defect in pulmonary function, reducing FRC by 30%, with similar consequences. This hypoxia can be corrected by PEEP and/or CPAP.

**Cardiogenic pulmonary oedema**

In addition to significant alveolar closure and reduction in compliance, large negative swings due to respiratory distress in pulmonary oedema increase left ventricular preload by increasing venous return. Also, they may increase the afterload by increasing effective left ventricular transmural pressure (and hence wall tension). CPAP and PEEP act to counteract these swings, thus reducing left ventricular preload and afterload. These measures do not reduce the amount of extravascular lung water, but they probably redistribute and accommodate it better by increasing lung volume, producing some clearing on chest X-ray and improvement in gas exchange.

The use of a non-invasive mask and nasal CPAP in patients with acute cardiogenic pulmonary oedema has reduced the need for intubation and ventilation and mortality. A cost-benefit study has shown a reduction in the need for ventilation from 35% to nil and a 5-fold decrease in costs when mask CPAP was introduced for all acute onset pulmonary oedema. In health, the work of breathing is about 5% of total oxygen delivery. In severe respiratory distress (e.g. pulmonary oedema), it can be 25–30%. Improvement in compliance produced by CPAP reduces the work of breathing and may prevent respiratory failure and the need for ventilation. Patients with chronic congestive cardiac failure may also benefit from long-term nocturnal nasal CPAP, with improved left ventricular function and respiratory muscle strength. However, high levels of CPAP and PEEP can increase pulmonary vascular resistance and hence right ventricular afterload, thus worsening right ventricular failure.

**Cardiothoracic surgery**

During thoracic surgery, one lung ventilation with an open chest can markedly reduce oxygenation. Applying PEEP to the dependent lung increases its FRC and ventilation by counteracting the effects of the elevation of the diaphragm on the dependent side and the weight of the mediastinum. However, PEEP should be applied carefully to the dependent lung as it may increase vascular resistance in the ventilated lung causing diversion of perfusion to the non-dependent lung; hence, increasing shunt and offsetting some of the improvement in oxygenation. The addition of CPAP to the uppermost lung will reduce the shunt and improve oxygenation but at the cost of some lung inflation which may interfere with surgical access.

After cardiopulmonary bypass, patients often suffer hypoxaemia due to atelectasis and re-perfusion pulmonary oedema. PEEP of 10 cmH2O certainly improves oxygenation. Elective use of mask CPAP has been shown to allow early extubation and, after single lung transplant, it reduces the incidence of reperfusion pulmonary oedema.

**Adult respiratory distress syndrome (ARDS)**

ARDS is the severest form of acute lung injury where FRC and compliance are reduced. Ventilation/perfusion ratio in some parts of the lung approaches zero (severe intrapulmonary shunting) resulting in severe hypoxaemia. At >30% shunt, an increase in inspired oxygen does not result in significant improvement in oxygenation. PEEP and CPAP can reduce the shunt by re-opening alveolar units, thus improving $P_{aO_2}$ for a given inspired oxygen concentration.

**Chronic obstructive pulmonary disease (COPD) and asthma**

Increased air trapping in patients with COPD or asthma can cause significant PEEPi. During inspiration, significant work is required by the inspiratory muscles to overcome PEEPi and
generate a subatmospheric alveolar pressure for breathing. Thus, PEEP increases the inspiratory load and work of breathing and causes dyspnoea. Application of CPAP may reduce work of breathing and dyspnoea in some patients with COPD by preventing the premature collapse of airways and air trapping.

In ventilated asthmatic or COPD patients, PEEPi can be >26 cmH2O. PEEPi increases with externally applied PEEP, short expiratory phases and large tidal volumes. The cardiovascular effects and risk of barotrauma of high levels of PEEPi are similar to externally applied PEEP and should be avoided.

PEEP in paralysed/sedated patients can be measured by occluding the expiratory port at the end of expiration, allowing a few seconds for equilibration and measuring the pressure. On-going expiratory flow at the termination of the expiratory phase, expiratory breath sounds interrupted by inspiratory breath sounds and a hyperinflated chest on X-ray signify the presence of PEEPi. Therapy to minimise PEEP includes bronchial toilet, bronchodilators, reducing tidal volume, increasing expiratory time and terminating PEEP.

There is little advantage in adding extrinsic PEEP in paralysed asthmatic or COPD patients.

Weaning from ventilation
CPAP for periods of increasing duration may be utilised as part of a ventilation weaning strategy using a high-flow T-piece circuit. It reduces airways collapse and work of breathing.

Upper airway obstruction
Patients with obstructive sleep apnoea can benefit from long-term nasal CPAP which improves airway patency by splinting open the oropharynx and reducing oedema. This reduces the number and severity of apnoeic episodes, daytime somnolence, hypertension and morbidity. CPAP improves forced expiratory flow in infants with tracheomalacia by increasing lung volume.

Other indications
The application of PEEP/CPAP has been shown to increase survival after gastric acid aspiration. CPAP has been advocated for pneumocystis pneumonia in AIDS in order to avoid mechanical ventilation. PEEP/CPAP can also splint the chest wall in patients with a flail chest injury.

Independent lung ventilation, using a double lumen endobronchial tube, allows the application of a higher level of PEEP to a less compliant diseased lung (pulmonary contusion, aspiration, pneumonia, atelectasis, single-lung transplant) and a lower level to the more compliant lung. This results in better gas exchange and less cardiovascular instability. Such measures are used only in refractory hypoxia.

How much PEEP?
The first aim of applying PEEP/CPAP is to improve the delivery of oxygen and not just P\textsubscript{a}O\textsubscript{2}. Thus, it is important to achieve an optimum increase in P\textsubscript{a}O\textsubscript{2}, whilst maintaining cardiac output. Several ‘ideal’ levels of PEEP/CPAP have been described: (i) optimal PEEP – lowest physiological shunt fraction; (ii) best PEEP – optimal static lung compliance; (iii) preferred PEEP – best oxygen delivery; and (iv) least PEEP – ‘acceptable’ value for P\textsubscript{a}O\textsubscript{2}, inspired oxygen concentration and cardiac output.

In a clinical situation, PEEP therapy may be guided by the pressure–volume curve, plotted by a modern ITU ventilator. It allows identification of improved compliance with increasing PEEP (above LIP), although this is only possible with a paralysed or heavily sedated patient. An alternative method is to increase the level of PEEP incrementally until no further increase in P\textsubscript{a}O\textsubscript{2} is achieved or cardiovascular instability occurs.

Typical values of PEEP that may be required in different lung pathologies are approximately 0–10 cmH2O in pneumonia, acute exacerbation of COPD or late ARDS, 7–10 cmH2O in severe pulmonary oedema or early ARDS, and 10–20 cmH2O in lung consolidation or severe ARDS. It is important that manoeuvres for ‘alveolar recruitment’ are performed intermittently. These include application of large tidal volumes and high end-inspiratory pressures (30–40 cmH2O), held for 20–40 s.

Alternative therapies should be considered if hypoxia is refractory to PEEP or where PEEP produces unacceptable side effects. Such therapies include prone ventilation, extracorporeal membrane oxygenation (ECMO) or a reduction in oxygen requirements (e.g. cooling, sedation).

Complications of PEEP/CPAP
CPAP is, by and large, a very safe technique. Complications are infrequent but include pressure effects from ill-fitting masks (pressure necrosis of the bridge of the nose), barotrauma and patient discomfort. Gastric distension, which may cause splinting of the diaphragm, atelectasis and gastric rupture is much less common. However, a nasogastric tube is often recommended.

Barotrauma
Barotrauma is the consequence of over expansion and rupture of alveoli. It leads to pulmonary interstitial emphysema, pneumomediastinum, pneumothorax, pneumopericardium, surgical
emphysema, pneumoretroperitoneum or pneumoperitoneum. Predisposing factors for barotrauma include prolonged ventilation, necrotising lung pathology, high peak airway pressures, secretion retention and inhomogeneous parenchymal disease. Rarely, air tracks into the vascular tree and an arterial air embolus may occur causing brain lesions or myocardial infarction.

**Cardiovascular effects**

The fall in cardiac output is proportional to the increase in mean intrathoracic pressure associated with PEEP/CPAP. Increased intrathoracic pressure decreases venous return and compresses the pericardium with reductions in atrial and ventricular compliance leading to low cardiac output. This effect is exaggerated by hypovolaemia. However, in the presence of poorly compliant lungs, PEEP will have a smaller effect on the intrathoracic pressure.

Over-distension of the lung can increase pulmonary vascular resistance resulting in an increased right ventricular end-systolic volume which causes bulging of the interventricular septum into the lumen of the left ventricle. This reduces left ventricular compliance and filling, contributing to the reduction in cardiac output. However, in patients with left ventricular failure, the effects of PEEP/CPAP on left ventricular filling may be advantageous and actually improve left ventricular performance.

The cardiovascular effects of PEEP can be minimised by maintaining a low intrathoracic pressure. This can be achieved by maximising the spontaneous respiratory effort utilised in the support mode, increasing expiratory time and avoiding lung hyperexpansion. In addition, intravenous fluids and inotropes may be required to maintain cardiac output.

**Systemic effects**

PEEP reduces cerebral perfusion pressure by increasing central venous pressure. Transmission of increased intrathoracic pressure may also result in increases in cerebrospinal fluid pressure and intracranial pressure. In general, intrathoracic pressure should be kept as low as possible in patients in whom cerebral perfusion is likely to be compromised. The use of 15–30° head-up tilt with a slightly flexed neck should allow the use of 10–12 cmH₂O of PEEP without increasing ICP.

Other effects associated with the application of PEEP are anti-diuresis and anti-natriuresis (possibly due to increased renal venous pressure), reduced release of atrial natriuretic factor (due to reduced atrial dilatation) and enhanced sympathetic activity with increased plasma norepinephrine, renin and anti-diuretic hormone. High levels of PEEP can also compromise hepatic and splanchnic perfusion secondary to reduced arterial pressure and increased venous congestion in the abdomen.

**Monitoring**

The risk of most complications can be reduced by using PEEP/CPAP for appropriate indications to a desired end-point and by using careful monitoring. Extensive respiratory monitoring (i.e. respiratory pressures and volumes, pulse oximetry, capnography, arterial blood gases) are mandatory. Monitoring of the cardiovascular system using arterial blood pressure, central venous pressure and pulmonary artery occlusion pressure (where indicated) is essential. It should be remembered that CPAP and PEEP may produce changes in CVP and PAOP readings that do not reflect a true change in trans-mural cardiac filling pressures, especially if the tip of the pulmonary artery flotation catheter is not in the dependent part of the lung. However, poorly compliant lungs may result in less transmission of the increased airway pressures of high levels of PEEP to other thoracic structures.

**Contra-indications for the application of CPAP/PEEP**

Relative contra-indications to PEEP include:

- **Lack of atelectasis/airways collapse as a cause for the hypoxia**
- **Emphysema (increased risk of barotrauma)**
- **COPD (hypoxia is usually secondary to V/Q mismatch, rather than shunting)**
- **Raised intracranial pressure (ICP)**
- **Untreated pneumothorax**
- **Lung cysts**
- **Low cardiac output states**

**Key references**


See multiple choice questions 55–57.