Respiratory assessment and monitoring

Skills and techniques

Update 2012 (pdf)

Module Authors (Update 2012)

Luigi CAMPOROTA	Department of Critical Care, Guy's & St Thomas' NHS Foundation Trust, London, UK
Francesca RUBULOTTA	Imperial College, Charing Cross Hospital, London, UK

Module Authors (first edition)

Francesca Rubulotta & Antonino Gullo	Dept of Anaesthesia and Intensive Care Policlinico University Hospital Catania, Italy
Umberto Lucangelo	Dept of Perioperative Medicine, Intensive Care and Emergency Medicine, University Hospital Trieste, Italy
Graham Ramsay	Medical Director West Hertfordshire Hospitals NHS Trust, United Kingdom

Module Reviewers	Lluis Blanch, Ola Stenqvist, Janice Zimmerman
Section Editor	Anders Larsson

Respiratory assessment and monitoring
Update 2012

Editor-in-Chief	Dermot Phelan, Intensive Care Dept, Mater Hospital/University College Dublin, Ireland
Deputy Editor-in-Chief	Francesca Rubulotta, Imperial College, Charing Cross Hospital, London, UK
Medical Copy-editor	Charles Hinds, Barts and The London School of Medicine and Dentistry
Self-assessment Author	Hans Flaatten, Bergen, Norway
Editorial Manager	Kathleen Brown, Triwords Limited, Tayport, UK
Business Manager	Estelle Flament, ESICM, Brussels, Belgium
Chair of Education and Training Committee	Marco Maggiorini, Zurich, Switzerland

PACT Editorial Board

LEARNING OBJECTIVES

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

1. Recognise acute lung diseases through history, clinical manifestations and imaging
2. Understand the relationship between PaO_2, SaO_2 and arterial oxygen content and the use of pulse oximetry
3. Evaluate respiratory function using end-tidal CO_2 measurements, analysis of capnographic curves, and dead space calculations
4. Interpret airway pressure and flow tracings and oesophageal pressure tracings
5. Select the appropriate parameters to monitor during mechanical ventilation and weaning.

FACULTY DISCLOSURES

The authors of this module have not reported any disclosures.

DURATION 7 hours

Contents

INTRODUCTION

Respiratory failure, the condition in which the respiratory system is unable to maintain adequate gas exchange to satisfy metabolic demands (oxygenation and/or elimination of carbon dioxide), is the most frequent cause of admission to the intensive care unit (ICU). Several diseases can impair the function of the respiratory system and although specific treatment for the underlying disease may differ, the ability to assess, interpret and monitor physiological changes in the respiratory function over time is essential to providing optimal supportive treatment and detecting the physiological response to therapeutic interventions.

The aim of this module is to provide a systematic approach to evaluating and monitoring patients with respiratory impairment. Monitoring is the assessment of a patient at predetermined intervals, repeatedly or continuously, with the intention of detecting abnormalities and triggering a response if an abnormality is detected. This starts with simple skills and devices and can be later supported by the increasingly more sophisticated equipment now available at the bedside. Critical care staff need to be familiar with the most common respiratory monitoring devices and techniques and develop an awareness of the more sophisticated monitoring modalities being adopted into respiratory critical care.

The initial assessment of a patient with respiratory failure requires a thorough clinical history and physical examination in conjunction with baseline investigations. Further respiratory monitoring is necessary to assess response to treatment and outcome.

Much of the material of this module relates to patients undergoing mechanical ventilation and it and the glossary of terms can be read in conjunction with the PACT module on Mechanical ventilation.

1/ HOW TO RECOGNISE LUNG DISEASES

It is fundamental to perform a full and systematic clinical examination on all critically ill patients who are to be admitted to the ICU. The initial clinical examination provides a baseline reference. It is essential for the differential diagnosis and treatment planning.

See the PACT module on Clinical examination.

The clinical examination of the respiratory system comprises history taking, physical examination (inspection, palpation, percussion and auscultation) and the evaluation of laboratory data and radiological findings.

Clinical history

The history taking includes the past medical and surgical history, current medications, as well as the presenting complaint.

Information about risk factors for lung disease is obtained:

- A history of current or previous smoking is noted and a record made of the number of years the patient has smoked, the number of cigarettes per day and the interval since smoking cessation.
- A history of significant passive exposure to smoke may be a risk factor for neoplasia or an exacerbating factor for airway diseases such as chronic obstructive lung disease.
- A history of sleep-disordered breathing is typical in obese patients. A complete history is collected comparing the patient's subjective symptoms with the objective sleep history reported by the family members. The pathologic increase in the $PaCO_2$ (partial pressure of carbon dioxide in the arterial blood) modifies the strength of the history reported by these patients. Obese patients suffering from sleep disorders may complain of early morning headache, daytime somnolence, and apnoea or shortness of breath during night-time. These disorders are also important as predictors of difficult intubation.
- Exposure to inhaled agents associated with lung disease is ascertained. Among these are inorganic dusts (especially asbestos and silica) and organic antigens (especially antigens from moulds and animal proteins). Asthma is often exacerbated by exposure to environment allergens or occupational exposure.
- Exposure to infectious agents can be suggested in previously healthy people having contact with individuals with known respiratory infections (tuberculosis). Healthy people travelling in specific areas of the world can be exposed to pathogens. For an appropriate management plan, a detailed travel history is important.
- Infections of the respiratory system should be suspected in all immunocompromised patients (oncology/haematology patients, transplants, HIV/AIDS). Immunisation status is evaluated in children and in the newborn or in adults with splenectomy.

    See the PACT module on Immunocompromised patients.

- Systemic rheumatic diseases (such as rheumatoid arthritis) are sometimes the cause of pleural and parenchymal lung diseases.
- Patients with a history of motor neuron diseases such as amyotrophic lateral sclerosis, neuromuscular junction diseases such as myasthenia gravis, immune-mediated neuropathies such as Guillain-Barré syndrome, or myopathies, might have multiple admissions to ICUs. These patients may need long-term non-invasive ventilation, perhaps via a tracheostomy.
- Treatment of non-respiratory disease can be associated with respiratory complications, either because of effects on host defence mechanisms (immunosuppressive agents, chemotherapy drugs) with resulting infection or because of direct effects on the pulmonary parenchyma (amiodarone) or on the airways (β-blockers, angiotensin-converting enzyme inhibitors).
- Family history is important for evaluating genetic risk factors (cystic fibrosis, α-antitrypsin deficiency, pulmonary hypertension, asthma) and predisposition for lung diseases.
- The past surgical history should pay particular attention to all operations performed in the neck, throat and thorax of the patient. It is important to exclude lesions of the phrenic nerve after surgery in the cervical or thoracic region. Pre-operative and postoperative lung capacities should be reported on the patient's record if the history taking is positive for a pneumonectomy, lobectomy or atypical lung resection.

Clinical signs/features of respiratory diseases

Common manifestations of respiratory diseases on admission are cough, sputum, haemoptysis, dyspnoea (shortness of breath), cyanosis, chest pain, altered mental status and clubbing of the fingers and toes.

Cough

Cough is the most frequent of all respiratory symptoms. There are various types of cough. It can be dry or productive of sputum; it can be **acute** (<3 weeks), **sub-acute** (>3 weeks) or **chronic** (>8 weeks). Chronic cough is common among tobacco smokers, and can occur in asthmatics, in patients with gastro-oesophageal reflux or on ACE inhibitors. Cough associated with inflammation of the pleura (pleurisy) is characteristically dry and short. Here the act of coughing causes pain owing to the movement of the inflamed pleura, and so the cough is cut short by the pain. Cough is accompanied by purulent sputum in bacterial infections.

Sputum

Sputum varies in amount and character according to the nature and extent of the lung disease. Sometimes in the early stages of disease, sputum may be absent and appears later when the lesion in the respiratory tract has progressed. Yellow sputum usually indicates a large number of white cells and underlying infection. However, light yellow sputum might be seen in patients with asthma because of a high sputum eosinophil count. Green discolouration indicates

3

stagnation of mucus, and red or brownish ('rusty') sputum is caused by the presence of red blood cells.

Haemoptysis

Haemoptysis of all grades of severity may occur, from slight streaking of the sputum with blood, which is a common symptom in acute and chronic bronchitis, to a massive haemorrhage (defined as >200–600 mL or 1–2 cups). Bronchial carcinoma, pulmonary infarction, pulmonary tuberculosis, bronchiectasis and mitral stenosis are the most common causes of massive bleeding.

Dyspnoea

Dyspnoea occurs as a symptom in a wide variety of lung and heart diseases. It is defined as the subjective experience or perception of uncomfortable breathing. It should be distinguished from **hyperpnoea**, where the minute ventilation is increased, but no abnormal sensation is felt, and **tachypnoea**, an excessive respiratory rate.

> **NOTE** In children, respiratory rate must be evaluated according to age.

Cyanosis

Cyanosis depends on the absolute amount of reduced haemoglobin in the blood. Cyanosis is evident when reduced haemoglobin exceeds 5 g/100 ml. **Peripheral** cyanosis is due to a greater oxygen extraction by the tissues from normally saturated arterial blood (normal SaO_2) when the circulation is impaired by vasoconstriction or low cardiac output. **Central** cyanosis is due to haemoglobin desaturation (low SaO_2) from poor gas exchange in the lungs, an abnormal haemoglobin derivative or the presence of a right to left shunt (e.g. congenital heart disease). A combination of central and peripheral cyanosis may occur as, for example, in cardiogenic shock with pulmonary oedema.

> ⚠️ Cyanosis is very difficult to see in anaemic patients, and in severe anaemia even marked arterial desaturations may not lead to the manifestation of cyanosis. The advice should be to always use pulse oximetry for correct diagnosis.

Chest pain

Chest pain caused by diseases of the respiratory system frequently originates from involvement of the parietal pleura. Chronic or recurrent chest pain may reflect pulmonary vascular or pleural disorders.

Physical examination

The physical examination should be directed both to lung and thoracic abnormalities and to generalised findings that may reflect underlying lung diseases. Normally the findings on physical examination of the chest are equivalent on both sides.

Finger clubbing

Clubbing of the digits (hypertrophic osteoarthropathy) may be hereditary, idiopathic, occupational (pneumatic hammer operators) or can be found in association with: metastatic lung cancer, interstitial lung disease and chronic lung infections such as lung abscess and empyema.

The chest

On inspection, the rate and pattern of breathing, as well as the depth and symmetry of lung expansion, are examined. Breathing that is associated with the use of accessory muscles indicates an increase in the work of breathing (see Task 3). A note should be made of the rate and characteristics of the breathing pattern, the type and severity of the cough and the amount and character of the sputum. Asymmetric expansion of the chest is always due to a localised process affecting one or other lung (e.g. endobronchial obstruction of the airway or phrenic nerve paralysis).

Thoracic abnormalities such as kyphoscoliosis and ankylosing spondylitis are recorded on inspection because of the related decrease in total lung capacity and increase in the risk of pneumonia. Skeletal abnormalities such as an increase in the antero-posterior diameter of the chest could be due to severe emphysema. Enlarged lymph nodes in the cervical and supraclavicular regions are evaluated, as they may be associated with several diseases, including cancer. Peripheral oedema (lower extremities) may be related to pulmonary vascular hypertension and right ventricular failure. It is wise to consider pulmonary hypertension in every patient with chronic respiratory failure.

> ⚠️ In patients with chronic respiratory failure, look for signs of cor pulmonale, in particular raised jugular venous pressure, signs of tricuspid regurgitation (TR), loud S2 and hepatomegaly. If these signs are present, it is appropriate to perform a transthoracic echocardiogram to look at TR jet velocity and to estimate pulmonary artery (PA) pressures.

On palpation, findings observed by inspection may be confirmed. The symmetry of lung expansion can be assessed. The chest wall should be carefully examined for soft tissue abnormalities such as cutaneous lesions, subcutaneous swelling or subcutaneous emphysema (crepitation on palpation), bulging or retraction of intercostal spaces. The consistency of lymph nodes is noted.

By percussion the sound of a normal lung is resonant while the consolidated lung or a pleural effusion is dull, and emphysema is hyperresonant.

On auscultation the quality and intensity of breath sounds are assessed using a stethoscope. The categories of findings include normal breath sounds, decreased or absent breath sounds, and abnormal breath sounds. Normal breath sounds are described as 'vesicular'. Vesicular sounds are smooth, low tone, and widespread over the thorax of normal patients.

Vesicular sounds are louder and longer during inspiration than expiration. These sounds are generated by air movements in the bronchi modified by the gas content in terminal bronchioli and the alveoli. Reduced breath sounds may reflect reduced airflow to a lung region due to its over-inflation (e.g. emphysema) or by the presence of air or fluid in the pleural space, or sometimes increased pleural thickness. When sound transmission is increased through consolidated lung with patent airways the sound in more pronounced in the expiratory phase and is of more tubular quality and is named a 'bronchial breath sound'. There are several types of abnormal breath sounds: rales, rhonchi and wheezes are the most common. Crackles (rales) are discontinuous, generally inspiratory, clicking, bubbling or rattling sounds. They are believed to occur when air opens closed alveoli (air spaces). Rhonchi are sounds that resemble snoring. They are produced when air movement through the large airways is obstructed or turbulent. Wheezes (sometimes called 'fine rhonchi') are high-pitched, musical sounds produced by narrowed airways, often occurring during expiration. Wheezing can sometimes be heard without a stethoscope. Pleural friction or rub is a diagnostic sign of pleural inflammation. It is a grating or creaking sound, unaltered by coughing, audible during both inspiration and expiration. Stridor is a specific sound, usually inspiratory, secondary to obstruction of upper airways.

Some diseases that most commonly affect the respiratory system, such as sarcoidosis, can have findings on physical examination not related to the respiratory system, including ocular findings (uveitis, conjunctival granulomas) and skin findings (erythema nodosum).

Management plans and differential diagnosis should be formulated following the history taking, physical examination, and review of available laboratory data and lung imaging (X-rays, computed tomography (CT) scan, ultrasound).

In your next ten patients, check the quality of your history taking and physical examination: how complete are they, how do you judge consistency? Ask a colleague to observe you while you take a history and perform a physical examination.

Investigations

Investigations used for the chest include radiological techniques and fibre optic techniques such as bronchoscopy.

In the practice of intensive care medicine, knowledge of imaging techniques for the chest, particularly of the chest X-ray, is mandatory.

> X-ray dose limits were unknown, and now these are the main concern

See PACT module on Clinical imaging.

Q. Which types of X-ray imaging are available?

A. Types of X-ray imaging include: plain radiographs, conventional tomography, fluoroscopy, digital subtraction angiography (DSA), and computed tomography.

Chest imaging

The plain chest radiograph is the most common radiological investigation in intensive care practice. A number of studies have shown that daily chest radiographs frequently demonstrate new, unexpected, or changing abnormalities, which result in changes in therapy. However, more recent studies support adoption of an on-demand strategy in preference to a routine strategy with a view to decreasing the use of chest radiographs in mechanically ventilated patients without a reduction in patients' quality of care or safety.

Hejblum G, Chalumeau-Lemoine L, Ioos V, Boëlle PY, Salomon L, Simon T, et al. Comparison of routine and on-demand prescription of chest radiographs in mechanically ventilated adults: a multicentre, cluster-randomised, two-period crossover study. Lancet 2009; 374(9702): 1687–1693. PMID 19896184

Oba Y, Zaza T. Abandoning daily routine chest radiography in the intensive care unit: meta-analysis. Radiology 2010; 255(2): 386–395. PMID 20413752

The plain radiograph is used both to provide anatomical information and to evaluate changes in the heart and lungs. In addition, it provides important data about abdominal contents just below the diaphragm (e.g. gas under the diaphragm) and the anatomy of the airway. On the other hand, over-interpretation of subtle changes on chest X-rays (especially portable X-rays) due to a change in exposure or technique may lead to erroneous assumptions (e.g. diagnosis of pulmonary oedema in particular).

A chest radiograph should be routinely obtained after the insertion of a subclavian or internal jugular central venous catheter, to confirm the correct placement of the catheter and to exclude a complication e.g. pneumothorax. Moreover, in the ICU patient a chest X-ray should be obtained after any intubation to exclude a complication and to evaluate the position of the tracheal tube. In all other patients, chest radiographs should be ordered only when needed.

See the PACT module on Clinical imaging.

> ⚠️ All Critical Care practitioners should be able to make a rapid diagnosis of life-threatening conditions such as pneumothorax as well as use radiological investigations to confirm the safe placement of tracheal tubes, nasogastric tubes, chest drains or vascular catheters.

> **NOTE** A normal chest X-ray does not rule out the following chest pathologies.

Diseases with no or minimal radiological features:
- Obstructive airway disease (e.g. asthma, moderate emphysema, bronchitis, bronchiolitis)
- Small lesions (e.g. masses of <1 cm diameter, endobronchial lesions without collapse or consolidation)
- Pulmonary emboli without infarction
- Early stages of infection/pneumonia
- Pulmonary fibrosis (early)

Interpretation of a bedside film is fraught by numerous pitfalls. In addition, a portable chest radiograph may be difficult to interpret due to poor positioning of the patient. Because lateral chest films cannot easily be obtained in the ICU, abnormalities in the posterior costophrenic area, within the mediastinum and adjacent to the spine, can be missed.

Q. Are ICU X-rays as reliable diagnostically as those done in the radiology department?

A. No, most X-rays performed on the critically ill are done in the ICU using mobile equipment with the patient in a bed. As such, these films are suboptimal, and this should be borne in mind when making comparisons with those taken in the radiology department, or with previous films.

Q. What are the differences between X-rays performed in the ICU and those taken in the radiology department?

A. The chest radiograph in the ICU is antero-posterior (A-P) rather than postero-anterior (P-A) as in the radiology department. On the A-P view, the heart and the mediastinum are about 15% wider than on an upright P-A chest radiograph, because of the increased distance of the heart from the film. Further magnification can also be due to the fact that the portable radiography is performed with the X-ray tube closer to the patient. A false cardiomegaly or a wide mediastinum are often erroneously presumed.

Q. What is the position of choice for performing chest X-rays in the ICU?

A. Patients, if possible, should be in the sitting or semi-erect position. This is necessary because pleural effusion can easily be missed in the supine position.

Fluids track posteriorly, resulting in a diffuse haziness of the lung fields. Fluid collections can be confirmed by ultrasonography. The pulmonary vasculature is distorted because blood no longer flows preferentially to the lower lobes in all supine patients. Bedside, changes in the lung blood flow can mimic signs of congestive heart failure. Every effort should be made to position the patient upright.

> ## Q. What are limitations to achieving this position in ICU?

A. The main limitations to this may be haemodynamic instability, kinking of femorally placed intra-aortic balloon pumps (IABPs), spinal injuries and hip fractures.

See the PACT modules on Clinical imaging and Acute respiratory failure.

The chest radiograph should be studied systematically: first the quality of images and the patient's position; then the location of all tubes and catheters, together with the evaluation of ribs, vertebrae, lung parenchyma, pleura, mediastinum and diaphragm; and lastly the assessment of signs of extra-alveolar air.

Computed tomography

Use of the computed tomography (CT) scan in the diagnosis of lung diseases includes the following indications:

- Investigation of pulmonary pathology
- Assessment of the mediastinum
- Tumour staging
- Interventional procedures such as biopsy
- High-resolution CT technique is used to assess interstitial pulmonary disease
- Assessment of thoracic trauma
- Assessment of aorta and blood vessels
- CT pulmonary angiography for suspected embolism

CT is a very useful and frequently employed technique in intensive care medicine. Conventional CT scan and high-resolution computed tomography (HRCT) are both used for evaluating aortic dissection, pleural disease and mediastinal masses, but HRCT is better for studying diffuse infiltrative lung diseases (e.g. in immunocompromised patients with pulmonary infiltrates). Spiral CT is most helpful in evaluating lesions at, or near, the diaphragm (less motion artefact), vascular structures (main pulmonary arteries in suspected pulmonary embolism), and small pulmonary nodules.

For more information see the PACT module on Clinical imaging.

CT scanning in ARDS

ARDS can be derived from two pathogenetic pathways: a direct insult to lung epithelial cells (pulmonary ARDS, ARDSp) or involve the lung indirectly (extrapulmonary ARDS, ARDSexp). The radiological (X-rays and CT scan) pattern in ARDSp has been said to be characterised by a prevalent asymmetry with a mix of dense parenchymal opacification and ground glass opacification

while the ARDSexp by a more diffuse pattern, a prevalent ground glass or reticular pattern reflecting an active inflammatory process involving the lung interstitium and abnormal thickening of the alveolar wall. However, it is difficult to discern between the two aetiologies. More information regarding ARDS can be found in the PACT module on Acute respiratory failure and in the following reference.

Desai SR. Acute respiratory distress syndrome: imaging of the injured lung. Clin Radiol 2002; 57(1): 8–17. Review. PMID 11798197

ARDS is characterised by a marked increase in lung weight and a heterogeneous loss of aeration. In this context, CT can be useful in quantifying lung inflammatory oedema, the proportion of alveolar collapse and increase in aeration following increase in level of positive end-expiratory pressure (PEEP), i.e. lung recruitability. CT, by estimating the volume of aerated lung, can identify patients who require lower tidal volume ventilation (patients with a larger non-aerated lung compartment); higher level of PEEP (patients with patchy or diffuse alveolar opacifications). A substantial decrease in non-aerated lung tissue between CT images taken at 5 cmH$_2$O PEEP relative to that at a recruitment pressure of 45 cmH$_2$O plateau pressure is indicative of successful recruitment.

However, quantitative analysis of the whole lung remains an elaborate and time-consuming process, which, together with the need to transport patients to the radiology department, precludes routine use of CT to assess disorders of lung aeration. All ARDS patients are now treated with low tidal volume ventilation – a lung protective strategy.

See PACT modules on Acute respiratory failure and Mechanical ventilation.

Gattinoni L, Pelosi P, Suter PM, Pedoto A, Vercesi P, Lissoni A. Acute respiratory distress syndrome caused by pulmonary and extrapulmonary disease. Different syndromes? Am J Respir Crit Care Med 1998; 158(1): 3–11. PMID 9655699

Desai SR, Wells AU, Suntharalingam G, Rubens MB, Evans TW, Hansell DM. Acute respiratory distress syndrome caused by pulmonary and extrapulmonary injury: a comparative CT study. Radiology 2001; 218(3): 689–693. PMID 11230641

Gattinoni L, Caironi P, Pelosi P, Goodman LR. What has computed tomography taught us about the acute respiratory distress syndrome? Am J Respir Crit Care Med 2001; 164(9): 1701–1711. Review. No abstract available. PMID 11719313

Gattinoni L, Pelosi P, Crotti S, Valenza F. Effects of positive end-expiratory pressure on regional distribution of tidal volume and recruitment in adult respiratory distress syndrome. Am J Respir Crit Care Med 1995; 151(6): 1807–1814. PMID 7767524

Malbouisson LM, Muller JC, Constantin JM, Lu Q, Puybasset L, Rouby JJ; CT
    Scan ARDS Study Group. Computed tomography assessment of positive
    end-expiratory pressure-induced alveolar recruitment in patients with
    acute respiratory distress syndrome. Am J Respir Crit Care Med 2001;
    163(6): 1444–1450. PMID 11371416

Pelosi P, Rocco PR, de Abreu MG. Use of computed tomography scanning to
    guide lung recruitment and adjust positive-end expiratory pressure. Curr
    Opin Crit Care 2011; 17(3): 268–274. PMID 21415738

Magnetic resonance imaging

Magnetic resonance imaging (MRI) requires special ventilation and monitoring
equipment, because of the strong magnetic field.

Some indications for MRI extend beyond those for CT scanning. MRI usually
does not require the use of intravenous contrast agents to identify blood vessels.
It is possible to differentiate between a dilated pulmonary vessel and a hilar
mass without using contrast. The reason is that flowing blood has no signal on
MRI images and consequently appears black. The following are indications for
using MRI rather than CT, most of which are not usual Critical Care issues.

Evaluation of:

- Thoracic aorta
- Mediastinal masses/Pancoast tumour
- Lymph nodes
- Vascular lesions such as arteriovenous malformations

Lung ultrasound

Lung ultrasound (LUS) is a useful bedside tool for assessing lung parenchyma,
pleural surfaces, pleural spaces and chest wall in critically ill patients. Normally,
ultrasound waves are not transmitted through the air-filled lung, however, some
pathological conditions lead to an increase in lung tissue density which
generates specific signs and patterns that can be assessed and monitored by
LUS.

LUS plays an important role in the following conditions:

- Diagnosis and estimation of volume and nature of pleural effusion
- Diagnosis of pneumothorax
- Guiding chest drain insertion
- Diagnosis of alveolar-interstitial syndrome (see below)
- Diagnosis of atelectasis and pulmonary consolidation

Pleural effusions: Ultrasound allows accurate assessment of the presence,
type and quantity of pleural effusion. Pleural effusion is detected as a
hypoechoic and homogeneous structure. Ultrasound characteristics of pleural
effusion may indirectly suggest the nature of pleural effusion; transudates are
always anechoic whereas exudates often appear echoic and loculated. The
volume of the pleural effusion is measured by the maximal distance between the

two layers of the pleura, visceral and parietal, at the posterior axillary line at the end of expiration in supine patients. The volume in mL of pleural fluid can be estimated multiplying the maximal distance between the two pleura layers by 20 (V (mL) = 20 × pleural distance (mm)). LUS has a high sensitivity and specificity in detecting effusions of volume between 500–1000 mL.

Pneumothorax: In normal conditions, the pleural line is identified 5 mm from the rib cortex and appears as a shimmering linear echogenic structure that moves with respiratory phase ('*lung sliding*') or with cardiac movements ('*lung pulse*'). The absence of lung sliding and/or the presence of 'A' lines (horizontal parallel hyperechoic artefacts arising from the pleural line, present in normally aerated lung) presents high sensitivity and specificity and 100% negative predictive value in the diagnosis of pneumothorax (in the absence of previous pleurodesis).

LUS can also identify and estimate the extent of a partial pneumothorax, through the presence of a '*lung point*' – intermittent visualisation of lung sliding from mobile partially collapsed lung – that indicates the transition between the patterns seen in pneumothorax (absent lung sliding plus 'A lines') during expiration and lung pattern (lung sliding or pathological comet-tail artefacts) during the inspiration.

Guiding placements of chest drains: Well established and routinely used by interventive radiologists in the drainage, for example, of loculated pleural collections.

Identifying alveolar-interstitial syndrome (AIS): Condition characterised by a decrease in lung aeration, diffuse thickening of the interstitial or alveolar compartment through oedema or fibrosis. AIS is identified by the presence of hyperecogenic, regularly spaced vertical B-lines '*comet-tail*' artefacts projecting from the pleural line, caused by an increase in non-aerated lung tissue. The presence of more than three B-lines indicates abnormal lung parenchyma. The number of these vertical B-lines depends on the degree of loss of lung aeration. Multiple lines 7 mm apart are caused by thickened interlobular septa characterising interstitial oedema (B7 lines). In contrast, lines 3 mm or less apart are caused by alveolar oedema (B3 lines).

Identifying lung consolidation (pulmonary contusion, pneumonia, atelectasis) – heterogeneous hypoechoic tissue structure that is poorly aerated. Within the consolidated lung, hyperechoic punctiform or linear artefacts, corresponding to the air bronchograms, can be seen.

Atelectasis: LUS allows detection and differentiation of atelectasis into compression atelectasis (presence of atelectatic lung, bronchogram and pleural effusion) and resorption atelectasis ('lung pulse' recognised as the absence of lung sliding with the perception of heart activity at the pleural line).

Pulmonary abscess: May be discussed with your radiology colleagues but more likely to need a CT.

Lung recruitment, optimisation of positive end-expiratory pressure (PEEP): LUS can be used to monitor gain in lung aeration following a lung recruitment or to guide PEEP setting or to evaluate resolution of pneumonia. To

that end, a lung re-aeration score has been proposed. The score assesses changes in LUS pattern (e.g., lung comets with well-defined and irregular spacing; abutting ultrasound lung comets; alveolar consolidation or normal pattern) in multiple regions of the lung at different time-points. An increase in lung ultrasound re-aeration score of +8 or higher has been associated with a PEEP-induced lung recruitment greater than 600 mL. An ultrasound lung re-aeration score of +4 or less was associated with a PEEP-induced lung recruitment ranging from 75 to 450 mL. However, LUS score was unable to estimate hyperinflation.

van der Werf TS, Zijlstra JG. Ultrasound of the lung: just imagine. Intensive Care Med 2004; 30(2): 183–184. Epub 2003 Dec 19. PMID 14685657

Koenig SJ, Narasimhan M, Mayo PH. Thoracic ultrasonography for the pulmonary specialist. Chest 2011; 140(5): 1332–1341. PMID 22045878

Bouhemad B, Brisson H, Le-Guen M, Arbelot C, Lu Q, Rouby JJ. Bedside ultrasound assessment of positive end-expiratory pressure-induced lung recruitment. Am J Respir Crit Care Med 2011; 183(3): 341–347. Epub 2010 Sep 17. PMID 20851923

Bouhemad B, Zhang M, Lu Q, Rouby JJ. Clinical review: Bedside lung ultrasound in critical care practice. Crit Care 2007; 11(1): 205. PMID 17316468

Electrical impedance tomography

Electrical impedance tomography (EIT) is an imaging technique that can visualise regional distribution of lung ventilation by measuring the distribution of lung conductivity that results from the application of small electrical currents and measuring the resulting potential differences via electrodes placed circumferentially around the thorax. These differences in resistivity are collected and converted into a 2D image by a mathematical algorithm. Within these images, changes in electrical resistance represent change in lung aeration and can be represented numerically, or graphically displayed using a thermal scale. Changes in aeration can represent changes in end-expiratory lung volume or tidal changes in ventilation.

EIT allows the generation of images representing both the 'global' change in aeration of both lungs, and 'regional' changes in lung behaviour. In other words, EIT can display separately changes in ventilation of the left or right lung, and within the dorsal or ventral lung regions (see figure).
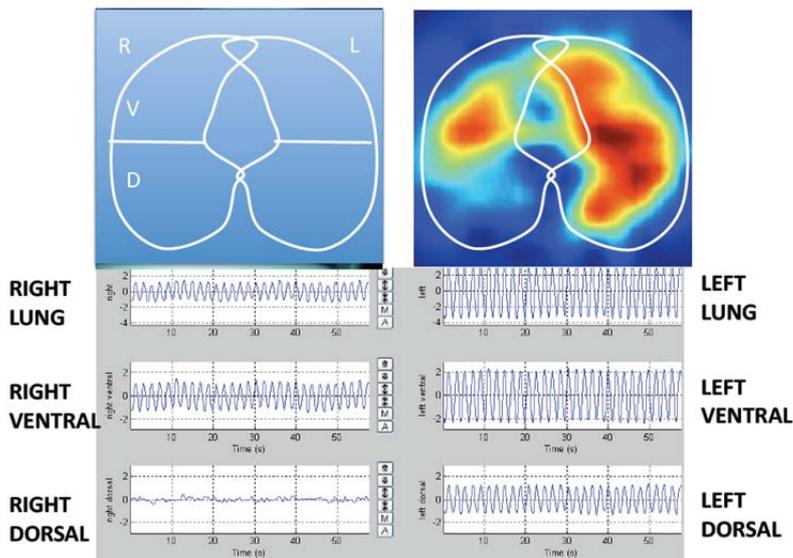
Electrical Impedance Tomography: Regional subdivision of cross-section of thorax (Top left) R= right; L= left; V= ventral region; D= Dorsal region. EIT image showing poor ventilation in the right dorsal region (Top right). In the lower panel changes in impedance during tidal ventilation are shown (Camporota – unpublished)

This ability to analyse different lung regions allows an understanding of regional inhomogeneities of aeration and response to treatment.

EIT has the following advantages:

- Non-invasive
- Radiation free
- Repeatable
- Fast responsiveness
- Bedside

Currently, EIT has a place in Critical Care to:

- Identify pleural effusions or pneumothorax. Both conditions are shown as a reduced or absent change in ventilation; however pneumothorax is characterised by a sudden increase in impedance and decreased ventilation, whereas pleural effusions, being more conductive than air, show a decrease in impedance together with a lack in change in impedance during tidal breathing.
- Allow dynamic evaluation of therapeutic interventions (e.g. physiotherapy, suctioning, post-bronchoscopy, drainage of pleural effusion or pneumothorax).
- Evaluate regional distribution of ventilation (classification of ARDS; distinction between focal or diffuse lung processes).
- Quantify local alveolar behaviour (recruitment, collapse and hyperinflation). To calculate the compliance of a compartment inside the respiratory system, it is necessary to know the local tidal volume and its corresponding driving pressure (calculated in pressure-controlled mode as plateau pressure minus PEEP, provided that both end-inspiratory and end-expiratory flows reach zero). Regional tidal volume can be estimated by EIT as regional change in impedance. Therefore regional compliance

may be calculated as [Change in Impedance/Pplateau-PEEP]. Using a standardised PEEP manoeuvre (stepwise increase and decrease in PEEP), it is possible to identify lung regions with different mechanical behaviour and change in regional compliance. During the increase in pressure: regions with increasing compliance (or larger changes in impedance) are recruited regions, whereas regions with decreasing compliance (or smaller changes in impedance) are region prone to overdistention. During the decremental phase: regions with increasing compliance (or larger changes in impedance) are regions recovering from hyperinflation, whereas regions with decreasing compliance (or smaller changes in impedance) are region that are collapsing.

- To facilitate the setting of PEEP (Conventional mechanical ventilation) and mPaw (High frequency oscillatory ventilation).

Future applications may include assessment of pulmonary blood flow and integration with ventilators to provide real-time monitoring of regional ventilation at a given ventilator setting.

Moerer O, Hahn G, Quintel M. Lung impedance measurements to monitor alveolar ventilation. Curr Opin Crit Care 2011; 17(3): 260–267. PMID 21478747

Other techniques

Radionuclide scanning – Radionuclide scanning and pulmonary angiography are used to detect pulmonary embolism. The two standard types of radionuclide scans in the lungs are perfusion and ventilation scans. These are used to detect and study pulmonary embolism. Spiral CT and CT pulmonary angiography (CTPA) have largely replaced radionuclide scanning for this indication.

Pulmonary angiography – This is performed by rapid injection of contrast media into the pulmonary arterial circulation with serial radiographic exposure. The procedure is invasive and not without risk. The main indication is congenital vascular abnormalities. Spiral CT has largely replaced angiography for initial diagnosis of pulmonary embolism.

> Whatever the benefits of proposed interventions, they must outweigh the risks of transporting the critically ill patient and those posed by the procedures themselves.

See the PACT module on Patient transportation and the following reference.

Shirley PJ, Bion JF. Intra-hospital transport of critically ill patients: minimising risk. Intensive Care Med 2004; 30(8): 1508–1510. Epub 2004 Jun 9. PMID 15197442

Bronchoscopy

Bronchoscopy is the process of direct visualisation of the tracheobronchial tree almost exclusively through a flexible bronchoscope. The use of rigid bronchoscopy is restricted to few selected situations.

Q. What reasons could lead ICU doctors to use a rigid bronchoscope?

A. The main benefit of a rigid bronchoscope is the presence of a large suction channel which may be used for retrieval of a foreign body, suctioning of a massive haemorrhage or for laser therapy.

Bronchoscopy is useful in some settings for visualising abnormalities of the airways and for obtaining a variety of samples from either the airways or the pulmonary parenchyma. Bronchoscopy may provide the opportunity for diagnosis as well as treatment.

Since the bronchoscope obstructs the tracheal tube, a number of consequences on the respiratory mechanics can be expected:
1) Increase in peak inspiratory pressure
2) Incomplete lung emptying with generation of a positive end-expiratory pressure effect generated by the expiratory resistance due to the bronchoscope in the tracheal tube.

These effects are more marked with smaller tracheal tubes and may lead to hyperinflation particularly when volume-controlled ventilation is used during the procedure. Furthermore, bronchoscopic suctioning generating a negative airway pressure may reduce PEEP and induce lung collapse, but may also alleviate lung hyperinflation. Suctioning can cause a rise in $PaCO_2$ and a decrease in PaO_2 partly by reducing the volume of gas available for exchange and partly by its effect on end-expiratory lung volume.

For more information regarding the use of bronchoscopy look at the following website. http://dpi.radiology.uiowa.edu/nlm/app/atlas/welcome2.html

2/ Monitoring respiratory function

Analysis of oxygenation

Blood gas analysis

Partial pressure of arterial oxygen (PaO_2) deriving from gas dissolved in the plasma – determines the percentage of haemoglobin saturated with oxygen and thus determines blood oxygen content.

> For a PaO_2 of 13.5 kPa (100 mmHg), SaO_2 is approximately 97% at normal pH

Efficient gas exchange relies on optimum matching between ventilation and perfusion within the lungs. The presence of increasingly greater shunt leads to increasing hypoxaemia.

Iso-shunt diagram: The figure shows the effect of increasing shunt fraction (decreasing VA/Q) on PaO_2 along a range of FiO_2.
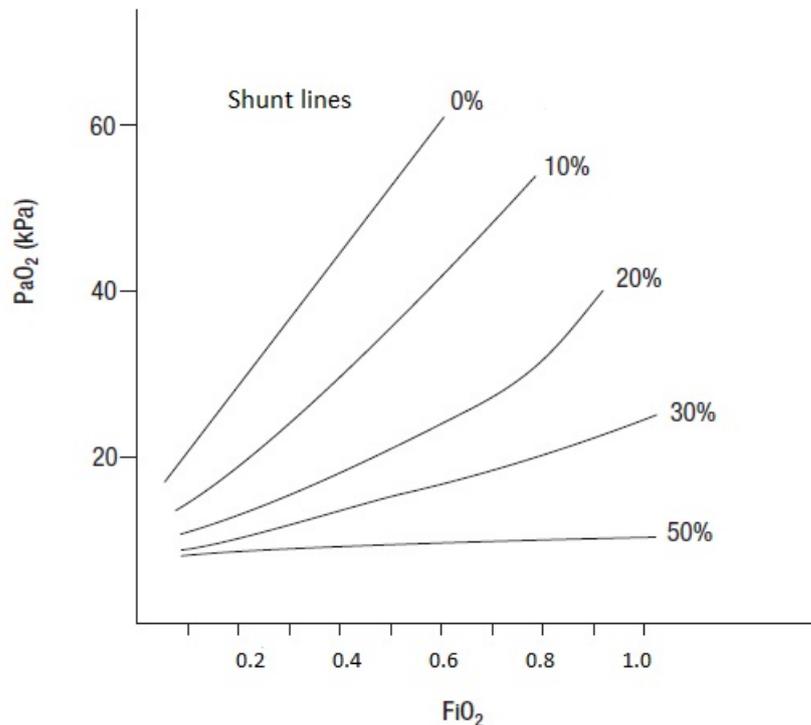
In normal conditions the amount of alveolar ventilation (V'A) in L per min nearly equals cardiac output value (L/min) producing a global ventilation/perfusion (VA/Q) ratio close to unity. However, each pulmonary unit may have its own regional VA/Q ratio ranging from regions with VA/Q= 0 (shunt compartment) to regions ventilated but not perfused (infinite VA/Q i.e. alveolar dead space).

The everyday clinical application of the 'iso-shunt diagram' is the insight it provides into why changes in FiO_2 frequently have little effect on oxygenation in major shunt scenarios e.g. lobar or lung collapse situations. Also the measurement of PaO_2 at an FiO_2 of 1.0 is used as a rapid assessment tool to

evaluate the level of shunt e.g. in potential donor lungs for transplantation.

Oxygen content and consumption

The vast majority of oxygen molecules are carried by haemoglobin, with only a small amount dissolved in plasma. Thus, arterial oxygen content (CaO_2) is largely determined by the SaO_2 and the haemoglobin (Hb) content. The factor 10 converts the final units to mL/minute; the small amount of dissolved oxygen is neglected.

> When Hb is 15 g/dL, CaO_2 is approximately 20 mL O_2 /100 mL blood
>
> 1 g/dL of Hb = 10 g/L = 0.62 mmol/L

Physically dissolved oxygen amounts only to 2–3 ml/L blood at air breathing.

Oxyhaemoglobin dissociation curve
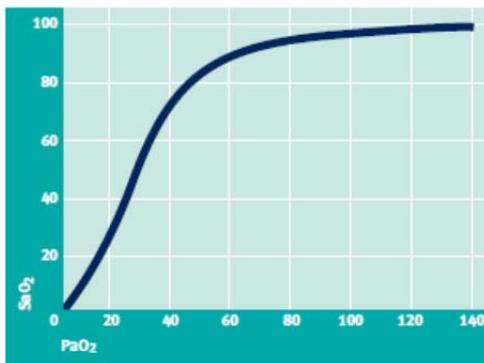
NOTE Anaemia does not affect SaO_2, but only oxygen content. Thus the oxyhaemoglobin dissociation curve PaO_2 vs SaO_2 is unaffected by haemoglobin content.

CaO_2 equals:
Amount of O_2 bound to haemoglobin + Amount of O_2 dissolved in plasma, or
(SaO_2 × Hb × 1.34) + (0.003 × PaO_2 in mmHg).

SaO_2 is the saturation percentage of haemoglobin with oxygen, Hb is the haemoglobin content in g/dL, 1.34 is the oxygen binding capacity of haemoglobin (mL O_2/g Hb), and 0.003 is the millilitres of oxygen that dissolve in 100 mL plasma per 0.135 kPa (1 mmHg) PaO_2. Normal arterial oxygen content is approximately 16 to 20 ml O_2/100 mL blood.

> Changes in haemoglobin concentration have a larger impact on arterial oxygen content than changes in PO_2 (oxygen partial pressure)

> **Q. What is the oxygen content for the following patient?**
> **35-year-old male**
> **Pulse 120/min, BP 154/82 mmHg, RR 24 /min**
> **Hb = 12 g/dL =120 g/L**
> **Hct = 36%**
> **ABGs (arterial blood gases): pH 7.39/PaO_2 13.5 kPa (100 mmHg)/$PaCO_2$ 4.5 kPa (34 mmHg)/96% SaO_2**

A. 15.4 mL/O_2 per 100 mL (154 mLs/Litre)

Hypoxaemia (a decrease in PaO_2) has a relatively minor impact on arterial oxygen content if the accompanying change in SaO_2 is small. PO_2 influences blood oxygenation only to the extent that it influences the saturation of haemoglobin with oxygen. Therefore, SaO_2 is a more reliable index of arterial haemoglobin oxygen content than PaO_2. The influence of anaemia relative to hypoxaemia on arterial oxygen content is relatively greater. See table below.

	Normal	Anaemia	Hypoxaemia
PaO_2	12 kPa (90 mmHg)	12 kPa (90 mmHg)	6 kPa (45 mmHg)
SaO_2	98%	98%	80%
Hb	15 g/dL	7.5 g/dL	15 g/dL
CaO_2	200 mL/L	101 mL/L	163 mL/L
% change in CaO_2		49.5%	18.6%

> **Q. You admit to your ICU a 22-year-old patient with multiple trauma. He is bleeding from his left leg, has a pelvic fracture and rib fractures. You perform an ABG and notice that his PaO_2 is low (9.3 kPa, 70 mmHg). Considering that his Hb is also low (7 g/dL), and is decreasing, which parameter would affect O_2 arterial content more?**

A. The low Hb will have the greater effect on O_2 content.

> Calculate CaO_2 content in the next ten patients requiring mechanical ventilation, and look at the relative effect of Hb and PO_2.

Knowing just the oxygen content of a patient's blood may not be sufficient to assess the adequacy of tissue oxygenation. Low or inadequate cardiac output may impair oxygen delivery (DO_2), which is the total cardiac output (CO) multiplied by arterial oxygen content (CaO_2). CO can be 'indexed' (CI, cardiac index) to body surface area.

Normal CI: 2.5–3.5 L/min/m²
$DO_2I = CI \times CaO_2$
By using a factor of 10, we can convert ml/dL (measurement of oxygen content) to mL/L
$DO_2I = 3 \times (1.34 \times Hb \times SaO_2) \times 10$ (presuming CI = 3 L/min/m²)
$DO_2I = 3 \times (1.34 \times 14 \times 0.98) \times 10$
$DO_2I = 550$ mL/min/m²
Normal range: 450–550 mL/min/m²

For more information on CO see the PACT modules on Hypotension and Haemodynamic monitoring.

DO_2 is the upper limit for the quantity of O_2 available to meet the total metabolic needs of the body. If oxygen utilisation exceeds the supply of O_2, the deprived cells must shift from aerobic to anaerobic metabolic pathways to supply their energy needs, leading to progressive lactic acidosis.

Oxygen consumption by the tissues (VO_2) can be measured non-invasively by *indirect calorimetry*, a technique that uses continuous analysis of inhaled and exhaled ventilatory gas flows, oxygen and carbon dioxide concentration allowing calculation of VO_2 and CO_2 production (VCO_2).

VO_2 can be calculated as the difference between the product of inspiratory volume and FiO_2, and the expiratory minute volume and the expired fraction of oxygen.

$VO_2 = (Vi \times FiO_2) - (Ve \times FeO_2)$

Bedside techniques now measure only the exhaled flow and O_2 and derive the inspiratory gases and flows using a mathematical relationship between Vi and Ve called *Haldane transformation*. In brief, this takes into account that nitrogen (N_2) is an inert gas and therefore the following identities can be established:

$Vi \times FiN_2 = Ve \times FeN_2$
$Vi = Ve \times (FeN_2/FiN_2)$; as $FiO_2+FiN_2=1$ and $FeCO_2+FeO_2+FeN_2=1$
$FeN_2=1-(FeCO_2+FeO_2)$

Therefore,

$ViO_2 = Ve \times [1-(FeCO_2+FeO_2)]/(1-FiO_2)$

The accuracy of this equation depends crucially on precise calculation of FiO_2 and Ve.

Accurate VO_2 values cannot be measured with a leaking airway, with $FiO_2 > 85\%$, when the respiration rate is >35/min or during some ventilatory modes (e.g., High frequency oscillatory ventilation, HFOV; Bilevel Positive Airway Pressure, BiPAP).

Alternatively, oxygen consumption can be indirectly calculated from the Fick equation:

$$VO_2 = CO \times (CaO_2 - C\bar{v}O_2)$$

The arterial–mixed venous content difference, $CaO_2 - C\bar{v}O_2$, represents the quantity of O_2 extracted by the peripheral tissues. Because CaO_2 and $C\bar{v}O_2$ share the same factor for haemoglobin binding ($1.34 \times Hb$), the equation can be rearranged isolating this term:

$$VO_2 = CO \times 13.4 \times Hb \times (SaO_2 - S\bar{v}O_2)$$

When DO_2 decreases and oxygen extraction reaches its maximum, VO_2 becomes dependent on DO_2. Many attempts to increase oxygen delivery in the intensive care setting have failed to increase oxygen consumption. Even in those few studies in which increased DO_2 augmented VO_2, no evidence exists of improved morbidity or mortality. Therefore, clinical interventions to achieve supranormal values of oxygen delivery and consumption in critically ill patients cannot be recommended.

Pulse oximetry

Pulse oximetry uses the principles of spectrophotometry to provide continuous non-invasive monitoring of the haemoglobin oxygen saturation of peripheral arterial blood (SpO_2).

The pulse oximeter probe consists of two light-emitting diodes with two-wavelengths on one side (660 nm, red; and 940 nm, infrared), with a light-detecting photodiode on the opposite side. The pulse oximeter can distinguish only two haemoglobin species on the basis of their different absorption of light: Oxyhaemoglobin absorbs more infrared light and less red light and the deoxyhaemoglobin has an inverse pattern of absorbance. Therefore, the haemoglobin saturation is calculated as the ratio of red/infrared absorbencies. However, not all the haemoglobin saturation is due to arterial blood and therefore, the oximeter also determines the pulsatile component of the light absorbance signal due to arterial blood pulsations (*photoplethysmography)*. The change in ratio of absorption between systole and diastole can then be used to calculate the arterial saturation of haemoglobin. This eliminates errors created by light reflection in nonpulsatile structures such as extravascular tissues and (nonpulsating) veins.

If there are significant quantities of other haemoglobin species, for example, methaemoglobin (MetHb) or carboxyhaemoglobin (COHb), the two-wavelength SaO_2 measurement can be enhanced by a four-wavelength haemoximeter ruling out the MetHb or COHb fraction. If there is a suspicion of carbon monoxide poisoning, a blood gas sample should be analysed by co-oximetry.

NOTE Pulse oximeters record light transmission through pulsating arteries only.

Q. Why is pulse oximetry used?

A:
- Superior detection of hypoxaemic episodes
- It is non-invasive
- Low morbidity and higher patient satisfaction
- Less expensive than blood gas measurement

Johnson PA, Bihari DJ, Raper RF, Haughton MA, Fisher MM, Herkes RG. A comparison between direct and calculated oxygen saturation in intensive care. Anaesth Intensive Care 1993; 21(1): 72–75. PMID 8447611

Wahr JA, Tremper KK. Noninvasive oxygen monitoring techniques. Crit Care Clin 1995; 11(1): 199–217. PMID 7736267

Stirling S, Langford R. Pulse Oximetry. In: Waldmann C, Soni N, Rhodes A, editors. Oxford Desk Reference: Critical Care. Oxford: Oxford University Press; 2008. pp. 94–95. ISBN 13: 978–0–19–922958–1

NOTE Hyperbilirubinaemia per se has been shown not to affect the accuracy of SpO_2 readings.

Changes in pH, temperature, and 2,3-diphosphoglycerate concentration alter the PO_2–SaO_2 relationship and may result in misleading calculations of oxyhaemoglobin saturation.

Limitations of pulse oximetry:
- Motion artefact (shivering, intra-aortic balloon pump assistance, and during transport)
- Abnormal haemoglobins (primarily COHb –falsely high SpO_2 –, and MetHb – falsely low SpO_2 when SpO_2 >85% and falsely high when SpO_2 <85%)
- Exposure of measuring probe to ambient light during measurement
- Low perfusion states
- Hypothermia
- Skin pigmentation
- Nail polish or nail coverings, when a finger probe is used
- Inability to detect saturations below 83% with the same degree of accuracy and precision seen at higher saturations but the level of discrepancy is not of clinical importance

Pulse oximetry offers the advantage of providing data on haemoglobin saturation rather than PO_2. SaO_2 reflects the 98% of arterial oxygen content that is normally carried by haemoglobin, while the PO_2 directly measures only the small amount of oxygen that is dissolved in plasma.

The normal relation between SaO_2 and PaO_2 is the oxyhaemoglobin dissociation curve, above. At PaO_2 values greater than 12 kPa (90 mmHg), SaO_2 is nearly 100% and becomes virtually independent of PaO_2. It is important to remember this during SaO_2 monitoring, where elevated inspired oxygen fraction (FiO_2) values will give no indication of early trends in PaO_2 until PaO_2 values less than 12 kPa (90 mmHg) are reached.

ANECDOTE An inadvertently hypoventilated patient was administered 100% oxygen during anaesthesia for hip arthroplasty and monitored with pulse oximetry alone. He developed a $PaCO_2$ of 35.3 kPa (265 mmHg) and an arterial pH of 6.65 despite maintenance of oxygen saturations of 94 to 96%.

Ayas N, Bergstrom LR, Schwab TR, Narr BJ. Unrecognized severe postoperative hypercapnia: a case of apneic oxygenation. Mayo Clin Proc 1998; 73(1): 51–54. PMID 9443679

Q. A 58-year-old male undergoes abdominal surgery. Six hours after extubation he becomes moderately short of breath. SpO₂ is 89%. After applying 40% oxygen, the patient's SpO₂ is 94%. What is the cause of this patient's hypoxaemia? What is the treatment?

A. This patient has a ventilation–perfusion abnormality, presumably caused by atelectasis in the dependent regions of his lungs, as a result of anaesthesia and surgery. The treatment is supplemental oxygen, chest physiotherapy, mobilisation and continuous positive airway pressure (CPAP).

CO₂ analysis

Capnography

Capnography, the measurement and the graphical display of CO_2 in expired gases, has evolved into a standard of care in anaesthesia and is recommended in ventilated critical care patients to improve patient safety (both in ICU and during patient transport), and provide additional physiological information on intrapulmonary gas mixing and ventilation–perfusion relationship that can assist clinical decision-making and treatment. It is important to be familiar with capnography and the interpretation of capnographic curves (capnogram).

There are two main types of capnography: time and volumetric.

Time capnography

CO_2 concentration in *inspiratory and expiratory* airway gas is plotted against time. This is commonly used in clinical practice

Time capnogram

Basically, the lung is made up of approximately 300–400 million alveoli, each having their own time constant for CO_2 elimination, and a gravitational variation in the ventilation/perfusion ratio. Therefore, in the capnogram of a healthy person, variations in angle width and in inclination of its segments may be observed. However, we can identify some constant patterns in CO_2 wave forms: an inspiratory phase and an expiratory period made up of three phases.

Normal time capnogram
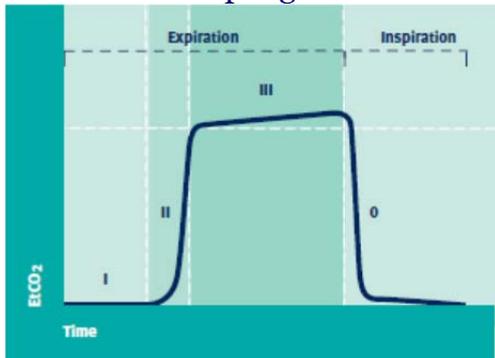
The first phase **(phase I)** represents the beginning of expiration. The analysed gas comes from the CO_2-free gas from the anatomical dead space i.e. pharynx, larynx, trachea and bronchi and, in mechanically ventilated patients, the endotracheal tube or the tracheostomy cannula. Therefore, concentration of CO_2 from these regions is close to zero.

From this point on, a second phase starts (upstroke of the capnogram, **phase II**), representing a rapidly changing mixture of alveolar and dead space gas (mixed air zone). It is a segment with an S-shaped trend with a sharp upswing due to mixing of the air coming from the dead space with the air coming from alveoli with rapid depletion. The third phase (**phase III**) otherwise called 'alveolar plateau' represents exhalation of pure alveolar gas, and it rises slightly (slope 0.27– 0.4 kPa/s; 2–3 mmHg/s), due to the mild inhomogeneity of the ventilation/perfusion ratio and of alveolar CO_2 concentration. The highest value of CO_2 during the alveolar plateau is called **end-tidal CO_2 (PetCO_2)**.

Once phase III is completed, the capnographic wave sharply descends towards zero due to the irrelevant amount of carbon dioxide usually present in inspiratory gas; the portion of the capnographic wave from beginning of inspiration to beginning of expiration is termed as **phase 0**; it consists of the descending limb of the capnogram, and the initial portion of the baseline.

In addition to the segments, two angles further characterise the capnogram: α (between phase II and phase III), which becomes wider with increasing Va/Q mismatch; and β (between phase III and phase 0), which widens as rebreathing and anatomical/apparatus dead space increases.

End-tidal CO_2 (EtCO_2) analysis can rely on a predictable alveolar–arterial gradient of about 0.67 kPa (5 mmHg) in most physiological conditions due to the high permeability of the alveolo-capillary barrier to CO_2. On the contrary, in

critically ill patients, the PaCO₂−PetCO₂ gradient can vary markedly, depending not only on lung pathology, but also on the mode of mechanical ventilation, inotropic support etc., making $EtCO_2$ inaccurate in predicting $PaCO_2$. The ΔCO_2 gap is an indicator of alveolar dead space.

Alveolar CO_2 essentially depends on two factors:

- Perfusion of the pulmonary capillary bed (directly correlated to end-tidal CO_2)
- Thickness of the alveolo-capillary membrane (inversely correlated to end-tidal CO_2). This rarely influences the CO_2 diffusion since CO_2 diffuses through the alveolar 20 times faster than O_2.

By monitoring $EtCO_2$, information can be obtained about perfusion, ventilation, and metabolism. The patient, in order to expel CO_2 from the lungs, must have an adequate cardiac output (or perfusion of pulmonary circulation). At the same time ventilation allows the constant washout of CO_2 from the alveoli. An increased body metabolism for any reason causes an augmented O_2 consumption and CO_2 production which can be detected by capnography.

> Remember the early signs of malignant hyperthermia:
> Masseter rigidity
> Tachycardia
> Hypercarbia
> Hyperthermia may be a late sign!

Verify that end-tidal CO_2 depends on pulmonary capillary bed perfusion, which in turn depends on cardiac output: select mechanically ventilated patients in your unit, excluding those with pulmonary impairment, and observe variations of stroke volume or, indirectly, arterial pressure. Relate such changes to end-tidal CO_2.

Clinical correlations of capnographic curves: shape analysis

Analysis of the shape of capnographic curves can give valuable diagnostic insight in the diagnostic process.

Obstruction to expiratory flow (bronchospasm)

Bronchial obstruction causes regional alveolar ventilation inhomogeneity, thus altering the normal ventilation/perfusion ratio. CO_2 is asynchronously exhaled from alveoli, resulting in a flattened phase II of the capnographic curve. As a consequence, the slope of phase III increases and there is widening of the α angle. The gradient of the phase II on a capnogram has been proposed as a non-effort-dependent test for the severity of bronchospasm.

Patient with bronchospasm
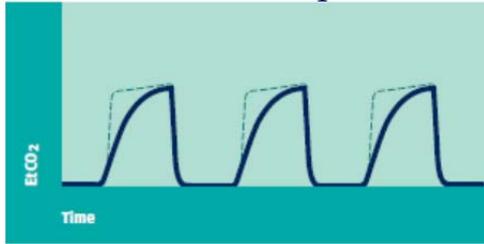
The main clinical causes of obstruction of the expiratory flow are asthma, bronchospasm, bronchiolitis in infants, and chronic obstructive pulmonary disease (COPD). Even emphysema produces a slanted upstroke in phase II.

⚠️ Obstruction of exhaled gas may be caused by external factors such as a kinked tracheal tube. This will usually be manifested by increased airway pressures with volume-controlled ventilation or as reduced tidal volumes with pressure-controlled ventilation, sustained flow at end-expiration and not by an elevation of phase III on the CO_2 curve.

Capnography is a clinical aid both in diagnosis and in detection of the efficacy of therapy. Bronchospasm resolution after salbutamol administration can be verified by checking the capnographic curve returning to a normal shape.

Wagstaff A. End-tidal CO_2 Monitoring. In: Waldmann C, Soni N, Rhodes A, editors. Oxford Desk Reference: Critical Care. Oxford: Oxford University Press; 2008. pp. 92–93. ISBN 13: 978–0-19–922958–1

Cardiac arrest

Cardiac arrest results in a dramatic reduction of pulmonary blood flow. CO_2 cannot be delivered to alveoli, so even if the patient is intubated and mechanically ventilated, CO_2 cannot be exhaled.

Patient in cardiac arrest

In a mechanically ventilated patient with sudden serious cardiac impairment, leading to cardiac arrest, end-tidal CO_2 warns of the serious reduction of pulmonary perfusion. The capnographic curve maintains a normal shape but becomes lower with every breath.

> **NOTE** Always check the tube position at cardiac arrest: A flat CO_2 curve in association with cardiac arrest may be due to a displacement of the endotracheal tube causing an anoxic cardiac arrest.

Levine RL, Wayne MA, Miller CC. End-tidal carbon dioxide and outcome of out-of-hospital cardiac arrest. N Engl J Med 1997; 337(5): 301–306. PMID 9233867

> THINK Pulmonary embolism also results in a reduction of pulmonary bed perfusion. How do you think the capnogram of a patient suffering from sudden pulmonary embolism will differ from the capnogram during cardiac arrest?

See the paragraph on dead space.

Spontaneous respiratory effort

A patient's respiratory efforts can be detected by an incisura in phase III of the capnogram (see diagram of a controlled breath). In the more usual patient-triggered breathing used in critical care, such an incisura might be expected to trigger a new inspiration. This indicates a patient–ventilator dyssynchrony; the trigger should be reset
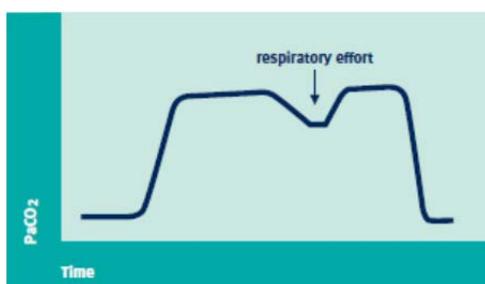
Volumetric capnography

The *expired* CO_2 concentration is displayed against expiratory flow rate to establish the relationship between CO_2 and flow. This allows calculation of total CO_2 production and respiratory dead space. It is not widely used in clinical practice.

Volumetric capnography, describes the partial pressure of exhaled CO_2 over the volume of one single breath. The integration of expiratory flow or volume signals with the CO_2 signal has the potential if introduced more widely to clinical practice to serve as an important monitoring and diagnostic tool, the graphical representation being shown in figures below. This plot of CO_2 vs volume has been divided into three phases labelled I through III, similar to the time capnography.
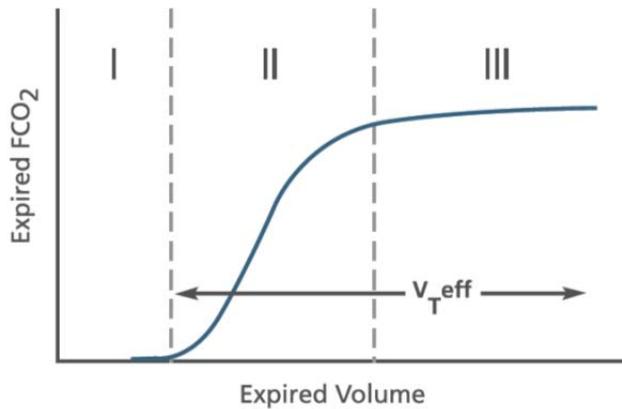
Using these three components of the volumetric capnogram, the volumes of each phase, the slopes of phase II and III and CO_2 as well as dead space to tidal volume ratio (using the Fowler's method), ratios of anatomic and physiologic dead space can be determined. Physiologic dead space, the sum of the anatomical dead space and alveolar dead space, can be calculated approximately knowing an estimate of the alveolar PCO_2 or arterial PCO_2. A respiratory unit that is ventilated but not eliminating CO_2 (i.e. deprived of its blood flow) is included in the alveolar dead space volume. Additionally, other physiological parameters can be derived from the volumetric capnogram including surrogates of alveolar CO_2, of ventilator efficiency, measurements of the non-synchronous emptying of alveoli with unequal ventilation/perfusion ratios.

Dead space

> **Q. Why can a patient's PaCO₂ be high when he has a minute ventilation of 30 litres per minute?**

A. The dead space may be relatively high thereby causing a relatively low alveolar ventilation.

Link to the PACT module on Mechanical ventilation.

> Clearance of carbon dioxide is determined by the alveolar ventilation and the physiologic dead space

Lucangelo U, Blanch L. Dead space. Intensive Care Med 2004; 30(4): 576–579. Epub 2004 Feb 28. PMID 14991089

The concept of dead space accounts for those lung areas that are ventilated but not perfused and it is constituted by the sum of anatomic dead space (Vd_{anat} – upper and lower airways) and alveolar dead space (Vd_{alv} – alveoli well ventilated but receiving minimal blood flow). Parts of the ventilator equipment (tracheal tubes, humidification devices and connectors) are considered instrumental dead space (Vd_{ins}) and are part of the anatomic dead space. The physiologic dead space (Vd_{phys}) is comprised of the sum of conducting airways (instrumental and anatomic dead space) and alveolar dead space (represented by the areas Z + Y in the Expired Breath diagram below) and it is usually reported in mechanical

ventilation as the portion (or fraction) of tidal volume (Vd/Vt_{phys}) or minute ventilation that does not participate in gas exchange.

Measurement of dead space using CO_2 as a tracer gas

Dead space ventilation (Vd) is the portion of Vt that does not encounter perfused alveoli. The analysis of the expired CO_2 as a function of exhaled volume (volumetric capnography) together with the measurement of $PaCO_2$ has the capacity to provide a precise quantification of the physiologic dead space fraction (Vd/Vt_{phys}). This is calculated from the Enghoff modification of the Bohr equation:

> Volumetric capnography simultaneously measures expired CO_2 and tidal volume

$$Vd/Vt_{phys} = (PaCO_2 - PECO_2) / PaCO_2$$

In this equation, $PaCO_2$ is arterial PCO_2 and $PECO_2$ is the partial pressure of CO_2 in mixed expired gas. PECO2 is equal to the mean expired CO_2 fraction multiplied by the difference between the atmospheric pressure and the water-vapour pressure.

The measurement of airway dead space (Vd_{anat})) is based on the geometric method of equivalent areas (p = q, see graph below), obtained by crossing the back extrapolation of phase III of the capnography curve, with a vertical line traced so as to have equal p and q areas. Airway dead space is then measured from the beginning of expiration (time zero of volumetric capnography diagram below) to the point where the vertical line crosses the volume axis – see diagram below.

Expired volumetric capnography breath diagram
Graph of expired CO2 concentration vs expired volume.
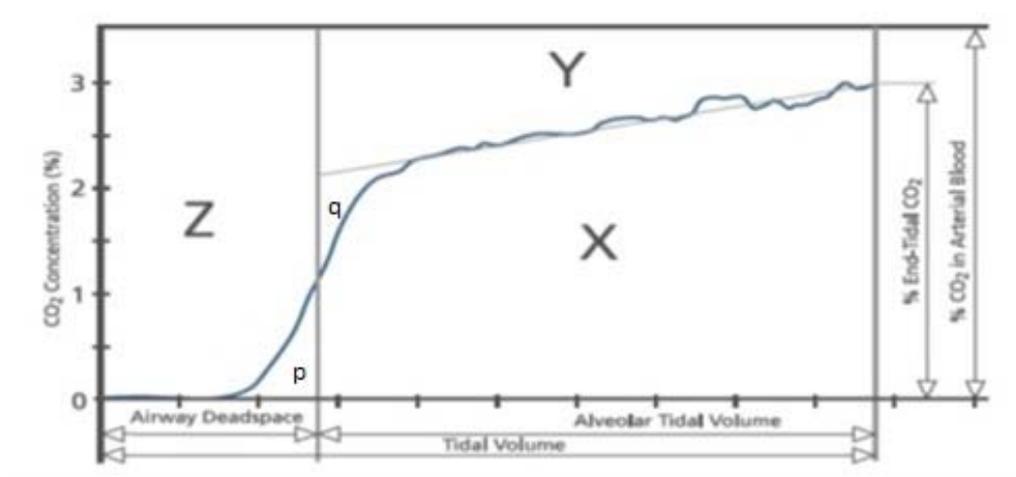
X represents the expired volume of CO_2 which is reflective of the volume in which there is ventilation of perfused alveoli. Z reflects airway dead space (Vd_{anat}) and Y reflects alveolar dead space (Vd_{alv}). The areas of Z + Y reflect the physiological dead space (Vd_{phys}).

Furthermore, the net volume of carbon dioxide elimination (VCO_2) can be viewed as the area between the expiratory and inspiratory curves (see figure below).
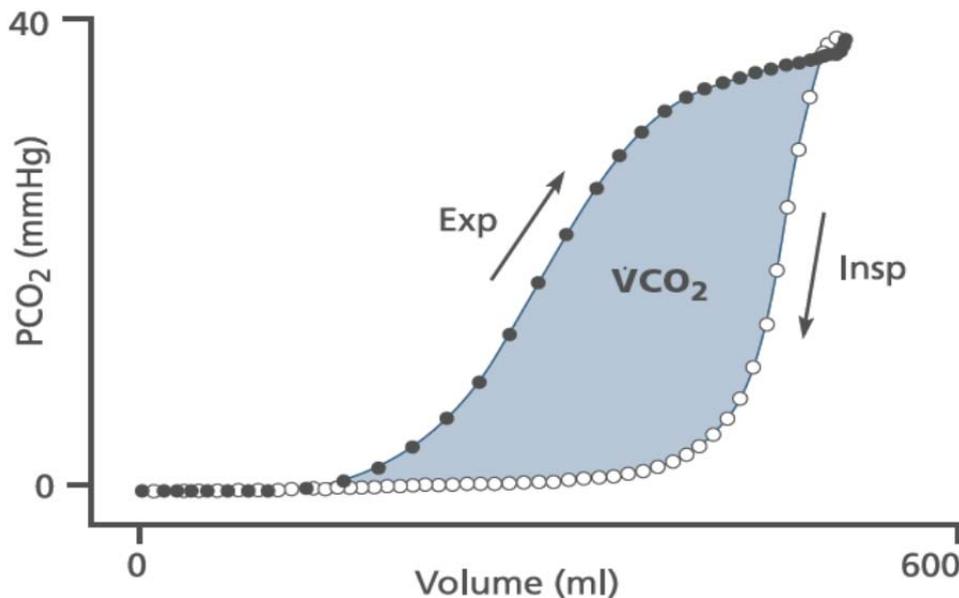
Fletcher R, Jonson B, Cumming G, Brew J. The concept of deadspace with special reference to the single breath test for carbon dioxide. Br J Anaesth 1981; 53(1): 77–88. PMID 6779846

Lucangelo U, Blanch L. Dead space. Intensive Care Med 2004; 30(4): 576–579. Epub 2004 Feb 28. PMID 14991089

Sinha P, Flower O, Soni N. Deadspace ventilation: a waste of breath! Intensive Care Med 2011; 37(5): 735–746. PMID 21394623

Evaluate Vd/Vt at different minute ventilation settings in the same patient.

Interpreting dead space

In patients with a normal cardiorespiratory system, physiologic dead space (particularly its alveolar component) is the primary determinant of the differences between arterial to end-tidal PCO_2 (ΔPCO_2) – see Expired Breath diagram above.

Alveolar physiologic dead space (Vd_{alv}) is increased by shock states, systemic and pulmonary hypotension and obstruction of pulmonary vessels (massive pulmonary embolus and microthrombosis) in the absence of a corresponding decrease in ventilation. Airway dead space (Vd_{anat}) is increased by lung over-distension and inappropriate instrumental dead space. Tracheal tubes, heat and moisture exchangers and other common connectors may increase instrumental

dead space (Vd_{ins}) and thus reduce alveolar ventilation and favour hypercapnia during low tidal volume ventilation.

The potential of volumetric capnography as a screening method for pulmonary embolism and as a monitor of thrombolysis therapy has been shown in recent studies: CO_2 versus time monitoring can also detect pulmonary embolism.

Verschuren F, Liistro G, Coffeng R, Thys F, Roeseler J, Zech F, et al. Volumetric capnography as a screening test for pulmonary embolism in the emergency department. Chest 2004; 125(3): 841–850. PMID 15006940

Verschuren F, Heinonen E, Clause D, Roeseler J, Thys F, Meert P, et al. Volumetric capnography as a bedside monitoring of thrombolysis in major pulmonary embolism. Intensive Care Med 2004; 30(11): 2129–2132. Epub 2004 Sep 18. PMID 15378240

> **NOTE** Dead space calculations are affected by the apparatus or instrumental dead space.

Application of PEEP is used to increase lung volume and to improve oxygenation in patients with acute lung injury. Recruitment in ARDS may be associated with a decreased ΔPCO_2 gradient.

> ⚠ Beware of tachypnoea with small tidal volumes: respiratory effort is being wasted, leading to severe muscular fatigue and acidosis.

Mixed/central venous gas analysis

$S\bar{v}O_2/ScvO_2$

Mixed venous oxygenation represents the amount of oxygen in systemic circulation that is left after delivery of oxygen to tissues. It is probably the best indicator of the balance between oxygen delivery and consumption and therefore is an indicator of the degree of oxygen extraction.

$S\bar{v}O_2$ calculation

The Fick equation for VO_2 helps to interpret the mixed venous oxygen saturation ($S\bar{v}O_2$) and partial pressure (PvO_2):

$VO_2 = CO \times (CaO_2 - C\bar{v}O_2)$
$C\bar{v}O2 = CaO_2 - VO_2 /CO$
$C\bar{v}O2 /CaO_2 = 1 - VO_2/(CaO_2 \times CO) = 1 - VO_2 /DO_2$

If the contribution of the dissolved oxygen is ignored, the equations for mixed venous oxygenation can be written as:

$$S\bar{v}O_2 / SaO_2 = 1 - VO_2 / DO_2$$
$$S\bar{v}O_2 = SaO_2 - (VO_2 / 1.34 \times Hb \times CO)$$

Accordingly, an increase in VO_2 and a decrease in haemoglobin, cardiac output and arterial oxygenation may result in a decrease of $S\bar{v}O_2$ if the other factors do not compensate for the reduction.

Mixed venous oxygenation is probably the best single indicator of the adequacy of global oxygen transport since it represents the amount of oxygen in systemic circulation that is left after passage through the tissues, and therefore is an indicator of the balance between oxygen delivery and consumption. $S\bar{v}O_2$ values between 70–80% represent an optimal balance between global oxygen supply and demand. A value < 50% corresponds to the theoretical critical PvO_2 of 3.5 kPa (the value when the capillary oxygen tension is too low to support aerobic tissue metabolism). $S\bar{v}O_2$ > 80% are generally seen in hyperdynamic shock and reflects the high cardiac index and low oxygen extraction (septic shock).

> ⚠ Since $S\bar{v}O_2/ScvO_2$ is the flow-weighted average oxygen content of the venous effluents from various tissues, hypoxia may still be present in a tissue receiving only a small proportion of cardiac output, despite relatively normal mixed venous oxygen saturation. Despite this limitation, mixed venous oxygen content and especially changes in this value will still reflect the adequacy of oxygen delivery in common clinical conditions.

There is no safe level of $S\bar{v}O_2$. A low $S\bar{v}O_2$ should always prompt a suspicion of inadequate tissue perfusion, as lower values of $S\bar{v}O_2$ may reflect an increase in VO_2 and a decrease in CO, Hb, and SaO_2. It is important to remember that a high or normal $S\bar{v}O_2$ does not always signify an adequate oxygenation in all organs, but it can reflect an impaired oxygen extraction ratio (O_2ER) or abnormal vasoregulation. In these circumstances, lactate levels will be elevated to reflect the O_2 debt of the anaerobic metabolism. As a practical guideline, an increased risk for tissue hypoxia or inadequate perfusion should be considered in the acutely ill patient when $S\bar{v}O_2$ <60–65 %. As for the other oxygen transport related variables, the changes in $S\bar{v}O_2$ in response to therapy are more important than single values. The changes in $S\bar{v}O_2$ can be monitored using fibre optic pulmonary artery catheters.

$S\bar{v}O_2/ScvO_2$ monitoring systems and PA catheters are presented in the PACT module on Haemodynamic monitoring.

Extravascular lung water

Critically ill patients are often at increased risk of pulmonary oedema because of systemic inflammatory states causing capillary leakage. Fluid management in these patients is a balancing act between avoiding pulmonary oedema, while maintaining a sufficient intravascular volume for adequate cardiac preload.

Measurement of extravascular lung water (EVLW) as a clinical tool for the assessment of pulmonary function has been found to:

- Be more appropriate than oxygenation parameters or radiographic techniques.
- Correlate with survival in critically ill patients (non-survivors have significantly higher EVLW values than survivors).

THINK why patients with severe sepsis can have capillary leakage?

For more information read the following references.

Kuzkov VV, Kirov MY, Sovershaev MA, Kuklin VN, Suborov EV, Waerhaug K, et al. Extravascular lung water determined with single transpulmonary thermodilution correlates with the severity of sepsis-induced acute lung injury. Crit Care Med 2006; 34(6): 1647–1653. PMID 16625129

Brown LM, Liu KD, Matthay MA. Measurement of extravascular lung water using the single indicator method in patients: research and potential clinical value. Am J Physiol Lung Cell Mol Physiol 2009; 297(4): L547–558. PMID 19617309

Sakka SG, Klein M, Reinhart K, Meier-Hellmann A. Prognostic value of extravascular lung water in critically ill patients. Chest 2002; 122(6): 2080–2086. PMID 12475851

Clinical assessment of the extent of pulmonary capillary leakage and pulmonary oedema is difficult. Among the methods intended to improve monitoring, the transpulmonary indicator dilution techniques have gained increased clinical recognition. These techniques provide detailed information about haemodynamic and volumetric variables including quantification of EVLW.

The most recent method is based on the intravenous injection of a single thermal indicator. EVLW, determined with the single transpulmonary thermodilution technique, correlates with that determined with the thermal dye dilution. The value of the single thermodilution technique for monitoring EVLW and for assessment of the severity of sepsis-induced ARDS is reported by Kuzkov and co-workers (reference above).

A correlation exists between EVLW measured by the single indicator transpulmonary thermodilution technique and post-mortem lung weight in humans. The normal EVLW value is approximately 7.4 ± 3.3 mL/kg.

Tagami T, Kushimoto S, Yamamoto Y, Atsumi T, Tosa R, Matsuda K, et al. Validation of extravascular lung water measurement by single transpulmonary thermodilution: human autopsy study. Crit Care 2010; 14(5): R162. PMID 20819213

A ratio between extravascular lung water and pulmonary blood volume using PiCCO gives an indirect measure of the permeability of the pulmonary vascular

bed (pulmonary vascular permeability index – PVPI), thus allowing differentiation between cardiogenic (increased hydrostatic pressure) and non-cardiogenic (increased permeability) pulmonary oedema.

Some investigators have noticed that therapy guided by EVLW measurements shortens the duration of mechanical ventilation and the length of ICU and hospital stay. However, since there are no clear-cut treatment algorithms including EVLW as an end point, it carries at least the indirect or theoretical risk of advocating unnecessary or even adverse treatment strategies as it would be theoretically possible to perform fluid restriction, based on EVLW data, in a condition of systemic hypovolaemia.

> ⚠️ Although not normally available clinically, digital chest roentgenogram has been used to differentiate a patient's volume status. Conventional interpretations of portable supine chest X-ray can differentiate PAOP >18 mmHg from PAOP <18 mmHg, but the value of individual signs varies among radiologists.

Ely EW, Smith AC, Chiles C, Aquino SL, Harle TS, Evans GW, et al. Radiologic determination of intravascular volume status using portable, digital chest radiography: a prospective investigation in 100 patients. Crit Care Med 2001; 29(8): 1502–1512. PMID 11505116

3/ Monitoring ventilator waveforms

Airway pressure

Volume-controlled ventilation (VCV)

During VCV, the pressure curve over time has a characteristic feature. With the beginning of inspiration, an almost vertical pressure rise occurs (see figure below), which is necessary to overcome the resistance provided by the airways and by the tracheal tube. The curve shape then changes, turning to a linear growth and following a given slope to its maximum value (Pmax), which occurs at end-inspiration.

Pmax represents the sum of the pressures produced by the ventilator to overcome the resistive forces (airways and endotracheal tube) and the elastic recoil of the respiratory system. Variations in Pmax in isovolumetric conditions depend on the pressure dissipated to overcome the resistance offered by the airways and by the tracheal tube – see peak and plateau pressures below.

Pressure-controlled ventilation (PCV)

Q. How relevant to clinical practice is analysis of the pressure trace during pressure-controlled ventilation?

A. The analysis of the pressure curve has limited clinical utility during PCV.

In the case of PCV, the dependent variable is the flow wave, which changes as the features of the respiratory system change: the machine will constantly adjust flow so that the inspiratory pressure is maintained during the entirety of the set inspiration time (see figure 'Flow and pressure waves during PCV' below).

Peak and plateau airway pressures

During VCV, an airway occlusion manoeuvre at end-inspiration (which prevents normal exhalation) results in a rapid drop of the pressure until a plateau or pause pressure is reached (plateau pressure – see diagram). During this time there is equilibration between mouth pressure and alveolar pressure, so that the plateau / pause pressure represents the pressure applied to the small airways and alveoli.

The plateau pressure (Ppause) represents the pressure produced by the ventilator to overcome the elastic recoil forces of the respiratory system (lungs and chest wall). The peak pressure (Pmax) is the pressure measured by the ventilator in the major airways, and it strongly reflects airway resistance.

VCV: Flow and pressure waves
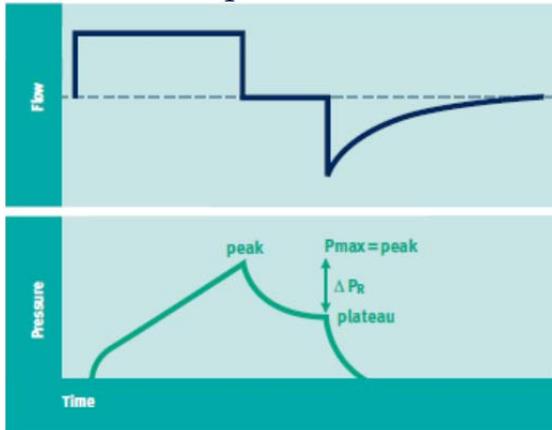
Q. Which clinical condition is characterised by high peak pressure (Pmax) and normal plateau pressure?

A. Bronchospasm

Lucangelo U, Bernabé F, Blanch L. Respiratory mechanics derived from signals in the ventilator circuit. Respir Care 2005; 50(1): 55–65; discussion 65–67. PMID 15636645

Airway flow

The normal flow pattern during spontaneous breathing of gas moving in and out of the lungs is sinusoidal.

In VCV, a variety of different wave patterns can be used. In clinical practice, constant and decelerating flow patterns are used; the latter is the same flow pattern as during pressure-controlled ventilation, but performed with a volume guarantee.

In PCV, the dependent variable is flow, which changes as the features of the respiratory system change: the ventilator will constantly adjust flow so that the inspiratory pressure is maintained during the entire set inspiratory time.

Q. What is the flow pattern in pressure control mode?

A. In pressure control mode (see PACT module on Mechanical ventilation), the flow pattern is always decelerating.

In the inspiratory flow waveform two phases can be observed: the initial peak which corresponds to the system pressurisation, and a second segment that

descends with a variable slope. The latter is a function of both the respiratory system compliance and the resistance.

During PCV the flow wave has the characteristic descending shape that approaches zero with prolongation of the inspiratory time. Along with the reducing flow, alveolar pressure approaches the working pressure set on the ventilator because the resistive pressure loss in patent airways is reduced. See the graph below.

In PCV, the pressure is constant and tidal volume varies with the patient's condition in contrast to VCV, where the tidal volume is constant and the pressure varies with the patient's condition.

PCV: Flow and pressure waves
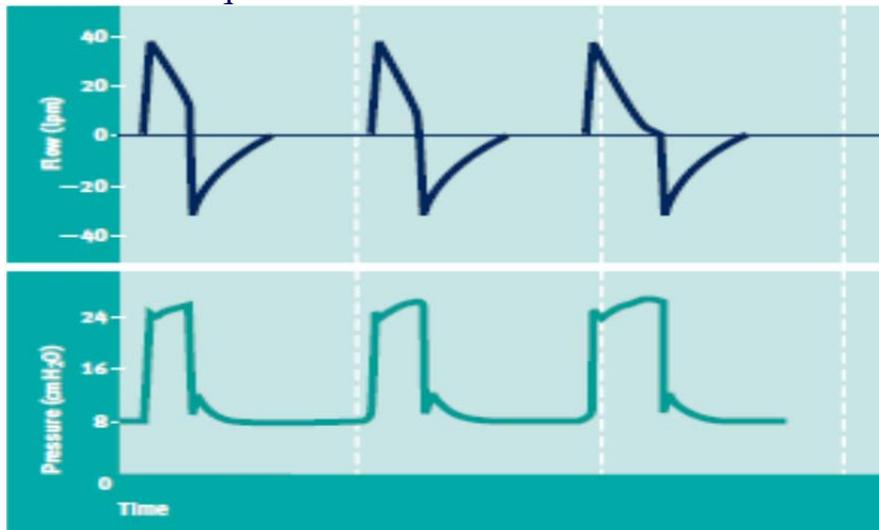
Access the electronic version of the PACT module on Mechanical ventilation and try out the simulators.

Loops

Pressure-volume loops

Monitoring pressure-volume curves during tidal breathing can facilitate optimisation of ventilator settings in patients with acute lung injury or in patients with obstructive lung disease. Pressure-volume loops may be helpful in detecting:

- Changes in compliance
- Inadequate trigger sensitivity
- Inadequate inspiratory flow setting
- Elastic and resistive work of breathing
- Patient–ventilator dyssynchrony

Flow-volume loops

The inspiratory limb of the curve reflects the setting of inspiratory flow. The expiratory limb shows a smooth decrease in expiratory flow. Analysis of the flow-volume loop may be helpful for identifying:

- Reduced expiratory flow
- Flow limitation during expiration – concave pattern
- Fixed airway obstruction
- Presence of excessive secretions – sawtooth pattern
- Air-leak
- Patient–ventilator dyssynchrony
- Airtrapping
- Presence of intrinsic PEEP

Dhand R. Ventilator graphics and respiratory mechanics in the patient with obstructive lung disease. Respir Care 2005; 50(2): 246–261; discussion 259–261.PMID 15691394

Oesophageal pressure

Because the body of the oesophagus generally behaves as a passive structure (except during swallowing), the pressure in the lower third of the oesophagus (oesophageal pressure, P_{es}) approximates the mean pleural pressure (P_{pl}) when the patient is upright. Although the absolute values of oesophageal pressures might not correctly represent the absolute pressures in the pleural space in the supine position, there are strong indications that the changes in oesophageal pressure with ventilation or pressure-volume manoeuvres reflect the changes in pleural pressures.

Pelosi P, Goldner M, McKibben A, Adams A, Eccher G, Caironi P, et al. Recruitment and derecruitment during acute respiratory failure: an experimental study. Am J Respir Crit Care Med 2001; 164(1): 122–130. PMID 11435250

Talmor DS, Fessler HE. Are esophageal pressure measurements important in clinical decision-making in mechanically ventilated patients? Respir Care 2010; 55(2): 162–172. PMID 20105342

NOTE In a spontaneously breathing patient, P_{es} is negative during inspiration.

The use of the oesophageal balloon catheter allows us to divide the compliance of the respiratory system (C_{rs}) into its components of lung compliance (C_L) and

chest wall compliance (C_{cw}). The calculations are:

$C_L = Vt/(P_{AO} − P_{es})$ end-inhalation − $(P_{AO} − P_{es})$ end-exhalation
$C_{cw} = Vt/(P_{es} − P_{atm})$ end-inhalation − $(P_{es} − P_{atm})$ end-exhalation

P_{AO} is the pressure at the airway opening.

For example, if we rely on the compliance of the respiratory system (C_{rs}) (see below), measured at the bedside, to follow changes in the severity of a patient's acute respiratory distress syndrome, we may see changes in the value that do not reflect changes in C_L but may reflect changes in C_{cw}.

> **NOTE** Compliance is defined as the unit change in volume in response to a unit change in pressure (see below).

Low chest wall compliance can be acquired in critical illness due to circumferential chest dressings, extensive oedema, and, in particular, raised abdominal pressure. Patients who have had large volume fluid resuscitation develop extensive tissue oedema, bowel distension, ascites and abdominal hypertension. These patients require higher airway pressure to achieve the same transpulmonary pressure, which means higher PEEP to restore end-expiratory lung volume.

Thus, a high abdominal pressure (estimated usually by bladder pressure, see module on Abdomen in acute/critical care medicine) will be transmitted to the pleura, thus increasing the pleural pressure. In these circumstances, a higher than normal airway pressure will not induce an unsafe transpulmonary pressure and may therefore be used.

Estimation of static airway pressures

On controlled ventilation

Identifying and interpreting the data provided by modern ventilators is helpful in evaluating respiratory mechanics during artificial ventilation. Modern ventilators provide complete monitoring of respiratory system mechanics, which is useful towards guiding the optimisation of ventilatory support and avoiding complications associated with mechanical ventilation.

Lucangelo U, Bernabé F, Blanch L. Respiratory mechanics derived from signals in the ventilator circuit. Respir Care 2005; 50(1): 55–65; discussion 65–67. Review. PMID 15636645

Compliance and resistance

Compliance: The assessment of the compliance of the respiratory system (C_{rs}) and of the resistance of the respiratory system (R_{rs}) in (pharmacologically)

paralysed patients can be made during constant-flow, Volume-Controlled Ventilation, with the end-inspiratory occlusion technique.

During the pause performed at end-inspiration, the flow drops rapidly to zero, the Vt is briefly trapped inside the lung and static airway pressure can be measured. The static airway pressure waveform (figure below) has a characteristic trend, with the highest peak at end-inspiration (peak inspiratory pressure;PIP), followed by a rapid drop after the occlusion (P1 in the figure below), and a slow decay until a plateau is reached (P2 in figure).

P2 is the static pressure of the respiratory system ($P_{st,rs}$) that, in the absence of flow, equals the alveolar pressure (P_{alv}), reflecting the elastic retraction of the entire respiratory system. The pressure drop from Pmax to P1, represents the pressure required to move the inspiratory flow along the airways without alveolar interference, thus representing the pressure dissipated by the flow-dependent resistances.

Static airway pressure wave
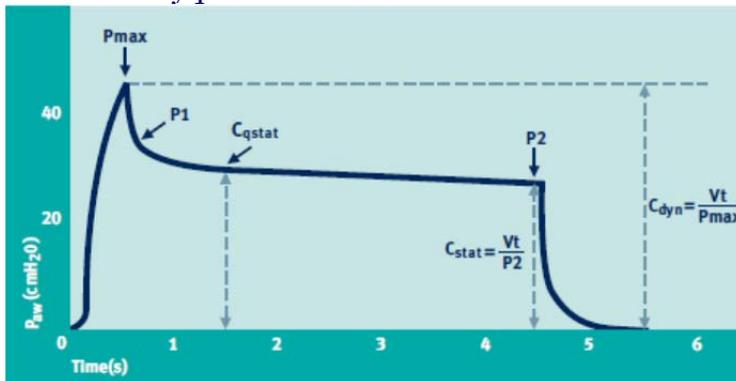
The slow decay after the occlusion from P1 to P2 depends on the visco-elastic properties of the system, i.e. the stress relaxation and on the pendulum-like movement of the air (Pendelluft).

Therefore, measurement of compliance (C_{rs}) will necessarily have to be performed with the following formula which also takes into consideration the possible use of PEEP.

$$C_{RS} = \frac{Vt}{P_{aw} - (PEEP + PEEPi)}$$

Static compliance (C_{stat}) and dynamic compliance (C_{dyn})

The difference between these is that for static compliance, the **volume variation** refers to the static plateau pressure (P2), while for dynamic compliance the **volume variation** refers to Pmax. Thus:

$C_{dyn} = Vt/Pmax$ and
$C_{stat} = Vt/P2$

P2 is also called Ppause or Pplat.

The static compliance of the respiratory system mirrors the elastic features of the respiratory system, whereas the dynamic compliance also includes the resistive (flow-dependent) component of the airways and the endotracheal tube.

In healthy subjects the difference between static compliance and quasi-static compliance (see figure above) is minimal, whereas it is markedly higher in patients who have acute respiratory distress syndrome or chronic obstructive pulmonary disease.

Resistance: The rapid airway occlusion technique during constant-flow inflation allows measurement of respiratory system resistances when the respective pressure gradients (Pmax – P2), (Pmax – P1), and (P1 – P2) are divided by the flow value ($\dot{V}$) immediately before occlusion.

> **NOTE** Resistance is defined as Pressure divided by Flow.

In particular, if we consider the (Pmax – P1)/$\dot{V}$), the airway flow-dependent resistance is obtained, which is commonly called 'initial' or 'minimal' or 'ohmic' resistance ($R_{RS,init}$). When the (P1 – P2) pressure gradient is taken into account, the so-called additional lung resistance value is obtained (ΔRrs), which reflects the Pendelluft phenomenon and the visco-plasto-elastic lung and thorax behaviour. Last, the total resistances (Rmax) that take into consideration the (Pmax – P2) pressure gradient represent the sum of $R_{RS,init}$ and ΔR_{rs}.

> **NOTE** In clinical practice, it is important to remember that the Pmax – P1 pressure gradient is flow-dependent, whereas P2 is affected only by variations in volume and/or compliance.

These calculated values are averages over the single breath. The values are not necessarily constant, however, but may vary due to, for example, expiration flow limitation, alveolar recruitment/derecruitment and alveolar over-distension. Measuring pressure from the trachea enables estimation of the within-breath variation of the elastic and resistive properties of the lung and airways. Such patient monitoring facilities are available on modern ventilators.

> **Q. What is the clinical relevance of Pmax? Is it directly related to the risk of barotrauma?**

A. In isovolumetric conditions, variations in Pmax depend on the pressure dissipated to overcome resistance. Therefore, airway secretions, bronchospasm, and the diameter of the endotracheal tube affect Pmax. Peak pressure should not be considered in the assessment of the risk of barotrauma, because it does not have any consequence at the alveolar level.

> **Q. Is there any other practical assessment of the patient breathing system that the end-inspiratory occlusion manoeuvre allows?**

A. The end-inspiratory occlusion manoeuvre can identify a leak in the respiratory circuit; if there is a leak, the plateau pressure cannot be reached.

Söndergaard S, Kárason S, Wiklund J, Lundin S, Stenqvist O. Alveolar pressure
    monitoring: an evaluation in a lung model and in patients with acute lung
    injury. Intensive Care Med 2003; 29(6): 955–962. Epub 2003 Apr 11.
    PMID 12690438

On assisted breathing

When the patient's breathing activity is entirely passive and the pressure
developed by the respiratory muscles is negligible, the driving pressure
necessary to move air in and out of the thorax can be described by the simplified
equation of motion:

$$P_{RS} = P_{AO} = \dot{V} \times R + \frac{V}{C} + k$$

In which P_{rs} is the respiratory system pressure, P_{AO} is the pressure at the airway
opening, $\dot{V}$ is flow, R is resistance, V is volume, C is the respiratory system
compliance, and k is a constant that represents the alveolar end-expiratory
pressure.

Compliance-resistance

Dynamic mechanics may be derived during pressure support ventilation (PSV)
or VCV in intubated patients without flow interruption. Therefore, the
respective values of the R_{rs}, C_{rs} , and k can be obtained by applying the above
equation to the sample values of P_{AO} , V, and $\dot{V}$ with a multiple linear
regression analysis, or linear least square fitting (LSF).

P_{AO}, V and $\dot{V}$ variables, which change throughout the respiratory cycle, can be
digitised at high speed (100 Hz). In this way, C_{rs} and R_{rs} can be calculated by the
use of computers from 100 or more equations per breath. The LSF method does
not require a particular inspiratory flow pattern; it can be applied during the
whole breathing cycle or only in the inspiratory or expiratory phase. LSF
provides an estimate of C_{rs} and R_{rs}.

For an understanding of the clinical relevance of compliance and resistance see
the PACT module on Mechanical ventilation.

Auto-PEEP: time constants vs flow limitation

During the post-inspiratory occlusion period there is a dynamic elastic
rearrangement of lung volume. This arrangement allows the different pressures
in the different alveoli at different constants of time to equal each other, The
parts of the lung that have a low constant of time (rapid zones), where the
alveolar pressure rises rapidly, are emptied into the parts of lung that have a
higher constant of time (slow zones), where the pressure rises more slowly due
to higher resistance or to a lower compliance.

At the end of a normal expiration, in a normal subject, the alveolar pressure is close to zero. In the presence of high resistance to expiratory flows and short expiratory times, the respiratory system is unable to return to its resting volume at the end of exhalation. As a result, a new resting state is established, such that there is a positive recoil pressure (PEEPi) at the end of expiration and volume trapping. This state of air trapping or dynamic hyperinflation is common in patients with obstructive lung disease; however it can be seen also in patients with ARDS depending on the ventilatory settings. Auto-PEEP can be seen as a persistence of flow at end-expiration in the flow-time waveforms or as a truncation of the expiratory limb in the flow-volume loop. Auto-PEEP can be quantified performing an end-expiratory occlusion manoeuvre.

For detailed information, see the PACT module on Mechanical ventilation. See also the PACT module on COPD and asthma and the following reference.

Blanch L, Bernabé F, Lucangelo U. Measurement of air trapping, intrinsic positive end-expiratory pressure, and dynamic hyperinflation in mechanically ventilated patients. Respir Care 2005; 50(1): 110–123; discussion 123–124. PMID 15636649

Work of breathing

Work of breathing (WOB) is performed when a pressure changes the volume of the system and results in a pressure and volume change.

During partial ventilatory support, the tidal changes in volume are due to the combined action of the changes in P_{aw} and of the changes in the pressure applied by the respiratory muscles to the passive components of the respiratory system.

It is necessary to distinguish between WOB performed by the ventilator (W_{vent}) and work performed by the patient (W_{pat}). W_{vent} can be assessed from measurements taken from the signals of P_{aw} and respiratory system volume change (Vol), but for W_{pat}, it is necessary to obtain a measurement of the pressure changes generated by the respiratory muscles. W_{pat} can be calculated from measurements taken on the signals of P_{es} and Vol.

Distinction should also be made between the work performed during the inspiratory phase of the cycle (W_{insp}) and the work performed during the expiratory phase (W_{exp}).

The dimension of work is pressure multiplied by volume, and the unit is the joule. One joule is equal to $1\,L \times 1\,kPa$, i.e. $1\,L \times 10.2\,cmH_2O$.

The work performed in any given phase of a breath, as the product of pressure and volume, corresponds to the area enclosed under the dynamic pressure-volume curve for that phase.

Although in practice WOB is calculated by numerical integration, a graphic approach allows an easier understanding of the procedures and of the meaning of the results.

Work of breathing
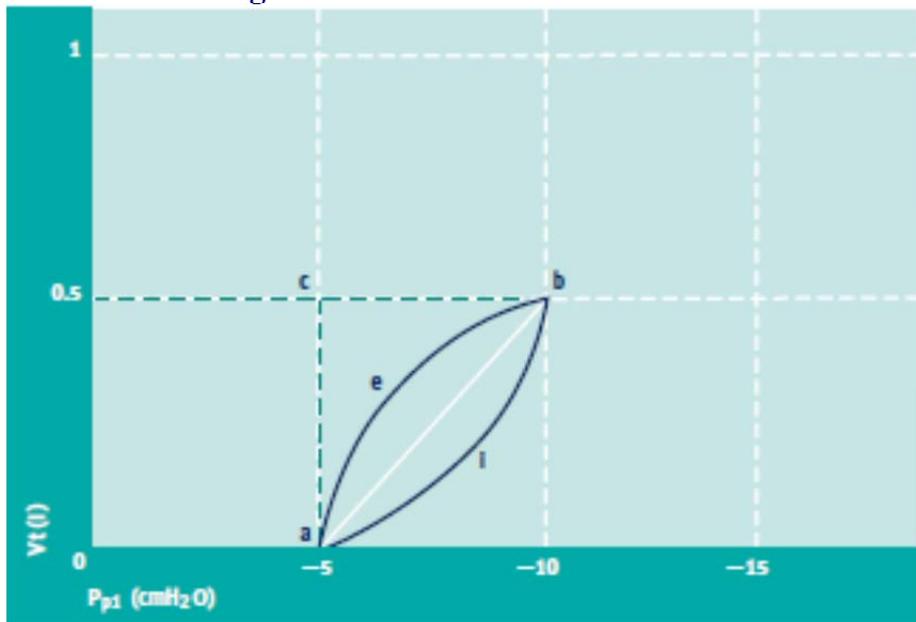
work = force × distance ≈ pressure × volume / 2
elastic work ∝ area a-b-c-a
inspiratory flow-resistive work ∝ area a-i-b-a
expiratory flow-resistive work ∝ area a-b-e-a
negative work ∝ area a-e-b-c-a (tone on inspiratory muscles during expiratory air flow)
total work ∝ Welastic + Winspiratory flow-resistive + Wnegative
passive recoil of lungs overcomes the work of expiratory flow resistance

The components of work
Elastic work – to overcome
- Lung elastic recoil
- Thoracic cage displacement
- Abdominal organ displacement

Frictional work – to overcome
- Airflow resistance (major)
- Viscous resistance (lobe friction, minor)

Inertial work – to overcome
- Acceleration and deceleration of air (negligible due to low mass of air)
- Acceleration and deceleration of chest wall and lungs (negligible due to overdamping)

Compared with the normal curve, the patient with emphysema has large increases in work due to increased airflow resistance (frictional work), whereas the patient with fibrosis has large increases in elastic work due to the stiffness of

the lung. Although the total inspiratory work loop is less in emphysema than in fibrosis, more work is required during expiration.

For functional residual capacity (FRC)/EELV (end-expiratory lung volume) considerations, see Task 4.

4/ LUNG RECRUITMENT AND PEEP

Concept of lung recruitment

Lung recruitment is an inspiratory phenomenon (different from PEEP in this respect) which can be defined as the inflation of previously collapsed alveolar units.

A **recruitment manoeuvre (RM)** is a transient increase in transpulmonary pressure intended to promote reopening of collapsed or flooded alveoli. A sustained pressure above the range of tidal ventilation is applied, and PEEP is used to prevent derecruitment. The result should be an increase in FRC/EELV (see below).

A variety of techniques have been described as recruitment manoeuvres:
- Sustained high-pressure inflation of 40 cmH_2O for 40 seconds using CPAP.
- Intermittent sighs, using three consecutive sighs at 45 cmH_2O per minute for 1 hour.
- A stepwise ('ramping') increase in PEEP every 2 min, keeping the driving pressure constant, up to peak pressures of 45–50 cmH_2O.
- Pressure-controlled ventilation (PCV) with PEEP of 25 to 30 cmH_2O and peak inspiratory pressure of 40 to 45 cmH_2O for 2 min.

Esan A, Hess DR, Raoof S, George L, Sessler CN. Severe hypoxemic respiratory failure: part 1–ventilatory strategies. Chest 2010; 137(5): 1203–1216. PMID 20442122

All recruitment manoeuvres can compromise the circulation by reducing venous return and inducing acute right heart failure (cor pulmonale), particularly if the patient is hypovolaemic. Therefore haemodynamics require careful monitoring during these procedures. A shorter procedure may be as effective as a long one, but will have a less adverse effect on the circulation. Below, only the (monitored) mechanical effects on the lung during recruitment manoeuvres are discussed.

Recent evidence shows that alveolar recruitment is achieved after a short RM and prolonging its duration increases adverse events, namely haemodynamic and barotrauma. Furthermore, more gradual, stepwise increases in pressure seem to be better tolerated and give greater recruitment than sustained high-pressure inflation.

NOTE A successful procedure will result in improved oxygenation, reduced arterial to end-tidal CO_2 gradient and improved compliance.

For more information, see the PACT modules on Acute respiratory failure and Mechanical ventilation.

Monitoring of lung recruitment

At the bedside, recruitment can be estimated from secondary indicators like respiratory system compliance (C_{rs}). Recent developments in ventilators have made automatic lung volume measurement available at the bedside thus providing direct information about the efficacy of the lung recruitment manoeuvres.

'Open up the lung and keep it open'

Olegård C, Söndergaard S, Houltz E, Lundin S, Stenqvist O. Estimation of functional residual capacity at the bedside using standard monitoring equipment: a modified nitrogen washout/washin technique requiring a small change of the inspired oxygen fraction. Anesth Analg 2005; 101(1): 206–212. PMID 15976233

CT

CT scans can detect and measure lung recruitability by quantifying the change in alveolar aeration following the application of two different levels of airway pressure. In ARDS, the percentage of potentially recruitable lung is variable and is strongly associated with the response to PEEP.

Gattinoni L, Caironi P, Cressoni M, Chiumello D, Ranieri VM, Quintel M, et al. Lung recruitment in patients with the acute respiratory distress syndrome. N Engl J Med 2006; 354(17): 1775–1786. PMID 16641394

Automated FRC/EELV measurements using nitrogen washout

Measuring functional residual capacity (FRC), or end-expiratory lung volume (EELV) when PEEP is applied, may help to measure the aerated lung available for ventilation and better monitor the effects of ventilation strategies. Nitrogen washout is the simplest FRC or EELV measurement method in the mechanically ventilated patient and can be measured without interrupting mechanical ventilation. However, the nitrogen washout method requires accurate measurement of inspired and expired oxygen and carbon dioxide through a metabolic monitoring module.

To calculate EELV with this method, a change in alveolar fraction of N_2 is measured after a step change in the inspired gas O_2 fraction. This follows the principle that the gas volume in the lung, includes a volume of nitrogen ($V_{(1)}N_2$) that is determined by the product of the alveolar fraction of nitrogen ($F_AN_{2(1)}$) (which varies inversely to the alveolar oxygen fraction) and the EELV.

If the alveolar nitrogen fraction ($F_AN_{2(2)}$) changes following a step change in FiO_2, a new nitrogen volume will be present in the lung so,

$$VN_{2(1)} - VN_{2(2)} = (F_AN_{2(2)} - F_AN_{2(1)}) \times EELV$$

And as the changes in F_AN_2 are proportional to the changes in FiO_2, the EELV can be calculated as:

$$EELV = \Delta N_2 \, (ml)/\Delta FiO_2$$

Calculation of EELV requires relatively stable conditions and moderate FiO_2 requirements with FiO_2 or 0.4–0.65.

Chiumello D, Cressoni M, Chierichetti M, Tallarini F, Botticelli M, Berto V, et al. Nitrogen washout/washin, helium dilution and computed tomography in the assessment of end expiratory lung volume. Crit Care 2008; 12(6): R150. PMID 19046447

Olegård C, Söndergaard S, Houltz E, Lundin S, Stenqvist O. Estimation of functional residual capacity at the bedside using standard monitoring equipment: a modified nitrogen washout/washin technique requiring a small change of the inspired oxygen fraction. Anesth Analg 2005; 101(1): 206–212. PMID 15976233

Lung mechanics

Dynamic Compliance

A decremental PEEP trial after full lung recruitment allows PEEP titration along the deflation limb of the pressure-volume (PV) curve while observing changes in both oxygenation and lung mechanics. During a decremental PEEP trial, the point of maximal tidal compliance and the point of maximum curvature have been shown to correspond to open lung-PEEP.

Suarez-Sipmann F, Böhm SH, Tusman G, Pesch T, Thamm O, Reissmann H, et al. Use of dynamic compliance for open lung positive end-expiratory pressure titration in an experimental study. Crit Care Med 2007; 35(1): 214–221. PMID 17110872

Constant-flow manoeuvres

During a 'low-flow manoeuvre', inspiratory volume is delivered at a flow of 3 L/min to minimise the effect of airway resistence. Pressure and volume of the respiratory system are measured. Calculating the hysteresis (difference in lung volume between expiration and inspiration at the same airway pressure) of a pressure-volume curve may quantify lung recruitment. Traditionally, the lower inflection point (LIP) has been interpreted as the average opening pressure

pressure at which collapsed alveoli are recruited. However, LIP represents only the pressure at which the collapsed lung units start to open, and most the opening occurs at higher pressure than LIP. More recently, interest has shifted towards the point of maximum curvature (PMC) on the expiratory limb of the pressure-volume loop, which represents the pressure at which lung volume starts to decrease. The average pressure at which derecruitment starts to occur is located at the maximum compliance of the expiratory limb (in agreement with the maximum compliance decremental PEEP trial, see above). PEEP levels below the point of maximal curvature of the expiratory limb or at the maximum compliance of the expiratory limb, are theoretically the targets for setting PEEP in ARDS.

Hickling KG. Best compliance during a decremental, but not incremental, positive end-expiratory pressure trial is related to open-lung positive end-expiratory pressure: a mathematical model of acute respiratory distress syndrome. Am J Respir Crit Care Med 2001; 163(1): 69–78. PMID 11208628

Link to ESICM Flash Conference: Anders Larsson. Pressure-volume curves: are they useful? ESICM congress, Berlin 2011

Transpulmonary pressure

The use of an oesophageal balloon to assess intrapleural pressure has been advocated to allow more precise setting of PEEP. If pleural pressure is high relative to alveolar pressure (i.e. PEEP), there may be potential for alveolar derecruitment. In this case, it is desirable to keep PEEP greater than pleural pressure. The transpulmonary pressure can then be adjusted by titrating applied PEEP, since airway pressure is related to applied PEEP. Titrating applied PEEP to an end-expiratory transpulmonary pressure between 0 and 10 cmH_2O may reduce cyclic alveolar collapse, while maintaining an end-inspiratory transpulmonary pressure ≤25 cmH_2O may reduce alveolar over-distension.

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

'Pressure-Time' index (Stress Index)

The stress index was recently proposed to assess the level of PEEP to avoid over-distension. This approach uses the shape of the pressure-time curve during tidal volume delivery. Worsening compliance as the lungs are inflated (upward concavity; stress index, >1) suggests over-distension, and the recommendation is to decrease PEEP. Improving compliance as the lungs are inflated (downward concavity; stress index, <1) suggests tidal recruitment and potential for additional recruitment, thus a recommendation to increase PEEP.

Ranieri VM, Zhang H, Mascia L, Aubin M, Lin CY, Mullen JB, et al. Pressure–
time curve predicts minimally injurious ventilatory strategy in an isolated
rat lung model. Anesthesiology 2000; 93(5): 1320–1328. PMID 11046222

5/ WEANING ASSESSMENT AND MONITORING

Intensive Care clinicians routinely wean 'three types' of patients in their ICUs:

- Patients admitted with acute illness leading to respiratory failure, but otherwise healthy e.g. poisoning, postoperative patients.
- Patients having acute prolonged illness causing respiratory failure with lung injury e.g. ARDS patients in the fibro-proliferative phase with no sequelae, patients with pneumonia, aspiration pneumonia or trauma.
- Patients improving after exacerbation of chronic diseases (COPD, cystic fibrosis, restrictive lung diseases). These are the most difficult to wean. Overall problems increase with the patient's age, length of the chronic disease and comorbidities.

Mechanical ventilation substitutes for the respiratory pump until these illnesses/disturbances have been reversed adequately to allow resumption of spontaneous breathing and gas exchange.

Q. Why is early weaning important?

A. Because mechanical ventilation has numerous risks, including infection and barotrauma.

Complications related to tracheal tube
- Tracheal tube malfunction: mucus plug, cuff-leak
- Tracheal tube malposition
- Self-extubation
- Nasal or oral necrosis
- Laryngeal oedema
- Tracheal erosion
- Sinusitis

Complications related to ventilator
- Alveolar hypoventilation/hyperventilation
- Atelectasis
- Ventilator-associated Pneumonia (VAP)
- Hypotension
- Pneumothorax
- Diffuse alveolar damage

Effects of other organ system
- Gastrointestinal hypomotility
- Pneumoperitoneum
- Stress gastropathy and gastrointestinal haemorrhage
- Arrhythmias
- Salt and water retention
- Malnutrition

To avoid or minimise complications, it is appropriate to determine each day whether the patient can be weaned. This task should highlight methods used to

identify patients who are appropriate candidates for weaning from mechanical ventilation.

> **NOTE** It is important to wean patients as soon as possible from mechanical ventilation, since the main risk factor for ventilator-associated pneumonia (VAP) is the presence of a trans-laryngeal tube.

After 72 hours, 50% of ventilated patients are colonised by Gram-negative bacteria. Some authors have shown that early-onset pneumonia may account for as many as 50% of cases of VAP, and most organisms represent common respiratory tract pathogens or normal (Gram-negative) oro-pharyngeal flora. Several studies have emphasised a high risk for VAP in the first week.

Rello J, Soñora R, Jubert P, Artigas A, Rué M, Vallés J. Pneumonia in intubated patients: role of respiratory airway care. Am J Respir Crit Care Med 1996; 154(1): 111–115. PMID 8680665

Chevret S, Hemmer M, Carlet J, Langer M. Incidence and risk factors of pneumonia acquired in intensive care units. Results from a multicenter prospective study on 996 patients. European Cooperative Group on Nosocomial Pneumonia. Intensive Care Med 1993; 19(5): 256–264. PMID 8408934

Q. What is the incidence of VAP after the beginning of mechanical ventilation?

A. During the first week of mechanical ventilation pneumonia rates of approximately 3% per day pertain. In the second week, it is 2% per day and, in the third week and beyond, the rate is 1%.

Cook DJ, Walter SD, Cook RJ, Griffith LE, Guyatt GH, Leasa D, et al. Incidence of and risk factors for ventilator-associated pneumonia in critically ill patients. Ann Intern Med 1998; 129(6): 433–440. PMID 9735080

Patients can be classified into:
1. **Simple to wean**: patients are extubated on the first attempt (about 70% of the cases);
2. **Difficult to wean**: patients requiring up to three extubation attempts and taking 7 days (or less) from commencement of weaning;
3. **Prolonged wean:** patients requiring >3 extubation attempts or >7 days from commencing weaning.

In some patients full weaning is not possible, due to the lack of recovery from a primary or intercurrent, cardiac or respiratory illness. The transition from mechanical ventilation to spontaneous ventilation might be difficult. In fact, controlled mechanical ventilation induces diaphragmatic muscle wasting within a day that may contribute to weaning failure. Positive pressure ventilation (PPV)

is the most common mode of mechanical ventilation used in critically ill patients

See the PACT module on Mechanical ventilation for more information.

PPV can deliver an inspiratory volume or pressure to the lungs and can provide PEEP. The principal effects of PPV and PEEP are to reduce left and right preload and left ventricular afterload. Right ventricular afterload might be increased by augmented lung volumes. When patients begin to breathe spontaneously, intrathoracic pressure turns negative. The consequence is that venous return and the left ventricular afterload increase.

> The transition from PPV to spontaneous breathing might be accompanied by multiple events including left ventricular failure, myocardial infarct and pulmonary oedema.

Healthy patients tolerate these changes with little or no problems. Weaning patients with coronary artery diseases might precipitate ischaemia. Sometimes, the increased venous return can be associated with pulmonary oedema and hypoxaemia. In these patients discontinuation of mechanical ventilation can precipitate the symptoms and signs of recurrent ventilatory failure.

Recognition of the cardiac origin of weaning failure is crucial since the use of cardiac medication e.g. vasodilators and/or diuretics may result in successful weaning.

Brochard L, Rauss A, Benito S, Conti G, Mancebo J, Rekik N, et al. Comparison of three methods of gradual withdrawal from ventilatory support during weaning from mechanical ventilation. Am J Respir Crit Care Med 1994; 150(4): 896–903. PMID 7921460

⚠ The discontinuation of mechanical ventilation increases the propensity for atelectasis, especially in patients with respiratory muscle weakness, respiratory depression (poisoning), or restrictive physiology such as obesity. In patients with lung injury, surfactant depletion and ultrastructural lung changes increase the likelihood of alveolar collapse.

Readiness to wean

Assessment of readiness to wean requires two steps:
1. Clinical criteria
2. Weaning predictor parameters

Clinical criteria

- The cause of the respiratory failure has improved.
- Adequate oxygenation (PaO_2/FiO_2) ≥20 kPa (150 mmHg) or an oxyhaemoglobin saturation (SpO_2) ≥90 per cent while receiving an FiO_2

≤0.4 per cent and a positive end-expiratory pressure (PEEP) ≤5 cmH_2O.
- Arterial pH >7.25.
- Haemodynamic stability, without myocardial ischaemia.
- The patient is able to initiate an inspiratory effort.
- Haemoglobin level ≥7 to 10 g/L.
- Core temperature ≤38 to 38.5 °C.
- A mental status that is either awake and alert, or easily rousable.
- Adequate cough and absence of excessive secretion load.

Weaning

Weaning parameters are objective criteria used to assess the readiness of patients to sustain successful, spontaneous ventilation. We will review the commonly used weaning parameters.

Oxygenation:
- SaO_2 >90% on FiO_2 <0.4
- PaO_2 >6.7–8.0 kPa (50–60 mmHg) on FiO_2 <0.5
- PaO_2/FiO_2 ≥20 kPa (>150 mmHg)

Measures of neuromuscular function:
- Maximal inspiratory pressure (MIP, Pimax): global assessment of the strength of the inspiratory muscles. Aim for −30 cmH_2O or less (i.e. more negative)
- Airway occlusion pressure generated in the first 0.1 second (P0.1) of inspiration is a measurement of respiratory drive. Values of P0.1 which are more negative than −4 cmH_2O reflect high respiratory load and have been associated with weaning failure in some studies
- Vital capacity (difficult in children and in adults who do not cooperate or who are not competent)

FVC as a guide to clinical assessment	
FVC >50 mls/kg	Normal
FVC 15–20 mls/kg	May self-ventilate for part of the day May require assistance to clear tracheal secretions
FVC 7–15 mls/kg	Likely to require ventilatory support for part of the day Likely to require assistance to clear tracheal secretions
FVC <7 mls/kg	likely to require ventilatory support most of the day Likely to require assistance to clear tracheal secretions

- Maximum voluntary ventilation (MVV) (difficult in children and in adults who do not cooperate or who are not competent)
- Oxygen cost of breathing. VO_2 on/off support.

Measures of respiratory muscle load:
- Minute volume (Vm): RR <35/min; Vt >5 mL/Kg; Vm <10–15 L/min

- Respiratory compliance
- Airway resistance

Measures of the effect of weaning on other organs:

Integrated indices. While several of these integrated indices look promising, none have been confirmed as having the high level of accuracy. The most important are:

- **Inspiratory effort quotient (IEQ)** = $[(0.75 \, Vt/C_{dyn}) \times (Ti/T_{TOT})] /$ MIP, where Vt is the tidal volume, C_{dyn} is the dynamic compliance, Ti is the inspiratory time, T_{TOT} is the respiratory duty cycle, and MIP is the maximal inspiratory pressure. An IEQ >0.15 has been suggested as the fatiguing threshold that predicts weaning failure.
- The **CROP index** (Compliance, Rate, Oxygenation, Pressure. ml/breath/min) = $[C_{dyn} \times MIP \times (PaO_2/PAO_2)] /$ Fr.
  CROP > 13 mL/breath/min is predictive of weaning success.
- **Weaning Index (WI)** – index of Jabour = PTI $\times$ (Ve40/VTsb)
  - PTI is the modified pressure-time index
  - Ve40 is the minute ventilation needed to bring $PaCO_2$ to 5.3 kPa (40 mmHg)
  - Vtsb is the tidal volume during spontaneous breathing. Both PTI and Ve40 are measures of ventilatory endurance. A threshold of 4 min^{-1}, the WI was highly accurate in predicting weaning outcome.
- **Integrative weaning index (IWI)** = $[(C_{stat}) \times SaO_2] / [Fr/Vt]$. An IWI ≥ 25 mL/cmH$_2$O/breaths/min/Litre predicted successful weaning
  - C_{stat} =the static compliance (C_{stat})
  - SaO_2 is the arterial oxygen saturation
  - Fr is the respiratory rate
  - Vt is the tidal volume.
- **Rapid Shallow Breathing Index (RSBI)** = Fr/Vt (measured in Litres)
  RSBI is also called the Rate:Volume Ratio.
  A value of <105 breaths/min/L is predictive of weaning success.

Yang KL, Tobin MJ. A prospective study of indexes predicting the outcome of trials of weaning from mechanical ventilation. N Engl J Med 1991; 324(21): 1445–1450. PMID 2023603. doi:10.1056/NEJM199105233242101.

⚠ The predictive accuracy of some mentioned weaning parameters is not consistent clinically because large studies have yielded conflicting results. The main reason could be that patient populations, methods of 'weaning' and definition of success and failure were different among these studies.

Spontaneous breathing trials (SBT): May be implemented with PSV e.g. PSV 5 cmH$_2$O and PEEP 5 cmH$_2$O (with automatic tube compensation if available) or by T-tube trial as both are suitable methods for successful

discontinuation of ventilator support in patients without problems in resuming spontaneous breathing. Trials of spontaneous breathing are advocated for all mechanically ventilated patients who are rousable, haemodynamically stable, without inotropes or vasopressors, without comorbidities, on low ventilatory and end-expiratory pressure requirements, and requiring levels of FiO_2 that could be safely delivered with a face mask or a nasal cannula. The oxygenation is considered adequate when, at a concentration of inspired oxygen of less than or equal to 50%, and at levels of PEEP less than or equal to 5 cmH$_2$O, the PaO$_2$ is greater than 8 kPa (60 mmHg). Excessive respiratory muscle loads such as severe bronchospasm must be reversed before the beginning of the trial.

An SBT is currently the best predictor of successful weaning, with at least 77% of patients passing an SBT being successfully extubatable.

> ⚠ There is little difference between low level PSV and T-piece trials as a pre-extubation mode, Esteban (reference below) showed that the WOB with PSV is slightly more compared to the WOB with T-tube. After a first trial of spontaneous breathing, successful weaning and extubation are achieved equally efficiently with assessments targeted to last 30 minutes or 120 minutes.

In conclusion, an SBT of 30 minutes is adequate to predict successful weaning in 60–80% of ICU patients.

Esteban A, Alía I, Gordo F, Fernández R, Solsona JF, Vallverdú I, et al. Extubation outcome after spontaneous breathing trials with T-tube or pressure support ventilation. The Spanish Lung Failure Collaborative Group. Am J Respir Crit Care Med 1997; 156(2 Pt 1): 459–465. PMID 9279224

Esteban A, Alía I, Tobin MJ, Gil A, Gordo F, Vallverdú I, et al. Effect of spontaneous breathing trial duration on outcome of attempts to discontinue mechanical ventilation. Spanish Lung Failure Collaborative Group. Am J Respir Crit Care Med 1999; 159(2): 512–518. PMID 9927366

There is an important distinction between liberation from mechanical ventilation and extubation. Once a patient has successfully performed a trial of spontaneous breathing, he/she is liberated from the ventilator but the physician should determine whether the patient can be extubated or whether he/she still needs an artificial airway (tracheal tube).

> **NOTE** In patients with mask ventilation, weaning involves periods of full spontaneous breathing, with or without CPAP. In patients with tracheostomy, the last step is normally represented by intermittent ventilation with periods of PSV alternated with periods of spontaneous breathing on CPAP or T-piece.

Weaning and sedation protocols

Check patients' breathing capabilities on a daily basis. Sedation protocols which incorporate the avoidance of continuous sedation and the performance of daily

neurologic examinations reduce, in adults, the median duration of mechanical ventilation, the median length of stay in the ICU and the number of diagnostic tests performed.

Kress JP, Pohlman AS, O'Connor MF, Hall JB. Daily interruption of sedative infusions in critically ill patients undergoing mechanical ventilation. N Engl J Med 2000; 342(20): 1471–1477. PMID 10816184

Protocols for weaning: Weaning screen, sedation hold and an SBT represent a protocol for weaning from mechanical ventilation that can be performed by nurses and respiratory therapists. Use of a weaning protocol was associated with increased collaborative decision-making for determining weaning and extubation readiness and weaning method. Nurse-led protocols are safe and can lead to more rapid extubation particularly in units with low medical staffing levels.

Kollef MH, Shapiro SD, Silver P, St John RE, Prentice D, Sauer S, et al. A randomized, controlled trial of protocol-directed versus physician-directed weaning from mechanical ventilation. Crit Care Med 1997; 25(4): 567–574. PMID 9142019

It has been recommended to use daily spontaneous breathing trials with T-piece or a low level of PSV with CPAP 5 cmH$_2$O (where not contraindicated) and accept a reintubation risk between 5–20%.

Automated weaning mode

Commercially available closed loop weaning software packages are able to automate weaning by pressure support. The aim of the expert weaning system (EWS) is to ventilate the patient in a comfort zone, while reducing pressure support to a minimum level. A comfort zone is defined as a level of pressure support in which patients breathe with a respiratory rate of 15–30/min, a Vt >250 mL if <55 kg BW, or >300 mL if ≥55 kg BW, and with a measured EtCO$_2$ 7.3 kPa (<55 mmHg) if no COPD or <8.7 kPa (65 mmHg) if COPD present. Once a patient is stable at a specific level of pressure support, the program automatically reduces the pressure support level and reassesses respiratory stability. Three trials have compared an automated weaning system to usual care with mixed results: two trials demonstrated a shorter duration of mechanical ventilation, one trial found no difference when compared to nurse-led weaning. No trial has shown harm from automated weaning. However, patients with PaCO$_2$ greater than the target range of the weaning software should not be weaned by automated systems.

Rose L, Presneill JJ, Johnston L, Cade JF. A randomised, controlled trial of conventional versus automated weaning from mechanical ventilation using SmartCare/PS. Intensive Care Med 2008; 34(10): 1788–1795. PMID 18575843

Lellouche F, Mancebo J, Jolliet P, Roeseler J, Schortgen F, Dojat M, et al. A multicenter randomized trial of computer–driven protocolized weaning from mechanical ventilation. Am J Respir Crit Care Med 2006; 174(8): 894–900. PMID 16840741

Dojat M, Brochard L. Knowledge-based systems for automatic ventilatory management. Respir Care Clin N Am 2001; 7(3): 379–396, viii. PMID 11517029

Dojat M, Pachet F, Guessoum Z, Touchard D, Harf A, Brochard L. NéoGanesh: a working system for the automated control of assisted ventilation in ICUs. Artif Intell Med 1997; 11(2): 97–117. PMID 9332706

Dojat M, Brochard L, Lemaire F, Harf A. A knowledge-based system for assisted ventilation of patients in intensive care units. Int J Clin Monit Comput 1992; 9(4): 239–250. PMID 1484275

CONCLUSION

Respiratory monitoring has made great strides in the past few decades helping us to develop the tools that are now established in Intensive Care Medicine. These facilitate the monitoring of ventilation in acute and complex disease states and also in facilitating the weaning from mechanical ventilation. Newer technologies, meanwhile, have provided methods that, although novel and 'glamorous', have not proven themselves superior in the clinical area. Ongoing experimental research continues to broaden our knowledge of underlying physiological processes and the challenge is in the translation from physiologic understanding to clinical efficacy.

6/ 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.

AP_L (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. (AP_L = 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 ventilatory 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.

CaO_2

Content of oxygen in arterial blood. CaO_2 is calculated as $1.34 \times Hb \times SaO_2$; the normal value is 16 to 20 mL O_2/100 mL blood.

Compliance

    C_{rs} (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 (C_{dyn})and static (C_{stat}) compliance.

    C_{cw}

Compliance of the chest wall.

    C_{dyn}

Dynamic compliance. It is calculated as $\dfrac{Vt}{Peak\ Paw - PEEP}$

    C_L

Lung compliance.

C$_{qs}$
(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 end-expiration. It mimics true static compliance and is termed quasi-static compliance. True static compliance is utilised mainly in research and is performed using pressure measurements after serial volume increments with a 'super syringe'.

C$_{stat}$ Static compliance (see above). It is calculated as

$$\frac{Vt}{Ppause - PEEP}$$

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$\bar{v}$O$_2$ Mixed venous oxygen content. It is measured as $1.34 \times Hb \times S\bar{v}O2$ (mixed venous oxygen saturation).

ΔPCO$_2$ Difference between arterial to end-tidal PCO_2.

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

DO$_2$ Oxygen delivery – measured as $CO \times CaO_2$.

EtCO$_2$ End-tidal CO_2 – see also $PetCO_2$.

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 PO_2 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 PI_{max}.
MVV	Maximum voluntary ventilation.
NI(M)V	Non-invasive (mechanical) ventilation.
NIF	Negative inspired force.
Normoxaemia	Normal blood levels of oxygen.
$PaCO_2$	Partial pressure of arterial carbon dioxide – normal range 4.7–6 kPa (35–45 mmHg).
P_{alv}	Alveolar pressure.
P_{AO}	Pressure at airway opening.
PaO_2	Partial pressure of arterial oxygen – normal range 10–13.3 kPa (75–100 mmHg).
P_{atm}	Atmospheric pressure.
P_{aw}	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.
$PECO_2$	Partial pressure of CO_2 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 H_2O) 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.
P_{es} (oesophageal pressure)	Ppressure in the lower one third of the oesophagus when the patient is upright. It equates to pleural/extra-alveolar pressure.
$PetCO_2$ (End-tidal CO_2)	The highest value of CO_2 partial pressure during the alveolar plateau of the capnography curve.
PI_{max}	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.
P_{pl}	Pleura pressure.
P_{rs}	Respiratory system pressure.
PSG	Polysomnography.
PSV	Pressure support ventilation.

R (Resistance)	Respiratory system resistance (R_{rs})– refers to airway resistance and comprises the inflating pressure divided by the (gas) flow. See second case in the PATCH for an example of its measurement.
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.
T$_{TOT}$	is the respiratory duty cycle
V	Volume.
$\dot{V}$	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 CO_2 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/Vt$_{phys}$	Physiologic dead space.
Vd$_{alv}$	Alveolar dead space (alveoli well ventilated but receiving minimal blood flow).
Vd$_{anat}$	Anatomic dead space (upper and lower airways).
Vd$_{ins}$	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.
VO$_2$	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.
W$_{exp}$	Work of breathing performed during the expiratory phase.

|	
W_{insp}	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.
W_{pat}	Work of breathing performed by the patient.
W_{vent}	Work of breathing performed by the ventilator.

SELF-ASSESSMENT

EDIC-style Type K

1. Regarding the use of plain chest radiographs in the ICU:

    A. Should be performed as a daily routine in all patients
    B. Is not necessary after insertion of a central venous catheter (CVC) to the superior vena cava
    C. Should be performed after endotracheal intubation
    D. Because of radiation exposure should be limited to once per day

2. A normal chest X-ray rules out the following diagnoses:

    A. Bronchitis
    B. Right side lower lobe atelectasis
    C. Pulmonary embolism
    D. Early stage of pneumonia

3. CT scanning of the lungs is an important diagnostic tool in ARDS. Which of the following statements is/are true?

    A. A high-resolution CT will sometimes differentiate pulmonary from non-pulmonary causes of ARDS
    B. Alveolar consolidation is a more prevalent finding with non-pulmonary ARDS
    C. A CT scan can give valuable information about the recruitability of the lung
    D. The CT scan can be used to evaluate the level of PEEP

4. Lung ultrasound is a useful bedside examination in the diagnosis of:

    A. Pleural effusion
    B. Pneumothorax
    C. Lobar pneumonia
    D. Atelectasis

5. The oxyhaemoglobin dissociation curve (see figure) changes during different clinical conditions: Indicate the true (and false) statements.

    A. Anaemia has no influence on the shape of the curve
    B. During hyperbaric conditions, the curve is shifted upwards (increase in SaO_2)
    C. With increasing age the curve shifts to the right (p50 increases)
   D. In hypothermia, the curve shifts to the left (reduced p50)

- ● p50
- — typical
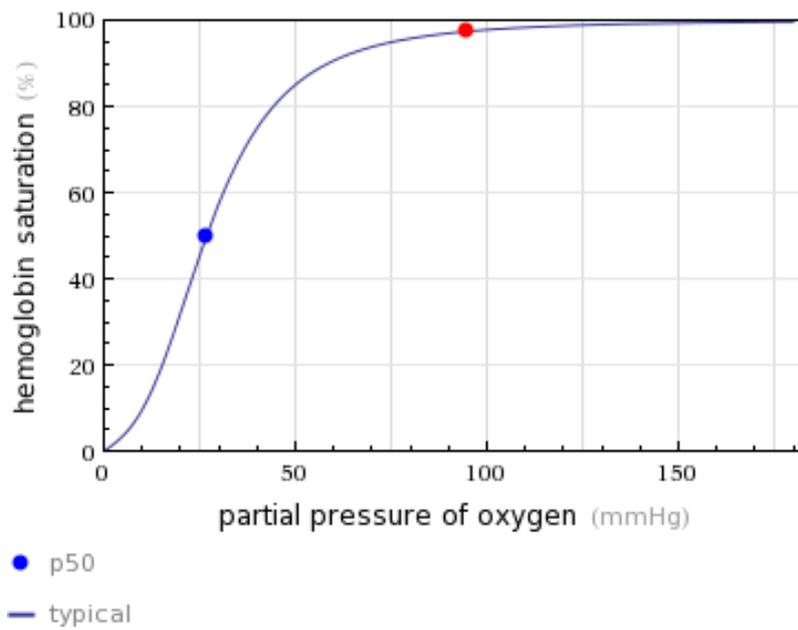
6. Oxygen delivery is dependent on

    A. Oxygen content in venous blood
    B. Cardiac output
    C. The influence of blood viscosity
    D. Haemoglobin content

7. The figure below shows time capnography traces; a normal capnogram (black) and a patient's abnormal capnogram (red). What are the most likely pathophysiological consequences/findings in such a patient?

    A. A fall in cardiac output
    B. A fall in $PaCO_2$
    C. A gradual rise in $PaCO_2$
    D. A gradual decrease in pH

CO₂ Conc.
(kPa, mmHg)

Time
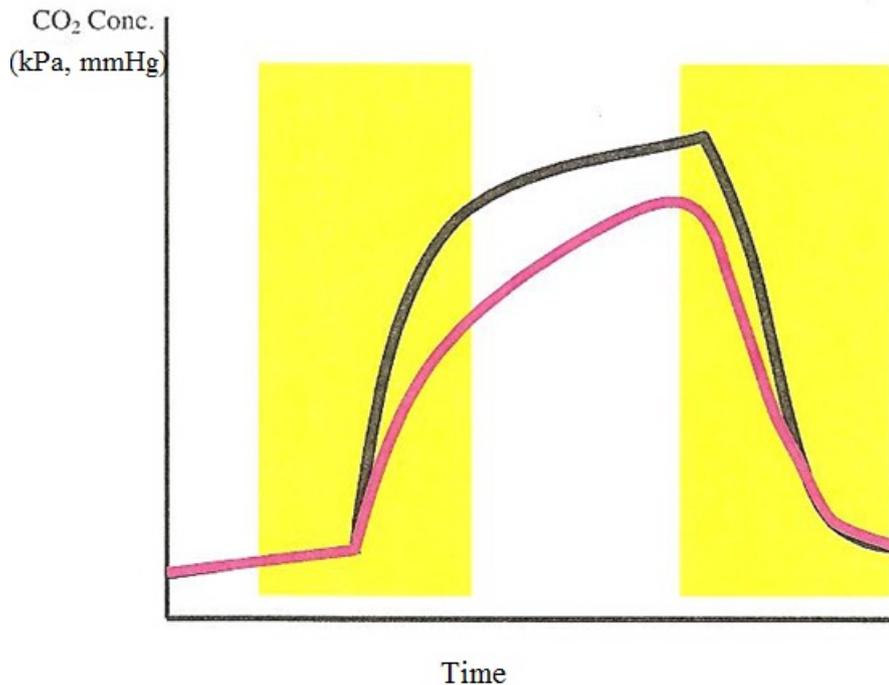
8. Regarding the use of extravascular lung water (EVLW) measurements in the ICU:

    A. Can be measured using a fibre optic pulmonary artery catheter
    B. The usual method is using transpulmonary thermodilution technique
    C. Normal values are >10 mL/kg
    D. Abnormal values are associated with increased duration of mechanical ventilation

9. The plateau/pause pressure (Ppause or Pplat) during intermittent positive pressure ventilation:

    A. Is difficult to measure during mechanical ventilation
    B. Is the most accurate reflection of alveolar pressure during inspiration
    C. Ppause or Pplat is not possible to measure during pressure-controlled ventilation
    D. Is considered to reflect airway resistance

EDIC-style Type A

10. Chest radiographs taken in the ICU differ from ordinary chest X-rays taken in the radiology department for all the following reasons EXCEPT

    A. The radiograph is antero-posterior in contrast to posterior-anterior
    B. The radiograph is more often taken in the supine position
    C. There is a decreased distance from the heart to the film
    D. A wide mediastinum is more often misdiagnosed
    E. The heart is approximately 15% wider.

11. Electrical impedance tomography can be used to evaluate or measure all of the following EXCEPT

    A. Regional distribution of ventilation
    B. Regional compliance
    C. The 'best PEEP' level
    D. Regional distribution of pulmonary blood flow
    E. Identification of a pleural effusion

12. A patient is admitted to your ICU with the following blood results:

    - PaO_2 10 kPa (80 mmHg)

    - $PaCO_2$ 3.9 kPa

    - SaO_2 87%

    - Hb 8.2 g/dL

    - Haematocrit 0.35

The oxygen content in 1000 ml blood in this patient is:

    A. 84 mL
    B. 96 mL
    C. 103 mL
    D. 110 mL
    E. 114 mL

13. Which of the following changes in a patient's clinical condition would have the largest impact on the oxygen content in the blood (all other factors assumed constant)?

    A. A reduction in PaO_2 from 14 to 10 kPa
    B. A reduction in haemoglobin concentration from 15 to 10 g/dL
    C. A reduction in Oxygen saturation from 96 to 86%
    D. An increase in Carboxyhaemoglobin from 0 to 10%
    E. A reduction from 3 atmospheric pressure to 1 atmospheric pressure, FiO_2 constant.

14. The following conditions are recognised causes of erroneous pulse oximetry readings EXCEPT

    A. Hypothermia
    B. Cardiogenic shock
    C. The presence of increased amount of carbon monoxide in the blood
    D. Hyperdynamic septic shock
    E. Varnished nails (when used on a finger)

15. A physiologically normal central venous oxygenation is:

    A. 50–55%
    B. 55–60%
    C. 60–65%
    D. 65–70%
    E. 70–80%

16. We often describe compliance using two different terms: static and dynamic compliance. Which is the correct answer regarding the differences between the two?

    A. Dynamic compliance is usually higher than the static compliance
    B. Static compliance is measured at the peak inspiratory pressure prior to the inspiratory pause
    C. Dynamic compliance is considered to mirror elastic forces in the lung
    D. Static compliance is equal to tidal volume (Vt)/end-inspiratory plateau pressure (Ppause)
    E. Static compliance reflects the elastic and flow-dependent components of the respiratory system.

17. A patient with severe asthma was intubated and ventilated. The ventilator was set to volume control, Vt 400 ml, RR 16, Insp: Exp time 1:3, PEEP 0, FiO_2 0.5. You measure the PEEPi and find it increased to 18 cmH_2O. What is the single most appropriate action?

    A. Decrease Vt
    B. Decrease RR
    C. Increase PEEP
    D. Increase inspiratory time
    E. Change to pressure-controlled ventilation

Self-assessment answers

1. FFTF
2. FTFF
3. TFTT
4. TTTT
5. TFFT
6. FTTT
7. FFTT
8. FTFT
9. FTFF
10. Correct: C
11. Correct: D
12. Correct: B
13. Correct: B
14. Correct: D
15. Correct: E
16. Correct: D
17. Correct: B

PATIENT CHALLENGES

A 68-year-old, 70 kg woman with COPD (40-year history of cigarette smoking) has worsening cough and dyspnoea and is admitted by ambulance to the emergency department (ED) of your hospital where she is found to be confused, in respiratory distress and coughing yellow sputum. She has no haemoptysis or chest pain; her medications are theophylline and albuterol. She has no other relevant history but her recent spirometric values at out-patient clinic showed a forced vital capacity (FVC) of 1.5 L and forced expiratory volume in 1 second (FEV1) of 0.7 L.

Immediate measures were to sit her up to reduce her work of breathing, start nasal oxygen 2 L/min, an i.v. for venous access and take blood for FBC and septic work-up. Pulse oximetry is commenced to measure and monitor SpO_2 and arterial blood gases are sample.

PACT module on COPD and asthma

LEARNING ISSUES
Pulse oximetry
Arterial blood gases for diagnosis and monitoring

The blood pressure is 160/90 mmHg, the heart rate is 105 per minute, the respiratory rate is 36 per minute, and the temperature is 37 °C (100.4 °F). The patient is lethargic, in respiratory distress, diaphoretic, and she is using respiratory accessory muscles. On auscultation you hear bilateral diminished breath sounds, wheezing and a markedly prolonged expiratory phase. The heart sounds are distant, and no cardiac extra-sounds are audible.

You start albuterol by nebuliser and initiate ECG (and BP) monitoring and request a chest X-ray.

> **NOTE** Chest X-ray is a first-line exam for patients in acute respiratory failure.

The chest radiograph shows hyperinflated lungs, without infiltrates, effusion, or cardiomegaly. The initial arterial blood gas (ABG) obtained on room air reveals a PO_2 of 5.3 kPa (40 mmHg), a PCO_2 of 10.6 kPa (80 mmHg), and a pH of 7.20. Pulse oximetry on room air is SpO_2 89%.

> Q. What further two drug therapies might you start now?
>
> A. Intravenous steroid e.g. methylprednisolone 60 mg for the bronchospasm and i.v. antimicrobials (after cultures taken) for the evident clinical infective exacerbation of COPD.

LEARNING ISSUES
Causes of diminished breath sounds in wheezing patients
Hypoxaemia
Respiratory acidosis

PACT module on Electrolytes and Homeostasis

You inform a critical care colleague about the patient's marginal condition, and ask the nurse who is in continuous attendance, to increase the oxygen therapy cautiously to O_2 28% via a Venturi mask. You are asked to attend another patient and when you return, after 25 minutes, the patient looks worse. Repeat ABG shows a PO_2 of 6 kPa (45 mmHg), a PCO_2 of 12 kPa (90 mmHg), and a pH of 7.08.

LEARNING ISSUES
ABG monitoring of acute respiratory failure

Q. Clearly the Oxygen 28% was not an adequate response to the patient's hypoxaemia. How should you react now – give more O_2?

A. The oxygen therapy was inadequate for the hypoxaemia but giving more oxygen runs the risk of further impairing the respiratory drive.

Q. Would you start non-invasive positive pressure ventilation (NIPPV) or intubate and ventilate the patient?

A. The patient requires ventilation. Given the reduced level of consciousness, it is probably too late for a trial of NIPPV. The patient should be intubated and ventilated.

LEARNING ISSUES
Indications for NIPPV

PACT module on Acute respiratory failure
PACT module on Mechanical ventilation

> **NOTE** This patient is unstable and in distress with respiratory depression causing acute on chronic respiratory acidosis. If supplemental O_2 is begun it is important to administer low concentration initially to avoid a further rise in CO_2.

Q. Would you initiate mechanical ventilation in this patient in the ED or would you wait until arrival in the ICU? Give arguments.

A. The patient should be intubated and ventilated expeditiously i.e. in the ED, prior to transfer to the ICU.

She is in acute respiratory distress which is exacerbating her chronic respiratory insufficiency. The patient is using respiratory accessory muscles and she is at high risk of respiratory arrest. The work of breathing (WOB) is elevated and this significantly increases the total body oxygen consumption. The oxygen therapy (which she needs for hypoxaemia) has exacerbated her hypercarbia and as a consequence, this patient needs urgent respiratory support.

LEARNING ISSUES
Transport of a ventilated patient

PACT module on Patient transportation

The patient is intubated in the ED using propofol and fentanyl, but she is not paralysed (pharmacologically) during the transport. She is placed on volume-controlled ventilation (VCV) with square inspiratory waveform at a rate of 20 per minute with a Vt of 840 mL (12 mL/kg) and an I:E ratio of 1:1.5. The BP drops to 80/45 and the HR rises to 120/min.

An ABG sample on FiO_2 0.6 shows a PaO_2 of 29.3 kPa (220 mmHg), a PCO_2 4 kPa (30 mmHg), and a pH of 7.6.

LEARNING ISSUES
ABG monitoring of ventilatory settings
I:E ratio

PACT module on Mechanical ventilation

> Q. The above settings have shortened her expiration time. What is the effect of this in this patient?
>
> A. The effect is to increase intrinsic PEEP (auto-PEEP), with likely air-trapping and a marked adverse heart-lung interactive effect.

The blood pressure fell because of the combination of positive pressure ventilation, PEEP, PEEPi, and the likely volume contracted status of this patient. Remember that elderly patients found at home may not have eaten and drunk enough in the period before the acute admission. The increase in the HR is a physiologic compensation to maintain a normal cardiac output in the face of volume contraction.

LEARNING ISSUES
Auto-PEEP

> Q. What is the best approach to ventilatory settings for this patient?
>
> A. Set rate (and tidal volume) to yield appropriate minute ventilation (target the pH, not the $PaCO_2$).

The Vt is reduced to 8 mL/kg ideal body weight and plateau pressure limited to below 30 mmHg.

> Q. Which is likely to be most efficacious in this situation, reducing Vt or reducing the ventilatory rate?
>
> A. Improvements are gained more by reducing frequency than reducing tidal volume.

Obstructive respiratory diseases require adequate expiratory time – beware of auto-PEEP. If auto-PEEP occurs during mechanical ventilation, the amount of time given over to expiration needs to be lengthened, either by reducing the respiratory rate or the inspiratory time, or both.

PaCO$_2$ is kept at the patient's baseline level, which may be higher than normal in COPD patients. If the patient's baseline PaCO$_2$ is unknown then ventilate to a normal pH. A pH of 7.18 suggests an acute on chronic respiratory failure, and it reflects an acutely increased CO$_2$ level.

LEARNING ISSUES
Patient–ventilator interaction
ABGs, particularly pH, to monitor ventilation in CO$_2$ retaining patients

PACT module on Electrolytes and Homeostasis
Ventilation modes – PACT module on Mechanical ventilation

> **NOTE** A 0.13 kPa (10 mmHg) increase in CO$_2$ corresponds to a decrease in pH of 0.8. Normal pH for COPD is assumed to be 7.36.

> Q. Does the patient need sedation? Does she need muscle relaxants?
>
> A. Sedation is needed to reduce the work of breathing (WOB) and to allow the best ventilation for this patient. Muscle relaxant drugs, however, should be avoided if possible. When necessary, they should be given by bolus pending resolution of the acute patient–ventilator dyssynchrony problem and not by continuous infusion.

After two days the sputum culture from your initial septic work-up is reported positive for *Hemophilus influenzae.* You retain the amoxicillin/clavulanate (as an appropriate antibiotic pending knowledge of the antibiogram) which you started in the emergency department for a total of seven days i.v. therapy.

> **NOTE** Features of bacterial pneumonia: clinical and radiological signs (infiltrates on the chest X-ray), sputum purulence, deterioration in gas exchange, increase in white cell count and fever.

The morning after her ICU admission, the patient is stable and you decide to stop continuous sedation and to give only boluses of sedation if needed.

After one week the patient is awake and haemodynamically stable. Renal function is normal and enteral nutrition has been established during the last four days. Homeostasis is restored and the patient has a normal pH and the antibiotic is stopped. The patient is alert, has no fever but she is still ventilated, on low level pressure support and achieving satisfactory gas exchange on 30% oxygen. After a successful T-tube weaning trial of spontaneous breathing, the patient is extubated and she is transferred to the medical ward on day eight.

LEARNING ISSUES
Modes of weaning
Criteria for successful weaning

A 62-year-old, 75 kg man with a previous history of emphysema, coronary artery disease and congestive heart failure, is admitted to the emergency department, with a history of increasing shortness of breath, cough productive of yellow sputum and fever to 39 °C. The patient had no history of COPD; however he reported to have been smoking ten cigarettes per day for 15 years.

Oxygen therapy and pulse oximetry monitoring is started

He has reduced vesicular sounds and crackles in the right lobe (mainly at the base). His respiratory rate is 38 breaths/minute, heart rate 130/min, blood pressure 160/100 and SpO_2 is 60% despite a high FiO_2 via a non-rebreathing mask. He is breathing using his accessory muscles and is becoming obtunded. You decide to intubate him in the emergency department because of refractory hypoxaemia, increasing work of breathing and clinical exhaustion. The patient is then admitted to the ICU.
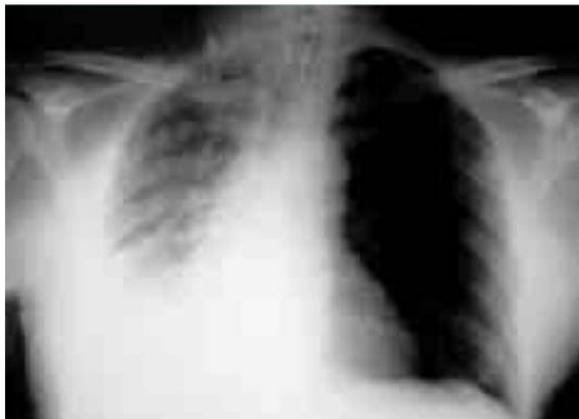
LEARNING ISSUES
Clinical indication for intubation
Community-acquired pneumonia (see PACT module on Severe infection)
Interpretation of SpO_2 measurements
Work of breathing (WOB)

The antero-posterior chest radiograph after intubation (below) reveals dense consolidation of the right middle and lower lobes.

The patient is ventilated for 24 hours, using 10 cmH$_2$O PEEP and 60% inspired oxygen to maintain a PaO_2 greater than 8 kPa (60 mmHg). Blood, sputum and urine are sampled and sent for culture. The patient is treated empirically with intravenous cefuroxime and erythromycin. Sputum cultures later grew *Streptococcus pneumoniae* and the cefuroxime and erythromycin were stopped and intravenous penicillin was given for another five days.

LEARNING ISSUES
Investigations

Q. Which bacteria are the most likely cause of pneumonia in this patient?

A. *Streptococcus pneumoniae, Hemophilus influenzae* and Mycoplasma are most often the causes of community-acquired pneumonia. *Staphylococcus aureus* classically complicates an influenza pneumonia.

See PACT module on Severe infection

The respiratory parameters improve leading to a gradual reduction of the PEEP to 5 cmH$_2$O and inspired oxygen to 50% yielding a PaO$_2$ of 10 kPa (75 mmHg). The patient remains haemodynamically stable and his fever decreases to a 24-hour maximum of 38 °C.

Q. Do you think there are major clinical contraindications to start weaning this patient? Give arguments.

A. The patient is conscious, stable from a haemodynamic and respiratory viewpoint, he is not receiving either vasopressors or inotropes, so there are no clinically apparent contraindications to starting a weaning trial.

His respiratory rate: tidal volume ratio in 1 minute of unassisted breathing is 60/breaths/minute per litre.

Q. His rapid shallow breathing index (RSBI) appears to be correspondingly satisfactory for a trial of weaning. What value of RSBI is predictive of a successful spontaneous breathing trial?

A. Less than 105.

LEARNING ISSUES
Weaning criteria
Rapid shallow breathing index

PACT module on Mechanical ventilation

The patient is placed on a T-piece trial. After only 15 minutes he becomes anxious, diaphoretic, tachycardic to 130/min and tachypnoeic to >40 breaths/min.

Q. What would you do?

A. Stop the weaning trial and assess for reversible causes of increased respiratory muscle load and reduced strength.

The patient's airway resistance is 20 cmH$_2$O/L per second, so aerosolised β-agonists are given leading to resistances of 18 cmH$_2$O/L per second after treatment. No clinical improvements are achieved with this treatment (figure).
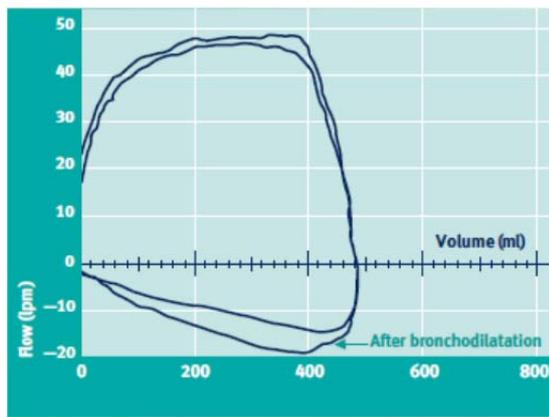
Interpreting flow-volume measurements

Respiratory compliance is 50 mL/cmH$_2$O, and spontaneous minute volume is 9.5 L/min. His white blood cell count is steadily decreasing. He has no fever and his pre-albumin is 12 mg/dL. He is receiving more than 30 kcal/kg/day and the carbohydrate intake is reduced to limit CO$_2$ production and to facilitate weaning. His serum electrolytes and thyroid functions are measured and found to be normal. He is considered for daily T-piece weaning trial.

Q. How long do you do a T-piece weaning trial?

A. Thirty minutes is a reliable period of time.

Useful duration of T-piece trial

For two days the patient fails daily T-piece trials due to tachycardia, hypertension, and subjective distress. You decide to perform T-piece trials with continuous ST-segment monitoring. The ECG reveals new ST depression in leads 1 and V3 to V6 just after the start of the weaning trial. The ECG changes reverse when the patient is put back on volume-controlled ventilation.

Continuous ECG trace ST-segment monitoring

PACT module on Haemodynamic monitoring

Q. What can be the cause of ECG changes during weaning trials?

A. This patient appears to have coronary artery disease which precipitates ischaemia during a weaning trial.

> Q. What is the mechanism of acute ischaemia during weaning?
>
> A. In general, when patients begin to breathe spontaneously, intrathoracic pressure becomes negative. The consequence is that venous return and the left ventricular afterload increase. Increased afterload is associated with increased ventricular wall tension and increased oxygen consumption, which may precipitate ischaemia where oxygen delivery is fixed as a result of coronary artery disease.

See PACT module on Acute myocardial ischaemia

> Q. What can you do to improve his heart function?
>
> A. If you recognise the cardiac origin of the failure, the use of vasodilators and/or diuretics may result in successful weaning.

Using topical nitrates the next T-piece trial does not yield any improvement. Finally, a nitroglycerin drip is started and titrated during the weaning trial to maintain the patient's systolic blood pressure below 130 mmHg, and to reduce all other symptoms, including anxiety. The patient remained in a 'comfort' zone for a one-hour T-piece trial receiving 100 mcg/min of nitroglycerin.

The patient was successfully extubated, and the nitroglycerin drip was stopped after one day and beta blockade was started pending coronary angiography which showed a 45% stenosis of the left anterior descending coronary artery. He was discharged from the ICU after three days on ongoing oral therapy. He was scheduled for an elective angioplasty three days later and he went home one week thereafter.

On reflection, the clinical course of the first patient demonstrates some of the common pitfalls in the acute management of COPD patients. A potential error at the time of intubation is the selection of overestimated ventilatory parameters. In this instance, the Vt and respiratory rate (Fr) chosen resulted in haemodynamic instability and severe alkalaemia. Blood gas monitoring helped in the recognition and correction of the difficulty.

A conscious attempt to wean the patient from the ventilator might have been started earlier than seven days. Initial ventilator dependence is typical of COPD patients and it is often due to unrecognised (and therefore untreated) muscular weakness in the face of unresolved bronchospasm (due to PEEPi). Respiratory monitoring of ventilatory parameters and ABGs might have been used to more actively guide weaning and extubation.

The clinical course of the second patient demonstrates some of the common difficulties in weaning complex patients. Adverse general factors are sometimes under-treated when compared to respiratory factors. The failure of weaning from mechanical ventilation is strongly related to cardiac function and to general homeostasis. Clinical factors and laboratory results need to be assessed before and after starting a spontaneous breathing trial and the correction of non-respiratory problems, particularly myocardial ischaemia and acute heart failure may be key to achieving successful weaning/extubation.