The Intensive Connection

Respiratory assessment and monitoring

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Respiratory assessment and monitoring

Current Status 2017

Awaiting major review

This module is updated and maintained by the (ARF) section

Latest Update

Update

Acute Respiratory Failure

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Update Info

Learning Objectives

After studying this module on **Respiratory assessment and monitoring**, you should be able to:

You should be able to:

- Understand the utility and limitations of history and examination tools for diagnosis of various lung disease states
- Understand the use of monitoring tools such as pulse oximetry and capnography
- Interpret common pressure and flow tracings used in invasive ventilation to improve patient care
- Understand the approaches and tools used to successfully wean patients from mechanical ventilation

eModule Information

Expiry date:

COBATrICe competencies covered in this module:

Competencies

1. Adopts a structured and timely approach to the recognition, assessment and stabilisation of the acutely ill patient with disordered physiology
2. Manages the care of the critically ill patient with specific acute medical conditions
3. Identifies the implications of chronic and co-morbid disease in the acutely ill patient
4. Recognises and manages the patient with acute respiratory failure and ARDS
5. Initiates, manages, and weans patients from invasive and non-invasive ventilatory support

Faculty Disclosures:
The authors of this module have not reported any disclosures.

Duration: 7 hours

1. Introduction

Respiratory failure is the most common reason for admission to critical care. Respiratory failure should be understood as when a patient is unable to maintain satisfactory gas exchange to allow metabolic pathways to proceed and can be detected in failure of oxygenation and/or carbon dioxide elimination.

The examination and monitoring skill set described in this module starts from basic bedside tests and extends to modern invasive monitoring and diagnostic techniques. A critical care doctor must understand the strengths and weaknesses of these sapproaches and use them to best care for their patients.

A large part of this module overlaps by necessity with that on mechanical ventilation, and they should be considered together.

2. How to recognise lung diseases

While many cases of respiratory failure may present with similar disorders of gas exchange, the treatment and prognosis will differ greatly. It is therefore essential to take a thorough history from the patient or relatives and perform a detailed examination.

2. 1. Clinical history

As well as the history of the respiratory illness, it is important to take a full past medical and surgical history including medication and other factors. See the key history points in respiratory disease below:

- Smoking: exacerbates and causes airway disease, provokes neoplastic changes, suggests reactive airways
- Weight: triggers questions on sleep and somnolence. Obesity can cause both obstructive sleep apnoea and obesity hypoventilation syndrome.
- Work: occupational lung disease often shows a waxing-waning pattern with shifts and weekends. Dust, industrial triggers.
- Travel: this can be contact with travellers from areas of endemic disease (TB) or travel in a patient to particular areas and resultant exposure
- Systemic diseases with respiratory impact: many multisystem diseases like rheumatological diseases carry with them both direct respiratory effects e.g Caplan syndrome, or side-effects of medication like methotrexate pneumonitis
- Family history: there are many genetic factors associated with respiratory disease, either causative (cystic fibrosis) or predisposing (alpha-1 antitrypsin deficiency)
- Surgical history: close inspection of thorax and neck to look for scars, previous drains or other interventions. Particular scars to be aware of include thoracotomy, sternototomy or retrosternal goitre explorations

2. 2. Clinical examination in respiratory diseases

The most common features seen clinically at the bedside are cough, productive sputum, haemoptysis (blood in sputum), dyspnoea, cyanosis, clubbing and chest examination signs. We will look at these separately.

2. 2. 1. Cough

Coughs can be both dry or productive; acute or chronic, painful or painless. Chronic cough is usually linked to either a chronic condition (COPD, asthma, acid reflux) or medication (ACE-inhibitors, inhaled steroids). Acute cough can be non-productive (the characteristically painful cough of pleural inflammation) or productive (bacterial infections of the respiratory tract).

2. 2. 2. Sputum

Sputum varies in respiratory disease depending upon timing, location and causation. Patients can struggle to differentiate between respiratory and oropharyngeal secretions, and occasionaly in fistulating disease the two share a common pathway. Colour and smell can give suggestions of cause, but are not pathognomic. Microbiological and mycological examination is important, and early morning samples are recommended for tubercular testing.

2. 2. 3. Haemoptysis

Literally the presence of blood in sputum, this can range from streaking caused by recurrent cough, to life-threatening haemorrhage. Significant haemoptysis is classed as >200ml in one episode. Common culprits include neoplasia, infarction and tuberculosis.

2. 2. 4. Dyspnoea

Dyspnoea is a subjective symptom defined as uncomfortable or distressing breathing. It may well accompany an objective rise in the work of breathing, or tachypnoea (breathing quickly). Dyspnoea may arise from pulmonary receptors, or may be centrally driven.

2. 2. 5. Cyanosis

Cyanosis is the presence of reduced haemoglobin the blood, largely due to deoxygenation of the blood. While classically this was described as being present when reduced haemoglobin exceeded 5g/100ml of blood, modern medicine relies on pulse oximetry and blood gas measurement so an increased incidence is now reported.

Peripheral cyanosis (discolouration of peripheries/digits) is largely due to an inadequate cardiac output leading to increased tissue extraction. Central cyanosis is mainly due to disorders of gas exchange during pulmonary blood flow.

2. 2. 6. Clubbing

Clubbing of the digits (fingers>toes) can be hereditary, idiopathic or associated with both pulmonary neoplasia and diseases of chronic infection like abscess and empyema.

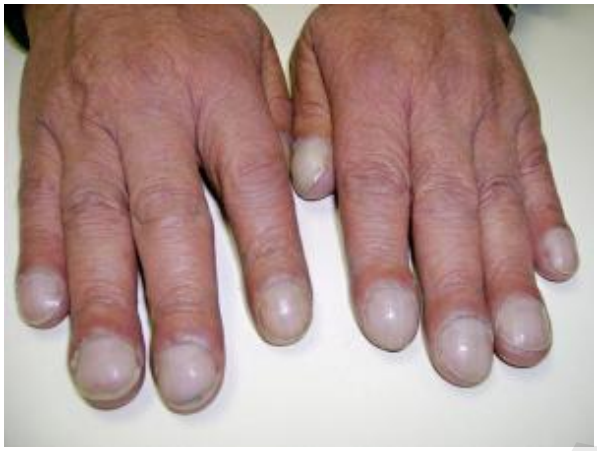

Figure 1: Clubbing. ESICM Academy, 2018.

2. 2. 7. Chest examination - inspection

By fully exposing the chest you can fully appreciate the work of breathing, including depth and expansion as well as symmetry. Accessory muscle use such as is seen in forced breathing can include use of the sternocleidomastoids, the trapezius as well as intercostal indrawing.

Disorders of chest wall and thoracic structures can lead to both congenital and inherited dysfunction. Kyphoscoliosis, polio and ankylosing spondylitis can all lead to restrictive chest wall defects that either continually affect gas exchange or via reduction in lung capacity affect gas exchange during illness. Chronic lung diseases including emphysema can produce hyper-expansion of the thoracic cage.

2. 2. 8. Chest examination – palpation

Disorders of expansion can be confirmed by bimanual palpation during the respiratory cycle. Swelling, emphysema or crepitus can all be assessed as can skeletal abnormalities. Lymph nodes should be palpated for in the supraclavicular fossae as well as the axillae.

2. 2. 9. Chest examination – percussion

While percussing out effusions and pneumothoraces have classicals signs, these have been shown to be of limited utility clinically and should not be relied upon for clinical assessment on their own.

2. 2. 10. Chest examination – auscultation

Auscultation with a stethoscope allows for assessment of the presence/absence and quality of breath sounds. Healthy breath sounds are termed vesicular, which are predominantly soft and heard best in inspiration. Reduced breath sounds can be due to reduced air entry from lobar collapse, or alternatively over-inflation as in emphysema, or even from pleural pathology.

Consolidation of lung tissue leads to increased transmission of breath sounds through patent upper airways, meaning pronounced expiratory flow that creates bronchial breath sounds – often found in pneumonic processes. Opening and closing smaller airways can cause crackles, but these can also be found in fibrotic lung diseases (so-called Velcro crackles). Bronchial crackles can often be reduced or cleared by coughing, while crackles from pleural pathology will not clear with any manoeuvre.

Important

You need to continually reflect upon your effectiveness of history taking and examination. Are your findings borne out by subsequent investigations? Consider having a colleague observe your practice and offer tips.

2. 3. Investigations

There are a wide range of imaging techniques available for the respiratory system, including radiology and bronchoscopy. Intensive Care Medicine requires an in-depth knowledge of how best to image the chest for the suspected condition. See ESICM module on Clinical imaging for more information.

2. 3. 1. Chest X-rays

The most common imaging used in hospital medicine, and certainly in Intensive Care. The practice of daily chest radiographs for every ICU patient has passed, with meta-analysis showing little benefit in non-focused use. Practitioners should be aware of the limitations of the modality, and not over-interpret limited studies.

Why are there limitations of ICU chest radiographs?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

Mobile units are less optimal, films are more likely to be taken supine with effects on pleural pathology detection and the antero-posterior technique falsely suggests cardiomegaly and wide mediastinum.

Plain films allow for assessment of parenchymal changes, the anatomy of the upper airways, cardiac changes and subdiaphragmatic acute changes including free air from visceral perforation.

It would be recommended to perform regular films after endotracheal intubation, and insertion of central venous catheters to assess position and immediate complications. Opinions of practictioners vary when fiberoptic placement of either endotracheal or tracheostomy tubes has been undertaken.

Important

All critical care doctors should be able to make time-critical diagnoses rapidly using chest radiographs including pneumothoraces, and reliably assess placement of invasive devices including endotracheal tubes, venous catheters and intercostal drains.

How should you assess a chest radiograph?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

A Multiple methods exist, any can be used as long as they are reliably followed and thorough. The quality of image should be assessed including patient position. The location of all indwelling devices should be commented upon, and the parenchyma pleura, mediastinum and diaphragm should all be considered.

2. 3. 2. Computed tomography (CT)

The use of CT scanning in lung disease has hugely expanded, as modern scanners are more available, quicker and offer less radiation dose than older devices. The difference between conventional CT and high-resolution CT (HRCT) has become less pronounced due to image acquisition techniques.

Current indications include:

- Pulmonary pathology
- Mediastinal evaluation
- Interstitial disease
- Pulmonary angiography for embolic disease
- Angiography of great vessels
- Interventional studies including drainage and biopsy

The use of CT in acute severe respiratory failure has grown, with interest in differentiating pulmonary and non-pulmonary causes of ARDS, as well as performing real-time recruitment studies with bi-level PEEP strategy to assess how recruitable lungs may be. Radiologists

can express recruited lung in terms of percentage of lung tissue and help guide ventilatory strategy in this high-risk group.
There still requires some consideration of the effect on a patient of transfer to CT.

2. 3. 3. Magnetic Resonance Imaging (MRI)

It is rarely necessary to use MRI scanning for ICU patients with respiratory disease. There is some utility in assessing lymph nodes and arteriovenous malformations, but this is rarely needed acutely.

MRI scanning carries with it issues including remote access, need for bespoke ventilators and monitoring and staff training needs.

2. 3. 4. Lung Ultrasound

Lung ultrasound is a technique that has recently gained interest from the critical care community that allows for bed-side evaluation of parenchyma, pleural disease and the chest wall as well allowing guidance of interventions. Normal lung parenchyma is almost completely air-filled, allowing for minimal transmission of ultrasound waves, but in pathological conditions this changes and increased density tissue allows for observation and correlation.

The technique by its nature is focussed, quick and replicable. The key for utility is image capture and avoiding over-interpretation.

Key conditions where ultrasound is of use include:

- Effusions
- Pneumothorax
- Aiding chest drain/pleural tap performance

Effusions can be detected, assessment made of volume, nature and site. The volume of an effusion can be made by measuring the maximal distance between the two pleural layers and multiplication of this millimeter distance by 20 to give a volume in millilitres. Ultrasound can be used in real-time to guide needle insertion for either sampling or Seldinger-style drainage.

Pneumothoraces can be detected by identifying the absence of lung sliding (lung moving below hyperechoic pleura) and by detecting a “lung edge” where partially collapsed lung edge is seen moving. This is particularly useful in detecting anterior pneumothoraces which are difficult to pick up on plain films.

2. 3. 5. Electrical impedance tomography (EIT)

EIT is a technique that uses a circumferential belt of electrodes to calculate potential differences across the thorax. A mathematical algorithm then displays a 2D image that can display changes in lung aeration either by a thermal display or numerical output. Changes

in aeration over time are summated and displayed as changes in either positive or negative direction.

The technique allows for real-time detection of changes in lung heterogeneity with easy identification of the anatomical location of pathology. More research is ongoing, but given it's non-invasive and radiation free nature interest is high in using EIT to guide dynamic evaluation of recruitment manoeuvres, bronchoscopy, drainage of effusions and similar clinical interventions.

2. 3. 6. Bronchoscopy

Bronchoscopy is the process where a fiberoptic scope is passed into the tracheobronchial tree, normally using a flexible scope. Images can be recorded and photographed. It offers the potential for investigation as well as treatment and is used by many practitioners to aid procedures like tube exchange and percutaneous tracheostomy.

Uses of bronchoscopy include:

- Detecting structural abnormalities
- Bronchoscopic alveolar lavage (BAL)
- Retrieving foreign bodies
- Sampling parenchyma

The bronchoscope occupies a large part of the tracheal tube leading to changes in respiratory mechanics including increases in peak pressures, leaks of tidal volume, increases in expiratory resistance and loss of lung volume especially when suction is used.

Figure 2: Modern disposable bronchoscope.
ESICM Academy, 2018.

3. Monitoring respiratory function

3. 1. Analysis of oxygenation

3. 1. 1. Blood gas analysis

Sampling arterial blood to analyse gas content is the most frequent blood test in ICU. While modern gas machines can produce values for the saturation of haemoglobin (which is the predominant source of oxygen), many practitioners prefer to target dissolved oxygen in plasma – the PaO_2. There is a relationship between PaO_2 and SpO_2 that can be seen in the oxyhaemoglobin dissociation curve.

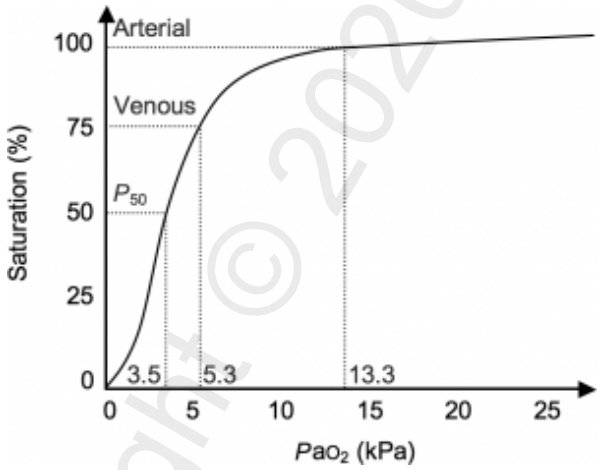

Figure 3: Oxyhaemoglobin dissociation curve.
ESICM Academy 2018.

Gas exchange relies upon matching of ventilation and perfusion. In health this is tightly controlled, but illness and medical intervention such as ventilation and anaesthesia can lead to disorders of VQ matching.

3. 1. 2. Oxygen content and consumption

Most oxygen is carried bound to haemoglobin, and as such arterial oxygen content (CaO_2 is mainly determined by SaO_2 and haemoglobin content).

$$CaO_2 = (1.34 * Hb * SaO_2 + (0.003 * PaO_2)$$

The correction factors above include the oxygen binding capacity of oxygen, and the amount of oxygen that can dissolve in 100ml of plasma Normal CaO_2 is 16-20ml O2/100ml blood.

Calculate the CaO_2 for this 35 year old man. He has a heart rate of 120, a systolic BP of 95mmHg and a haemoglobin of 120g/L. His blood gas shows a SaO_2 of 96%, and a PaO_2 of 13.5kPa.

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

15.4ml O_2 per 100ml

The influence of anaemia on oxygen carriage is far greater than the effect of hypoxaemia given the relative contribution to the equation.

It should be noted that oxygen delivery (DO_2) is affected not only by oxygen content, but by low cardiac output states and by impaired tissue extraction of oxygen.

$$DO_2 = CO * CaO_2$$

which can be indexed to

$$DO_2I = CI * CaO_2$$

Historically various studies have attempted to manipulate morbidity and mortality by altering either oxygen delivery (raising cardiac output or transfusion) or reducing oxygen consumption (VO_2). The failure of these studies to translate to patient-centred benefit has led to this approach being largely confined to lab studies (cf gastric tonometry, supranormal physiology).

3. 1. 3. Pulse oximetry

Pulse oximetry uses spectrophotometry to give continuous beat-by-beat monitoring of the haemoglobin oxygen saturation of peripheral blood (SpO_2). This allows for beat-to-beat monitoring of oxygen saturation, albeit with an in-built sampling delay.

The probes work by a modification of the Beer-Lambert law: “the attenuation of light is relative to the absorbance and depth of a material”. The probe shines light from two separate wavelengths – 660nm red and 940nm infrared – detected by a photodiode on the other side of the probe. Oxyhaemoglobin absorbs more infrared than red, and ddeoxyhaemoglobin vice versa. The ratio of absorbance allows for calculation of saturation, after subtraction of non-pulsatile elements.

Different haemoglobin species can confuse the oximetry picture, and if suspicion exists to this end then blood co-oximetry should be carried out. Modern probes have attenuated many previous problems with nail polish, bilirubinaemia and skin pigmentation, but problems still exist in hypothermia and low perfusion states where pulsatility is lost.

Does pulse oximetry reflect PaO_2 reliably?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

No. Above a PaO_2 of 12kPa the saturation of haemoglobin is close to 100%, and as such care needs to be taken to avoid hyperoxia (given evidence of potential harm in many critically ill patients).

3. 2. CO analysis

3. 2. 1. Capnography

Capnography which is the graphical display of expired carbon dioxide, has evolved into a widely accepted standard of care in anaesthesia, and also now in ventilated critical care patients. Its widespread use has made a positive impact on patient safety, both for detection of airway misplacement and dislodgement, as well as monitoring and adjusting minute ventilation to optimise physiology.

Important

All critical care physicians should be confident in interpreting capnography waveforms and acting appropriately upon their conclusions.

3. 2. 2. Time capnography

The most commonly used clincal tool is a time capnograph that plots CO_2 concentration in both inspired and expired gas over time. An in-line or out-line sampler passes gas through a CO_2 analyser which produces a typical waveform. The waveform is a summation of a complex system of alveoli that empty with different time-constants and under the influence of differing ventilation-perfusion ratios but producing a largely uniform waveform made of an inspiratory phase and an expiratory period split into three distinct phases.

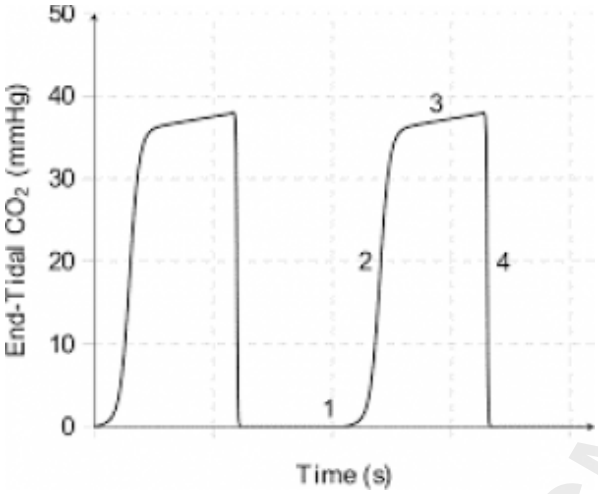

Figure 4: Capnography. ESICM Academy, 2018.

Phase 1 is the end of inspiration, with gas largely coming from communicating airways and equipment, close to zero. This may be altered in circumstances where rebreathing of CO_2 occurs, rare outside of the operating theatre.

Phase 2 is the first upstroke, with a rapid change of mixed alveolar and dead-space gases. Phase 3 represents a plateau of pure alveolar gas. There is always a slight rise due to the heterogenous time constants of alveolar emptying, leading to the highest value of $EtCO_2$ (end-tidal CO_2). There is then a rapid descent to zero in phase 4.

Figure 5: Real time EtCO2 monitoring.ESICM Academy, 2018.

In states of health there is a fairly reliable alveolar-arterial gradient of 0.7kPa in relation to CO_2, largely due to the high solubility of the gas and permeability of the alveolar membrane. This cannot be assumed in the critically ill, with many patients having a significant $PaCO_2$-$EtCO_2$ due to varying combinations of lung pathology, vascular tone and mechanical ventilation. Cardiac output and subsequent pulmonary perfusion is also a key factor in $EtCO_2$ production, with a linear relationship between O_2 consumption and CO_2 production.

Curve analysis

Analysing the waveforms can give diagnostic information to the discerning clinician.

Bronchospasm

Bronchospasm from either smooth muscle contraction or obstruction produces a pronounced increase in the heterogeneity of alveolar emptying. This leads to a marked

increase in the slope of Phase 3, as lung units continue to empty, and no plateau may be seen, especially if an inadequate I:E ratio is chosen. As alterations are made to ventilation, or bronchodilators are administered then the slope may reduce.

It is important to note that external factors may cause obstruction to the capnograph waveform, such as kinking or pressure applied to the endotracheal tube.

Cardiac arrest

As previously described, $EtCO_2$ is reliant on pulmonary blood flow. Previous teaching was that no $EtCO_2$ should be expected during cardiac arrest, but with chest compressions there should still be detectable changes in the baseline of capnographic measurement. Modern resuscitation guidelines advocate use of capnography both for detection of endotracheal tube placement, and for guiding quality of chest compression.

Spontaneous breathing

True spontaneous ventilatory modes will show a capnography trace similar to those already described. Spontaneous efforts during mandatory ventilation can be detected by downwards deflections, or incisurae, during the respiratory cycle. They reflect an attempt by the patient to trigger the ventilator, and if they are not followed by an augmented breath then this allows the clinician to be aware of patient-ventilator dyssynchrony.

3. 2. 3. Volume capnography

This is not widely used in clinical practice. It uses the expired CO_2 concentration and displays this against flow rate to allow calculation of CO_2 production and respiratory dead space. It allows for calculation of anatomical dead space and alveolar dead space.

Volumetric capnographs use a derivation of Fowler's method (based on single-breath nitrogen washout curves) to produce anatomical dead space and calculate alveolar dead space using this and a measure of alveolar CO_2. Limitations occur due to the assumptions used to derive the input numbers.

In normal patients, physiological dead space is the main determinant of delta PCO_2 (the difference between arterial and end-tidal or mixed gas CO_2). Physiological dead space is increased in shock states, pulmonary hypoperfusion and in states where pulmonary vessels are obstructed (be that massive emboli or scattered microthrombi). Anatomical dead space in the ventilated patient is increased by connectors, HME filters and long tubing – this is not normally a problem but can exacerbate dead-space especially when low tidal volume strategies are employed.

> ★ **Important**
>
> High frequency but low volume ventilatory strategies that are used in ARDS patients can lead to a marked increase in Vd/Vt that leads to hypercarbia and wasted ventilatory effort.

3. 3. Mixed venous gas analysis

3. 3. 1. SvO/ScvO

Mixed venous/mixed central venous oxygenation is the amount of oxygen left in venous blood after systemic oxygen delivery. It has been characterised as an indicator of supply-delivery demand, and a marker of oxygen extraction.

SvO_2 is an aggregation of all venous effluents into the right side of the heart. Due to the averaging of flows, one tissue bed may be hypoxic while the other larger beds dilute the effect – however it remains a useful tool. SvO_2 values between 70-80% are quoted as being ideal, with a balance between supply and demand at tissue levels. Falling SvO_2 levels potentially represent reduced oxygen supply, while high levels >80% can be seen in hyperdynamic states where tissue extraction is inadequate.

Measurements of SvO_2 should be evaluated in light of other markers of tissue perfusion (UOP, lactate, BE). Published work has failed to show a consistent benefit in targeting therapy to measurements of venous oxygenation, after a flurry of interest in goal-directed therapy. Like with many other critical care interventions individual patients may benefit from measurement while others may receive unnecessary interventions. Trends and response to therapy are more useful than single measurements.

Inbuilt SvO_2/$ScvO_2$ systems and pulmonary artery catheters are considered in the ESICM module on Haemodynamic Monitoring.

3. 4. Extravascular lung water

The critically ill often are exquisitely balanced between fluid need to maintain tissue perfusion, and the risk of capillary leakage leading to tissue and more importantly alveolar oedema. Cytokines and vascular destruction predisposes the outwards leak of capillary fluid as disruption of the glycocalyx leads to dysfunction.

Extravascular lung water (EVLW) can be measured by transpulmonary thermodilution and can be expressed as a ratio of pulmonary blood volume to create a permeability index that can suggest the differentiation between cardiogenic and non-cardiogenic pulmonary oedema.

Some investigators have suggested that targeting EVLW as a marker of fluid resuscitation can shorten duration of mechanical ventilation, but this has not been borne-out in large-volume study populations.

In text References

(Sakka et al. 2002)

References

- Sakka SG, Klein M, Reinhart K, Meier-Hellmann A., Prognostic value of extravascular lung water in critically ill patients., 2002, PMID:12475851

4. Monitoring ventilator waveforms

4. 1. Airway pressure

4. 1. 1. Volume-controlled ventilation (VCV)

During VCV there is a classical pressure waveform, moving from a rapid increase to overcome resistance of the tube and airway before a linear growth up to a maximum pressure (P_{max}). There is then a rapid drop to a lower pressure known as plateau pressure or pause pressure (P_{plat}) when equilibriation occurs between the “tube” pressure and the alveolar pressure. P_{plat} respresent the pressure needed to overcome recoil forces of chest wall and lung, while P_{max} reflects the pressure measured in large airways.

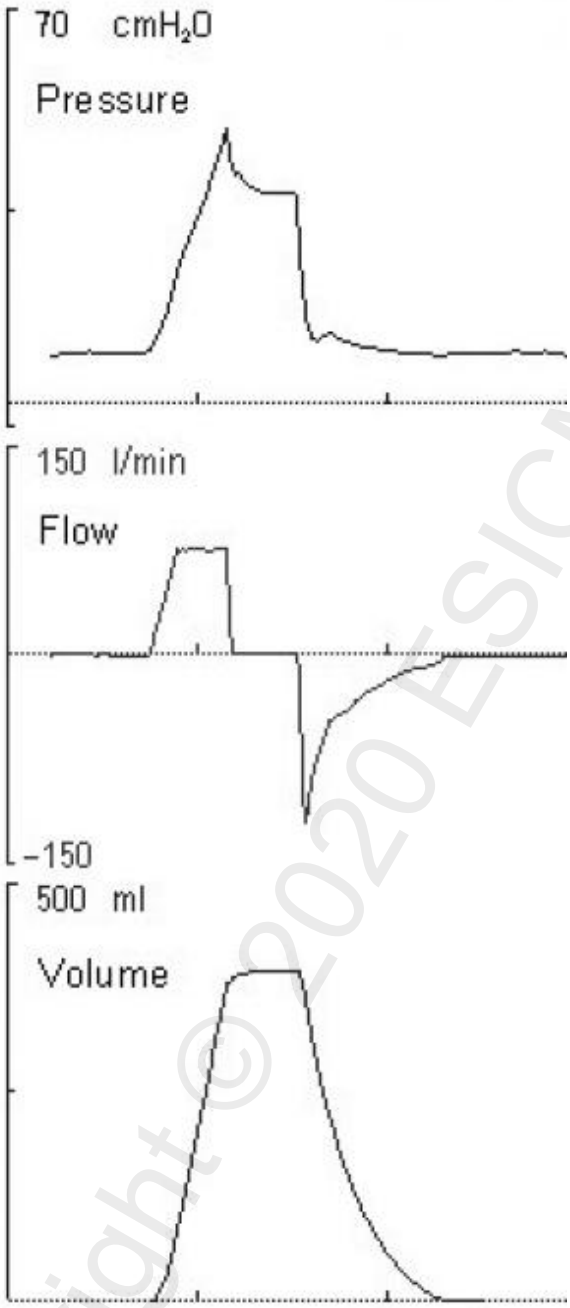

Figure 6:VCV Characteristics. ESICM Academy,2018.

4. 1. 2. Pressure-controlled ventilation

In PCV the dependent is flow with a constant pressure, ventilators adjust flow continually dependent upon compliance and other factors to achieve a set inspiratory pressure.

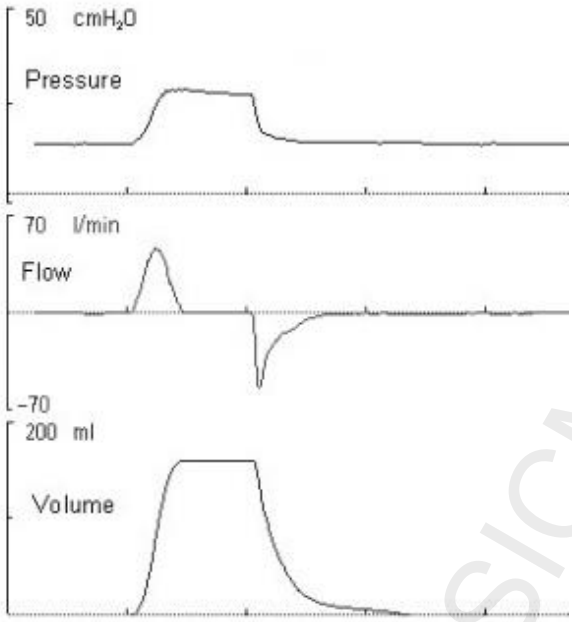

Figure 7:PCV characteristics. ESICM Academy,2018.

4. 2. Airway flow

As can be seen in Figure 6 and 7, flow characteristics change between VCV and PCV. In practice most modern ventilators use a decelerating flow pattern for VCV, PCV and PSV which most accurately mimicks “natural” breathing flow patterns.

Several online simulators exist to allow clinicians to model various flow patterns seen in different ventilatory modes.

4. 3. Loops

4. 3. 1. Pressure-volume loops

Pressure volume loops can allow for optimisation of settings during tidal breathing both for patients with lung injury and obstructive lung disease. They can allow for detection of compliance issues, alveolar recruitment, airway collapse, dyssynchrony and resistive work.

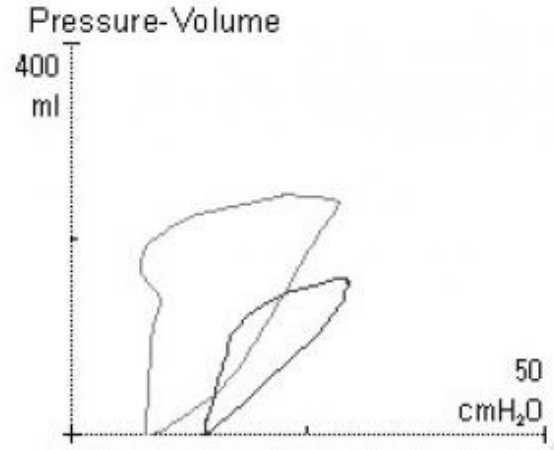

Figure 8: Pressure-volume loop. ESICM Academy,2018.

4. 3. 2. Flow-volume loops

Analysing flow volume loops allows for identifying flow limitiation, airway obstruction, secretions (sawtooth), air-leaks and airtrapping.

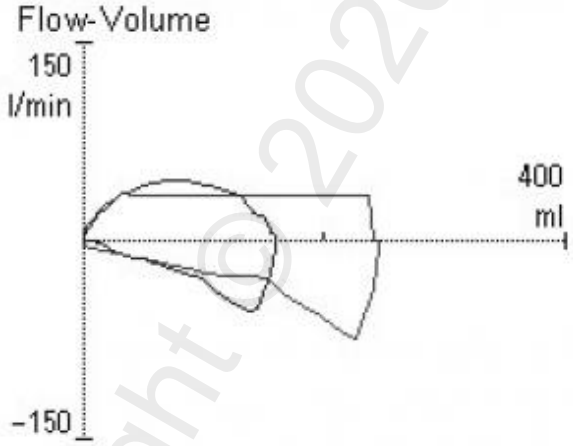

Figure 9: Flow-volume loop. ESICM Academy,2018.

4. 3. 3. Oesophageal pressure

The pressure in the thin-walled oesophagus (P_{es}) approximates the mean pleural pressure (Ppl). Although absolute values may not be exact, correlation is highly likely between Pes and Ppl during positive pressure ventilation. A catheter fed into the distal third of the oesophagus with a transducing balloon as part of the set-up can allow for a set of calculations that allow for separation of total respiratory compliance (Crs) into lung compliance (C_L) and chest-wall compliance (C_{cw}).

$$CL = \frac{Vt}{(P_{AO} - P_{es})end - inhalation - (P_{AO} - P_{es})end - exhalation}$$

and

$$C_{CW} = \frac{Vt}{(P_{es} - P_{atm})end - inhalation - (P_{es} - P_{atm})end - exhalation}$$

Where VT = tidal volume, Patm is atmospheric pressure and PAO is pressure at the airway opening.

Oesophageal manometry allows the factoring of C_{cw} into decisions around ventilatory practice. Low chest wall compliance can occur in critical care either as an existing issue (morbid obesity) or developed such as in fluid overload, raised intra-abdominal pressure or chest burns. Higher airway pressures are needed to maintained volumes by achieving the same trans-pulmonary pressure, These pressures are exerted across the chest wall rather than across the alveolus.
In ARDS some protocols proposed to titrate PEEP according to end expiratory transpulmonary pressure or to elastance related en inspiratory transpulmonary pressure.
End inspiratory and expiratory airway and oesophageal pressures has to be measured during a 5 sec pause of the ventilator in passive patient, Variables can be calculated using the following equations:

- $$Elastance = \frac{1}{Compliance}$$

Elastance Related end inspiratory Transpulmonary Pressure:

- $$PL, EL = Pplat * \frac{lung\ elastance}{respiratory\ system\ elastance}$$

End Expiratory Transpulmonary Presssure:

- $PLexpi = end\ expiratory\ airway\ pressure - end\ expiratory\ esophageal\ pressure$

Airway driving pressure:

- $$DPaw = Pplat - total\ PEEP$$

Transpulmonary driving pressure (DPL):

- $DPL = DPaw – (end\ inspiratory – end\ expiratory\ esophageal\ pressure)$

- $$Respiratory\ system\ elastance = \frac{Pplat – total\ PEEP}{V_T}$$

- $$Lung\ elastance = \frac{DPL}{V_T}$$

- $$Respiratory\ system\ elastance = Lung\ elastance + Chest\ wall\ elastance$$

Moreover oesophageal pressure can be used in spontaneous breathing patient to assess work of breathing and patient-ventilator interaction with asynchronies.

In text References

(Akoumianaki et al. 2014)

References

- Akoumianaki E, Maggiore SM, Valenza F, Bellani G, Jubran A, Loring SH, Pelosi P, Talmor D, Grasso S, Chiumello D, Guérin C, Patroniti N, Ranieri VM, Gattinoni L, Nava S, Terragni PP, Pesenti A, Tobin M, Mancebo J, Brochard L; PLUG Working Group (Acute Res, The application of esophageal pressure measurement in patients with respiratory failure., 2014, PMID:24467647

4. 4. Estimation of dynamic and static airway pressures

4. 4. 1. During controlled ventilation

Modern ventilators calculate and display a number of calculation of mechanics in addition to previously described loops which can help with evaluation of respiratory support.

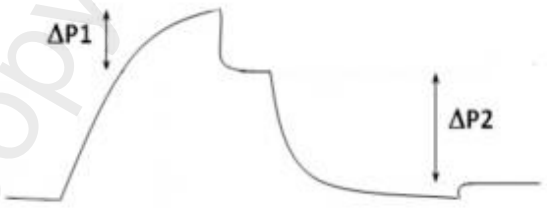

Figure 10: Static compliance reflect. ESICM Academy, 2018.

Static compliance reflects the elastic features of the respiratory system while dynamic compliance includes flow-dependent resistive elements including endotracheal tubes and ventilator tubing. The figure above illustrates the characteristic waveform used for analysis

of static compliance where inspiratory flow is stopped at end of inspiration. The peak pressure rapidly falls from P1 (Ppeak) to P2 (Pplat). The gradient of decay of ΔP1 is dependent upon the resistive properties of the system.

$$Resistances = \frac{\Delta P1}{Flow}$$

The formula that gives us respiratory system compliance needs tidal volume (VT), Pplat which is end inspiratory plateau pressure and PEEPi which is intrinsic PEEP. ΔP2 is commonly referred to as driving pressure and has been recently reappeared in interest with regards to ARDS.

$$CRS = \frac{V_T}{P_{plat} - (PEEP + PEEPi = VT/\Delta P2)}$$

In text References

(Amato et al. 2015)

Is there a clinical significance in measuring the difference between Pmax and Pplat?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

Yes. Airway secretions, bronchospasm and problems with the endotracheal tube can all negatively affect Pmax while measuring Pplat reassures the clinician that high airway pressures are not being delivered to the alveolus.

4. 4. 2. During assisted ventilation

Dynamic compliance can be calculated during pressure support ventilation (PSV) using the formulae in the above section. Static compliance can be more difficult to calculate due the problems with performing end-inspiratory occlusions with spontaneous breathing efforts. However in ARDS patients measuring compliance of the respiratory system might be very useful in spontaneous breathing patient. End inspiratory pause can be done automatically with proportional assist ventilation or manually if the pause is less than 300 ms. It is important because ventilation might not be protective if compliance of the respiratory system is low, even with 6 ml.kg-1 of PBW. In that situation spontaneous driving pressure can be too high even with low level of assist and VT.

In text References

(Bellani et al. 2019)

4. 4. 3. Auto-PEEP

During normal tidal ventilation, at the end of expiration there is a pause where alveolar pressures equalise, and alveolar pressure is close to zero. As standard ventilatory therapy PEEP is applied to prevent small airway collapse and reduce atelectrauma. In certain lung conditions (ARDS, obstructive lung disease, acute bronchospasm), the respiratory system is prevented from returning to resting volume. This is termed intrinsic or auto-PEEP, with flow still occurring and detectable at end-expiration, or expiratory truncation of a flow-volume loop. It can be detected and quantified on a ventilator by performing a an end-expiratory occlusion in a passive patient or with oesophageal pressure monitoring in a spontaneous breathing patient.

See ESICM module on COPD and Asthma and on Mechanical Ventilation.

References

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- Bellani G, Grassi A, Sosio S, Foti G, Plateau and driving pressure in the presence of spontaneous breathing., 2019, PMID:30006893

4. 5. Work of Breathing

In the presence of spontaneous breathing efforts while receiving mechanical ventilation, direct measurement of the level of effort may help the clinician to better adjust the ventilator settings and/or the sedation level. Respiratory muscle effort can be assessed by calculating work of breathing (WOB) and the pressure–time product (PTP) of the esophageal pressure (PTPes),reflecting the effort done by all of the respiratory muscles, or the pressure–time product of the transdiaphragmatic pressure (PTPdi), reflecting mostly the effort done by the diaphragm.

Work of breathing (WOB) is the energy spent on inspiring and expiring. It can be given the nomenclature of joules/minute or expressed as an oxygen consumption. Work can be divided between that done by the ventilator and by the patient, or by work spent inspiring and expiring. Work done in any phase can be viewed as the area of a dynamic pressure-volume curve.

WOB can be viewed as the energy expended to overcome elastic forces (thoracic cage, lung recoil), frictional work (airway resistance), and inertial work (largely non-contributory but include the compressive nature of gases). WOB usually consumes <5% of cardiac output, but this can rise to 15-20% during respiratory distress.

5. Lung recruitment and PEEP

5. 1. Concept of lung recruitment

Lung recruitment can be defined as an intervention undertaken during the inspiratory cycle that aims to re-inflate previously collapsed alveolar units.

Recruitment manoeuvres (RM) are transient interventions that significantly increase transpulmonary pressure in an attempt to reopen closed alveolar units. A successful RM should maintain its effect via the use of appropriate PEEP and result in an increase in FRC.

A number of different techniques have been described including sustained high-pressure (>40cmH_2O) holds, incremental steps of PEEP, “sigh breaths or intermittent high pressure tidal volumes. All can have a deleterious effect upon haemodynamics, largely through reduction in atrial return and risk barotrauma.

Recent evidence is less supportive of the use of RMs, but the heterogenous nature of respiratory failure makes it hard to generalise.

In text References

(Cavalcanti et al. 2017)

References

- Cavalcanti AB, Suzumura ÉA, Laranjeira LN, Paisani DM, Damiani LP, Guimarães HP, Romano ER, Regenga MM, Taniguchi LNT, Teixeira C, Pinheiro de Oliveira R, Machado FR, Diaz-Quijano FA, Filho MSA, Maia IS, Caser EB, Filho WO, Borges MC, Martins PA, Matsui M, Effect of Lung Recruitment and Titrated Positive End-Expiratory Pressure (PEEP) vs Low PEEP on Mortality in Patients With Acute Respiratory Distress Syndrome: A Randomized Clinical Trial., 2017, PMID:28973363

5. 2. Monitoring of lung recruitment

There are a number of modalities for measuring the effect of recruitment, from imaging to secondary measures like changes in compliance. Some modern ventilator allow for automatic volume measurement.

5. 2. 1. CT

CT scanning can detect and measure recruitability of a lung, by direct comparison of aeration at two different levels of positive pressure. This approach has been adopted by a number of respiratory failure centres.

In text References

(Gattinoni et al. 2006)

5. 2. 1. 1. EIT

Electrical impedance tomography allows bedside measurement of lung aeration. It can be used as a monitoring tool in order to titrate ventilation and find the best possible compromise between lung collapse and overdistension.

In text References

(Franchineau et al. 2017)

5. 2. 2. Automated nitrogen washout methods

Meausring FRC or end-expiratory lung volume (EELV) can help measure aerated lung and alveolar recruitment. Nitrogen washout in the mechanically ventilated lung needs a metabolic module to accurately measure inspired and expired gases. A fractional change of nitrogen is measured after a stepwise change in the inspired fraction of oxygen.

In text References

(Dellamonica et al. 2011)

5. 2. 3. Lung mechanics

Both dynamic compliance and hysteresis curves have been used to analyse the effect of recruitment. This can be done both by using changing inflection points and also by using signs of derecruitment to signify optimal values.

In text References

(Maggiore et al. 2001)

5. 2. 4. Transpulmonary pressure (TPP)

Using an oesophageal balloon to give an intrapleural pressure can help set PEEP and improve recruitment. If pleural pressure is higher than PEEP then derecruitment can occur. Titrating PEEP to keep end-expiratory TPP between 0 and 10 may stop atelectasis

In text References

(Talmor et al. 2008)

References

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6. Weaning assessment and monitoring

It is important to wean patients from mechanical ventilation as soon as is appropriate, as many of the effects of both ventilation and the sedation techniques allied to it are deleterious to patient health. Patients may be ventilated for primary lung disease, for acute illness that has no respiratory component, or for acute exacerbations of chronic lung conditions.

Every day in intensive care the question should be asked – can this patient be weaned from ventilation? By asking the question, it prompts teams to be employing screening tools and to actively try and reduce the harms associated with prolonged ventilation.

Patients can be classified as follows:

- Simple to wean – extubated on the first attempt (the majority)
- Difficult to wean – needs up to 3 extubation attempts and <7 days after wean starts
- Prolonged wean - >3 extubation attempts and >7 days after weaning commences

There are a small number of patients in whom full weaning never occurs. This is normally due to either cardiac or respiratory dysfunction that doesn't normalise, and require some form of extended support either with long-term invasive ventilation (rare) or intermittent non-invasive support (more common).

Invasive positive pressure ventilation has effects on reducing preload (both RV and LV) and left ventricular afterload. Ceasing ventilation causes reversal of these effects. This is usually tolerated well by patients, but in the presence of known or occult cardiac disease can produce marked ischaemia or myocardial dysfunction.

6. 1. Readiness to wean

Assessing the readiness to wean requires both clinical criteria and knowledge of weaning parameters.

6. 1. 1. Clinical criteria

- Underlying cause for ventilation is reversed/improved
- Adequate oxygenation (FiO_2 ~ 0.4 and PEEP ~ 5)
- Not acidotic
- Haemodynamically stable
- Not profoundly anaemic

- Normothermic
- Rousable
- Cough able to clear secretion load

6. 1. 2. Weaning parameters

These are an attempt at objectivity, though none have been confirmed as gold standard tests.

- Airway occlusion in first 0.1 second – P0.1 – a measure of respiratory drive. Values of P0.1 more negative than -4cmH_2 O show high respiratory load and are associated with non-readiness to wean.
- Diaphragm electrical activity (neural drive) monitored with a specific naso gatric tube covered by 10 electrodes. It will provide neuro-ventilatory coupling and efficiency.
- Vital Capacity (VC) – difficult to assess while ventilated, but >25ml/kg is likely to predict the ability to be liberated from ventilation
- Work of breathing (pressure time product).
- CROP index – Compliance, Rate, Oxygenation, Pressure
- Rapid Shallow Breathing Index – rate/Vt (litres) – a value of <105breaths/min/L predicts success in extubation but requires separation from ventilator and use of a volumeter

These tests and others have been shown to have varying success in clinical practice which probably mirrors the varying practice of clinicians in how weaning occurs and how liberation from ventilation is achieved.

6. 1. 3. Weaning protocols

Use of protocols in both weaning and sedation practice have been shown to reduce time to extubation, both when used by medical staff and by nursing staff. Patients should be screened daily as to appropriateness to wean, and encouragement of sedation holds allows for spontaneous breathing trials (SBTs).

SBTs can be performed using T-piece systems where patients are disconnected from the ventilator, or by using low-level pressure support (with 5cm CPAP). There is little evidence to support one method over the other. Recent evidence suggests that if T-piece trials are used, that reconnection to a ventilator for 1 hour improves the success rate of extubation.

Automated weaning programmes have been used, relying on closed loop feedback to wean patients to a target minute volume. There are mixed results in studies, but similar interfaces are being investigated in non-invasive ventilation to improve patient-ventilator interaction.

7. Conclusion

There are many tools at the disposal of the modern Intensive Care clinician to assess respiratory function. These can help us both assess underlying function and response to therapy. Although emerging therapies such as impedance tomography and automated washout curves are intriguing, there is little evidence available to support whole-sale adoption into clinical practice. Ongoing research should help us integrate laboratory practice and clinical application.

8. Glossary

Combined glossary of terms for the modules Mechanical Ventilation and Respiratory Assessment and Monitoring with acknowledgement to Dr Ed Carton for finalising its composition.

Airway pressure: Pressure at a specified point in the patient's airway.

ALI: A descriptor of an Acute Lung Injury process; since a recent consensus conference, no longer recommended as a categorisation of the severity of ARDS. Recommended categorisation of ARDS now changed to Mild, Moderate and Severe.

APL: (Transpulmonary pressure) This is the pressure distending the respiratory system (and the functional residual capacity of the lung) and is the airway pressure minus the pleural pressure. (**APL =** Ppause – Poes). However, Poes (equivalent to pleural pressure) and FRC measurement at the bedside are not common in clinical practice.

ARDS: Acute Respiratory Distress Syndrome.

Atelectrauma: Lung injury caused by the cyclic collapse and reopening of unstable small airways and alveoli resulting in 'shear injury'.

Auto-triggering: The inadvertent triggering of inspiratory ventilator support when a patient is not breathing.

Barotrauma: Lung injury due to high airway (distending) pressure.

Biotrauma: A diffuse lung injury and possible injury to other organs due to the release of inflammatory mediators.

CaO2: Content of oxygen in arterial blood. CaO2 is calculated as 1.34 × Hb × SaO2; the normal value is 16 to 20 mL O2/100 mL blood.

+ Compliance

Crs: (Compliance of the respiratory system) It is defined as the lung volume change per unit airway pressure change or the slope of the pressure–volume curve. In positive pressure ventilation, it is measured by dividing the Vt by the inflation pressure. See below for dynamic (**Cdyn**)and static (**Cstat**) compliance.

Ccw: Compliance of the chest wall.

Cdyn: Dynamic compliance. It is calculated as Vt / (Peak Paw - PEEP)

CL: Lung compliance.

Cqs: (Compliance–quasi-static) Compliance derived from measurements made during a 'relaxed' double prolonged occlusion manoeuvre i.e. during a four second pause at end-inspiration and at endexpiration. It mimics true static compliance and is termed quasi-static compliance. True static compliance is utilized mainly in research and is performed using pressure measurements after serial volume increments with a 'super syringe'.

Cstat: Static compliance (see above). It is calculated asVt / (Ppause - PEEP)

CO: Cardiac output.

COHb: Carboxyhaemoglobin.

CPAP: (Continuous positive airway pressure) Refers, by convention, to the end-expiratory airway pressure in a spontaneous breathing respiratory system.

C$\bar{v}$O2: Mixed venous oxygen content. It is measured as 1.34 × Hb × S$\bar{v}$O2 (mixed venous oxygen saturation).

Δ**PCO2:** Difference between arterial to end-tidal PCO2.

De-escalation: A continuous effort to reduce the mechanical ventilator support as soon and as much as possible.

DO2: Oxygen delivery – measured as CO × CaO2.

EtCO2: End-tidal CO2 – see also PetCO2.

EVLW: Extravascular lung water.

EWS: Expert weaning system.

FRC: (Functional residual capacity) The volume of gas in the patient's respiratory system at end-expiration. Its capacity is a key determinant of oxygenation.

Fr: (Frequency) The number of ventilatory or patient breaths per minute; also termed the ventilatory (or respiratory) rate.

Hb: Haemoglobin content of blood. Usually expressed as in g% or as g/100 mLs (normal value varies between males and females but is approx. 15 g/dL).

Hypercapnia: More than the normal level of carbon dioxide in the blood.

Hypocapnia: Less than the normal level of carbon dioxide in the blood.

Hypoxaemia: An abnormally low PO2 in arterial blood.

I:E ratio: The ratio between the time (duration) of inspiration relative to duration of expiration. It is normally 1:1.5 to 1:2.

Impedance: The combined effects of airway resistance, respiratory system (including chest wall) compliance and intrinsic PEEP (PEEPi – see below) in opposing the flow and volume change produced by the ventilator.

k : Constant that represents the alveolar end-expiratory pressure (in the 'driving pressure' equation).

LSF: Least square fitting.

MetHb: Methaemoglobin.

MIP: Maximal inspiratory pressure, see also PImax.

MVV: Maximum voluntary ventilation.

NI(M)V: Non-invasive (mechanical) ventilation.

NIF: Negative inspired force.

Normoxaemia: Normal blood levels of oxygen.

PaCO2: Partial pressure of arterial carbon dioxide – normal range 4.7–6 kPa (35–45 mmHg).

PaIv: Alveolar pressure.

PAO: Pressure at airway opening.

PaO2: Partial pressure of arterial oxygen – normal range 10–13.3 kPa (75–100 mmHg).

Patm: Atmospheric pressure.

Paw: Airway pressure.

PCV: Pressure-controlled ventilation.

Peak airway pressure: The peak (or highest) pressure measured by the ventilator; the pressure at the level of the major airways.

PECO2: Partial pressure of CO2 in mixed expired gas – usually collected/measured in a Douglas bag but not a standard clinical measurement.

PEEP: (Positive end-expiratory pressure) Defined as an elevation of airway pressure at the end of expiration. End-expiratory pressure is normally zero (atmospheric) during spontaneous breathing but is often set at a positive level (measured in cms H2O) during mechanical ventilation.

PEEP: (External) The PEEP effected by the ventilator and set by the operator.

PEEPi: (PEEP Intrinsic) Elevated positive end-expiratory pressure which is 'intrinsic to the patient'. It is associated with certain lung pathologies particularly where there is destructive lung disease, dynamic collapse of airways and active expiration. It is caused by insufficient expiratory time or a limitation on expiratory flow and dynamic pulmonary hyperinflation may result. It is measured during a prolonged, 'relaxed' expiratory ventilatory pause.

Total PEEP: Is the combination of the above two pressures. However, in certain circumstances the effect of external PEEP may be to reduce the level of PEEPi.

Pes: (oesophageal pressure) Ppressure in the lower one third of the oesophagus when the patient is upright. It equates to pleural/extra-alveolar pressure.

PetCO2: (End-tidal CO2) The highest value of CO2 partial pressure during the alveolar plateau of the capnography curve.

PImax: Maximal inspiratory pressure.

PIP: Peak inspiratory pressure.

Pmax: The sum of the pressures produced by the ventilator to overcome the elastic and resistive forces (airways and endotracheal tube) of the respiratory system.

Pmus: Pressure generated by muscle contraction.

Ppause: The airway pressure observed during prolonged (4-second), 'relaxed' end-inspiratory pause/hold. Also termed Plateau(Pplat) or End-inspiratory hold

pressure: It is used in the determination of static compliance. In the absence of airflow (no resistance), it represents the pressure applied to the small airways and alveoli during peak inspiration. It depends on a number of factors including the Vt, PEEP, intrinsic PEEP and compliance.

PPV: (Positive pressure ventilation) Process of exerting a pressure, which is positive relative to atmospheric pressure, to achieve entry of air or respiratory gases into the lungs. Term IPPV used for Intermittent Positive Pressure Ventilation.

Ppl: Pleura pressure.

Prs: Respiratory system pressure.

PSG: Polysomnography.

PSV: Pressure support ventilation.

R: (Resistance) Respiratory system resistance

(**Rrs):** refers to airway resistance and comprises the inflating pressure divided by the (gas) flow.

Recruitment manoeuvres: Manually or ventilator-assisted lung inflation to achieve an increase in FRC (by 'alveolar recruitment') and thereby an improved oxygenation.

RCe: (Respiratory system expiratory time constant) The product of resistance and compliance and quantifies the speed of exhalation. It may vary between different lung units in pathological circumstances.

Rmax: Total resistance.

SaO2: Oxygen saturation percentage of the available haemoglobin (normal value is 98%).

Shunt: Is due to perfusion of non-ventilated lung regions and is the commonest cause of clinical hypoxaemia. Extrapulmonary causes are those (right to left shunts) that may occur in the presence, for example, of an atrial septal defect (ASD).

Te: (Expiratory time) The time from the start of expiratory flow to the start of inspiratory flow.

Ti: Inspiratory time.

Transthoracic pressure: The pressure in the pleural space measured relative to the pressure of the ambient atmosphere outside the chest.

Trigger: Usually relates to inspiratory rather than expiratory triggering (see below) and as such, it refers to the process of initiating the inspiratory breath of the ventilator. Inspiratory triggering is usually effected by a pressure change or flow change in the breathing system generated by patient effort.

Triggering: The mechanism of initiating the inspiratory (and expiratory) phase(s) of the ventilator function.

TTOT: is the respiratory duty cycle

V: Volume.

Flow: (Volume per unit of time).

VALI: (or **VILI)** Ventilator-associated lung injury or Ventilator-induced lung injury.

VAP: Ventilator-associated pneumonia.

VCV: Volume-controlled ventilation.

VA: (Alveolar volume) The proportion of Vt that is useful in gas exchange..

V'A: (Alveolar ventilation) The proportion of Vm that is useful in gas exchange. It is comprised of Alveolar volume (VA) multiplied by respiratory rate (Fr) i.e. V'A = VA × Fr. V'A is directly proportional to CO2 elimination.

Vd: (Dead space) Respiratory system areas that are ventilated but not perfused. Or the volume of the airways filled with inspired gas that does not take part in gas exchange.

Vd/Vtphys: Physiologic dead space.

Vdalv: Alveolar dead space (alveoli well ventilated but receiving minimal blood flow).

Vdanat: Anatomic dead space (upper and lower airways).

Vdins: Instrumental dead space i.e. the dead space resulting from parts of the breathing system, ventilator equipment, endotracheal tubes, humidification devices and connectors. It is considered part of the anatomic dead space.

V,ee: (End-expiratory lung volume) The volume of gas in the patient's respiratory system at end-expiration. Though, it is often used interchangeably with FRC (see above), this acronym should be used only for patients mechanically ventilated and receiving PEEP.

Ventilation mode: Represents a specific operating logic (or software program) for the mechanical ventilator, based on one or more approaches to respiratory cycle management. The specific mode is chosen by the operator.

Vm: (Minute Volume) The volume of gas ventilating the respiratory system per minute. It is comprised of Tidal volume multiplied by the Respiratory rate (Vt × Fr).

Ve: Expired minute volume.

Vi: Inspired minute volume.

VO2: Oxygen consumption by the tissues.

Volutrauma: Lung injury due to alveolar overexpansion secondary to high lung volume (with or without high pressure).

Vt: (Tidal volume) The volume of gas intermittently inhaled or exhaled, by the patient or ventilator, with each breath 'on top of' the volume of the functional residual capacity (FRC).

Weaning: Is the final step in de-escalation, involving the patient's complete and continuing freedom from mechanical support and removal of the artificial airway.

Wexp: Work of breathing performed during the expiratory phase.

Winsp: Work of breathing performed during the inspiratory phase of the cycle.

WOB: (Work of breathing) The work required to accelerate gas in the airways, to overcome airway resistance and to expand the elastic lung tissue so that air can be brought into the lungs and then exhaled.

Wpat: Work of breathing performed by the patient.

Wvent: Work of breathing performed by the ventilator.