Lung function tests for the general physician

Lung function tests are commonly ordered, but often poorly understood by junior doctors. Indications for lung function tests (Miller et al, 2005) can be grouped into:

- Evaluating symptoms (dyspnoea, chest pain, cough)
- Evaluating signs (kyphoscoliosis, chest wall deformity, hyperinflation)
- Evaluating abnormal tests (chest X-ray, arterial blood gas, polycythaemia)
- Measuring the effect of disease on lung function
- Screening high risk groups (smokers, industrial exposure)
- Performing some routine examinations (preoperative assessment, suitability for high intensity exercise programmes).

Lung function tests can be broken down into spirometry, lung volumes and diffusion studies: each area is covered in more detail below.

Spirometry

Spirometry is the bread and butter of lung function tests, the basis of which will be a gentle reminder from medical school. It is relatively straightforward to understand how the forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) are measured.

Forced expiratory volume in 1 second

FEV_1 measures the power of the patient's lungs, by measuring the expulsion of air forced out in 1 second. Compared to peak flow, it is taken over a longer time period and is generally less dependent on effort. It is reduced in both obstructive and restrictive deficits. FEV_1 relies on age, height and population means to derive significance on its own, but it is a good marker of disease progression in patients with chronic obstructive pulmonary disease, and can be quickly and easily measured in a clinic setting. It is converted into a percentage of predicted FEV_1 which relies on population averages.

Forced vital capacity

FVC gives an indication of the lung volumes by measuring the amount of air that can be blown out completely. As explained by Chapman et al (2014), FVC will be reduced in restrictive lung disease (kyphoscoliosis, neuromuscular weakness) where the lungs are either stiff, inspiratory muscles are weak or the airways are narrowed so the small airways collapse during expiration. It becomes a less reliable measure of lung volumes in concurrent obstructive deficits where there is hyperinflation and air trapping leading to falsely decreased FVC, known as pseudorestriction.

FEV_1:FVC ratio

This is calculated using the absolute values of both the above rather than the percentage. In an obstructive deficit, the FEV_1 is reduced but the FVC remains normal, leading to a reduced FEV_1:FVC ratio. In a restrictive deficit, the FEV_1 and the FVC are both reduced, leading to a normal or even elevated FEV_1:FVC ratio. A FEV_1:FVC ratio of less than 70–75% generally indicates an airflow obstruction. The ratio is barely affected by gender, height, age and ethnicity as the individual measures are, so it provides a more robust measure of lung function. *Table 1* outlines causes of obstructive and restrictive deficits. Putting this into practice, the GOLD (Global Initiative for Chronic Obstructive Lung Disease) criteria (2016) place great emphasis on the FEV_1:FVC ratio in classifying the severity of chronic obstructive pulmonary disease as seen in *Table 2*.

Flow–volume loops

The flow–volume loop is a plot of volume (x axis) against inspiratory and expiratory flow (y axis). A normal flow–volume loop shows a sharp rise to peak flow followed by a relatively steep decline in the expiratory segment of the graph, with a gentle curve underneath completing the inspiratory segment as seen in *Figure 1a*.

Table 1. Common causes of obstructive and restrictive deficits

Obstructive	Chronic obstructive pulmonary disease
	Asthma
	Cystic fibrosis
	Bronchiectasis
Restrictive	Kyphoscoliosis
	Neuromuscular disorders
	Pulmonary fibrosis (including idiopathic, autoimmune, environmental and medication related)
	Hypersensitivity pneumonitis
	Obesity
	Alveolar haemorrhage
	Large pleural effusions

Dr Jessica Gates, Core Medical Trainee, Department of Acute Medicine, The Royal London Hospital, London E1 1BB

Dr Rajesh Banka, Consultant Respiratory Physician, Department of Respiratory Medicine, King George Hospital, Ilford, London

Correspondence to: Dr J Gates (jessica.gates3@nhs.net)

Table 2. GOLD criteria for classification of severity of chronic obstructive pulmonary disease

Classification	Spirometry	Symptoms
I: Mild chronic obstructive pulmonary disease	■ FEV_1:FVC <0.7 ■ FEV_1 ≥80% predicted	At this stage, the patient may not be aware that his/her lung function is abnormal
II: Moderate chronic obstructive pulmonary disease	■ FEV_1:FVC <0.7 ■ 50% ≤FEV_1 < 79% predicted	Symptoms usually progress at this stage, with shortness of breath typically developing on exertion
III: Severe chronic obstructive pulmonary disease	■ FEV_1:FVC <0.7 ■ 30% ≤FEV_1 < 49% predicted	Shortness of breath typically worsens at this stage and often limits the patient's daily activities. Exacerbations are especially seen beginning at this stage
IV: Very severe chronic obstructive pulmonary disease	■ FEV_1:FVC <0.7 ■ FEV_1 <30% predicted or FEV_1 <50% predicted plus chronic respiratory failure	At this stage, quality of life is very appreciably impaired and exacerbations may be life-threatening

From the Global Initiative for Chronic Obstructive Lung Disease (GOLD) (2016). FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity.

Obstructive deficits as seen in chronic obstructive pulmonary disease show an inability to rise as far as the normal curve, in keeping with the reduced FEV_1, followed by a slow exhalation out which gradually tails off (*Figure 1b*).

Restrictive deficits (*Figure 1c*) show a quick rise to peak flow, similar to the normal curve, but the patient soon runs out of breath and the curve sharply declines down before inhalation begins again.

The shape of the flow–volume loop can identify the likely location of the airflow obstruction in a number of anatomical disorders (*Figure 2*). In a case of variable extrathoracic obstruction, the large airways become obstructed during the inspiration loop. This is seen predominantly in cases of vocal cord paralysis, large goitre and laryngeal tumours (*Figure 2a*).

In cases of variable intrathoracic obstruction the opposite is true, with the obstruction being pushed away from the large airways in inspiration, but being sucked into the large airways on expiration, resulting in a flattening of the expiratory loop. This is typically seen in intrathoracic tumours close to the trachea (*Figure 2b*).

In fixed upper airway obstruction, as seen in tracheal stenosis or a circumferential tracheal tumour, the flow–volume loops are flattened in both inspiration and expiration (*Figure 2c*).

Further explanation of flow–volume loops can be found in Sewa and Ong (2014) and Chapman et al (2014).

Figure 1. **a.** Normal flow–volume loop. **b.** Flow–volume loop in severe obstructive deficit. **c.** Flow–volume loop showing restrictive deficit. X axis represents inspiratory and expiratory flow and Y axis represents volume. The top curve represents inspiration and the bottom represents expiration.

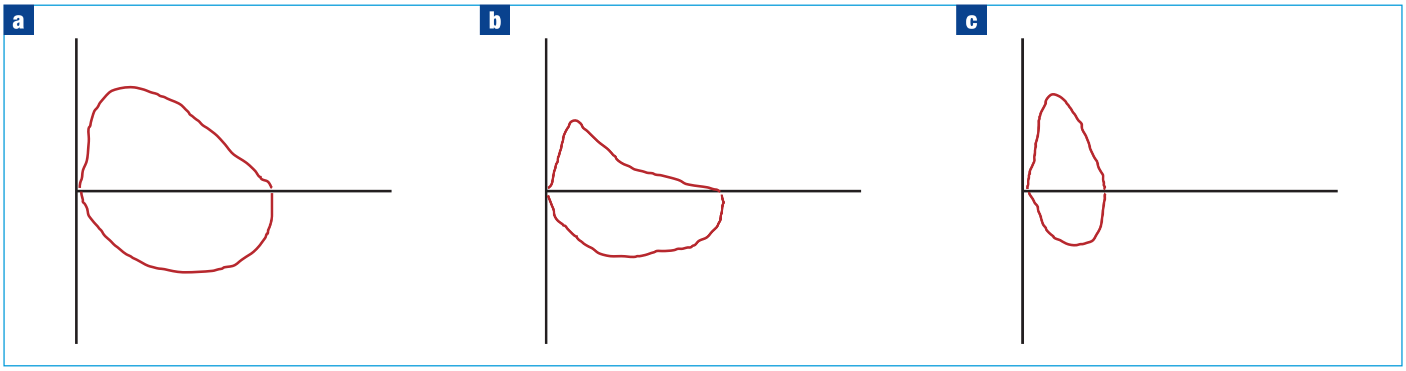
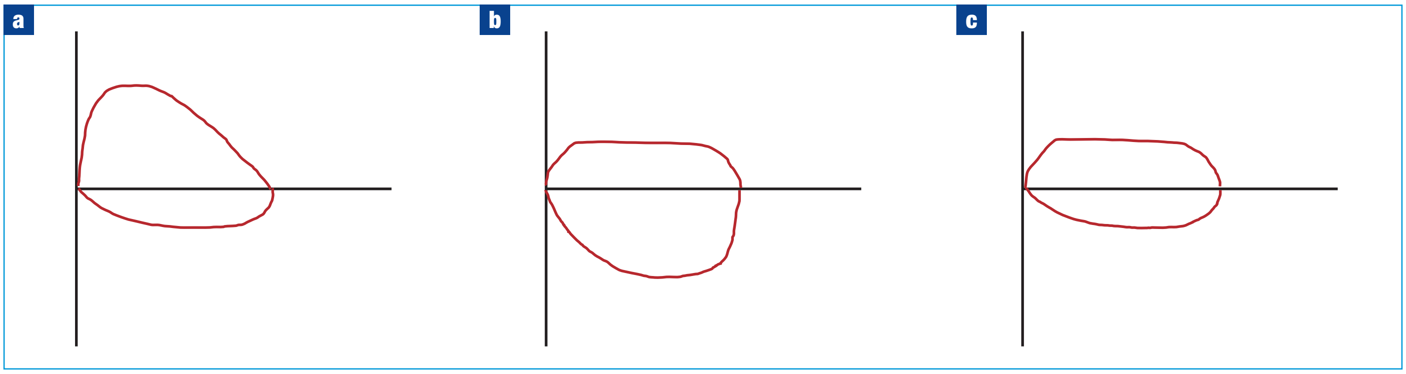


Figure 2. Flow–volume loops in anatomical disorders. **a.** Variable extrathoracic obstruction. **b.** Variable intrathoracic obstruction. **c.** Fixed upper airway obstruction. X axis represents inspiratory and expiratory flow and Y axis represents volume. The top curve represents inspiration and the bottom represents expiration.



KEY POINTS

- An obstructive deficit is detected by a reduced forced expiratory volume in 1 second (FEV_1) and a reduced FEV_1: forced vital capacity (FVC) ratio, whereas a restrictive deficit is detected by a reduced FVC and a normal or elevated FEV_1:FVC ratio.
- The GOLD (Global Initiative for Chronic Obstructive Lung Disease) framework sets out the prognostic criteria for mild, moderate, severe and very severe chronic obstructive pulmonary disease as based on the patient's spirometry results.
- Flow–volume loops are a clear diagrammatic way of showing the extent and the pattern of respiratory deficits, and may give a clue to the cause.
- Diffusion studies aim to describe the gas exchange potential in the lungs in a specific situation, and can be corrected for lung volume.
- Causes of a lower total lung carbon monoxide include diffuse alveolar damage, pulmonary hypertension, reduction in the number of red blood cells per ml of blood and airflow obstruction.
- Causes of a raised total lung carbon monoxide include increased pulmonary blood flow, incomplete alveolar expansion and pulmonary haemorrhage.
- Cardiopulmonary exercise testing is becoming more widely used and can be considered in the investigation of unexplained exertional dyspnoea in centres where this is available.

Diffusion studies

In contrast to the above results, diffusion studies are poorly understood and therefore more difficult to assign meaning, but once understood they can be instrumental in diagnosis and assessment.

Diffusion studies describe how well oxygen is able to move from the alveoli to the blood. They gives clinicians an assessment of the integrity of the quality of gas exchange in the lungs: this would be expected to be reduced in conditions causing alveolar or microvascular destruction, e.g. emphysema. The quality of gas exchange can be quantified and measured by inhaling a mixture of carbon monoxide and helium. The carbon monoxide crosses the alveolar membrane and allows calculation of the total lung carbon monoxide by measuring the difference between the inhaled and exhaled carbon monoxide concentrations. This result is then adjusted for haemoglobin levels as explained later.

Results may be given as the total gas transfer (total lung carbon monoxide) or corrected for lung volume (gas transfer per unit lung volume).

Gas transfer per unit lung volume gives an indication of the gas exchange efficiency, whereas total lung carbon monoxide is the gas exchange potential in a specific situation.

Interpreting the total lung carbon monoxide and gas transfer per unit lung volume is best done by understanding the reasons for the abnormal result (Hughes and Pride, 2012; Shiner and Steier, 2013; Chapman et al, 2014).

- Total lung carbon monoxide and gas transfer per unit lung volume will be raised in increased pulmonary blood flow, as is seen in asthmatics, hyperkinetic states and on exercise. For each 100% rise in cardiac output on exercise, the total lung carbon monoxide and gas transfer per unit lung volume rise by 20%
- Polycythaemia will raise the total lung carbon monoxide and gas transfer per unit lung volume with an increased number of red blood cells per ml of blood, and therefore carbon monoxide reacts more readily with haemoglobin. The opposite is true for the reverse state of anaemia which leads to a lower total lung carbon monoxide and gas transfer per unit lung volume
- Pulmonary haemorrhage, as may be seen in systemic lupus erythematosus, granulomatosis with polyangiitis, or Goodpasture's syndrome, will falsely raise the total lung carbon monoxide and gas transfer per unit lung volume for a few days as the carbon monoxide reacts with the red blood cells in the alveolar spaces.
- Total lung carbon monoxide and gas transfer per unit lung volume will be low in conditions which are associated with pulmonary hypertension, e.g. pulmonary oedema and pulmonary emboli, where there is no ventilation problem but there is reduced alveolar perfusion.
- Emphysema is the state that will see the lowest total lung carbon monoxide and gas transfer per unit lung volume as a result of airway destruction and obstruction. To a lesser extent the same causative issues are seen in acute pneumonia.
- Total lung carbon monoxide and gas transfer per unit lung volume may be reduced in pulmonary fibrosis as the alveolar wall membrane may be thick enough to reduce carbon monoxide transfer.

Some conditions may cause an increased gas transfer per unit lung volume with a normal or reduced total lung carbon monoxide:

- Pneumonectomy or lobectomy (loss of units)
- Scoliosis, kyphosis, neuromuscular weakness, ankylosing spondylitis (incomplete alveolar expansion).

Smoking 2–3 cigarettes in the 2 hours before diffusion studies will reduce the end result by approximately 5% (Hughes and Pride, 2012).

Cardiopulmonary exercise testing

Cardiopulmonary exercise testing has existed as an enhanced exercise test for many years now, but has not been widely used until recently, when its application as an investigation for unexplained dyspnoea has seen it gain popularity among some respiratory physicians.

Traditional investigations for assessing dyspnoea such as spirometry, echocardiography and cross-sectional imaging are criticized for trying to assess a condition which occurs during exercise, yet is measured while the patient is at rest (Albouaini et al, 2007).

The aim of cardiopulmonary exercise testing is to measure and calculate the physiological response of the lungs, heart and muscles to exercise. As an evaluation of dyspnoea it can be helpful in detecting exertional desaturation, exertional bronchoconstriction and also give helpful information regarding the underlying organ dysfunction, whether that is primarily lung, cardiac or muscular (Datta et al, 2015).

Cardiopulmonary exercise testing has been used by anaesthetists for several years to assess the preoperative suitability for high risk patients undergoing anaesthesia and can be helpful to prognosticate and decide about postoperative care.

The equipment for cardiopulmonary exercise testing is extensive, and it is by no means a first-line recommendation or widely accepted by the respiratory community. However, it is a useful option to consider in a patient with refractory exertional dyspnoea with no clear cause and can be offered at tertiary dyspnoea clinics.

Conclusions

This article has explained some of the concepts of lung function testing and briefly explored the potential future of this field. For further reading the authors recommend Shiner and Steier's (2013) *Lung Function Tests Made Easy*. The best way to gain confidence in interpreting lung function results is to go and practice by interpreting the results seen in patients every day. **BJHM**

Conflict of interest: none.

Albouaini K, Egred M, Alahmar A, Wright DJ. Cardiopulmonary exercise testing and its application. Postgrad Med J. 2007 Nov 01;83(985):675–682. https://doi.org/10.1136/hrt.2007.121558

Chapman S, Robinson G, Stradling J, West S, Wrightson J. 2014. Oxford Handbook of Respiratory Medicine. 3rd edn. Oxford: Oxford University Press:867–880.

Datta D, Normandin E, ZuWallack R. Cardiopulmonary exercise testing in the assessment of exertional dyspnea. Ann Thorac Med. 2015;10(2):77–86. https://doi.org/10.4103/1817-1737.151438

Global Initiative for Chronic Obstructive Lung Disease (GOLD). 2016. Spirometry for Healthcare Providers. (accessed 1 January 2018) http://goldcopd.org/wp-content/uploads/2016/04/GOLD_Spirometry_2010.pdf

Hughes JMB, Pride NB. Examination of the carbon monoxide diffusing capacity (DL(CO)) in relation to its KCO and VA components. Am J Respir Crit Care Med. 2012 Jul 15;186(2):132–139. https://doi.org/10.1164/rccm.201112-2160CI

Miller MR, Hankinson J, Brusasco V et al. Standardisation of spirometry. Eur Respir J. 2005 26:319–338. https://doi.org/10.1183/09031936.05.00034805

Sewa DW, Ong TH. Pulmonary Function Test: spirometry. Proceedings of Singapore Healthcare. 2014 Mar;23(1):57–64. https://doi.org/10.1177/201010581402300110

Shiner R, Steier J. 2013. Lung Function Tests Made Easy. Oxford: Churchill Livingstone

TOP TIPS

- Advise patients to not smoke for at least 2 hours before lung function testing, and not to use any bronchodilator that day.
- The best way to get a useful result is to check the patient's technique.
- Work out whether the spirometry is normal, or shows an obstructive or a restrictive deficit.
- Some patients may have normal or borderline spirometry, and then have deranged diffusion studies. For example, patients who have chronic thromboembolic disease can have normal spirometry and low total gas transfer.