*Pulmonary function tests (PFTs)

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Pulmonary function tests (PFTs) include the measurement of velocity and volume of expiratory flow (**spirometry**), static volumes **(lung volumes) ,** diffusion of gases across the alveolar-capillary membrane **(diffusion Capacity DLco)**, and respiratory muscle strength.

PFTs can (1) identify patterns and quantify the severity of a variety of respiratory system diseases. the indications for performing PFTs may include (2) diagnostic evaluation of symptoms, (3) monitoring of disease stability or progression, (4) assessing acute or long-term response to treatment, or (5) providing preoperative pulmonary assessment.

1- Spirometry is the determination of expiratory volume and flow rates.

the primary spirometric values include:

the forced expiratory volume in 1 second (**FEV_1**; the quantity of air in liters exhaled in 1 second),

forced vital capacity (**FVC**; the total quantity of air exhaled in a maximum voluntary exhalation),

peak expiratory low rate (**PEFR**; the maximum velocity of air during a forced exhalation in liters/second).

the **ratio of FEV_1/FVC** differentiates obstructive (reduced ratio) and restrictive ventilatory (preserved or elevated ratio) deficits.

Spirometric measurements are effort dependent.

To determine FEV1 and FVC, a subject is instructed to inhale maximally to total lung capacity (TLC) followed by a vigorous, maximal forced expiratory effort to residual volume (RV).

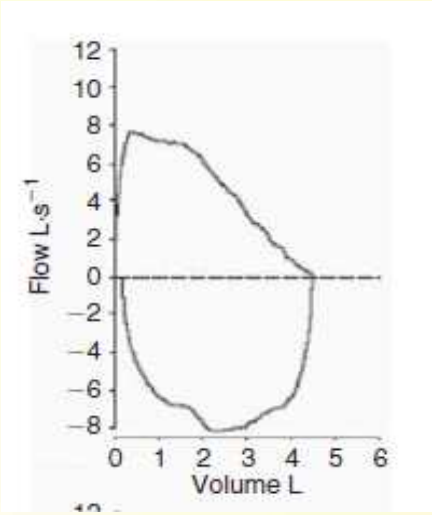
the subject exhales into a mouthpiece attached to a flow meter through which expiratory flow rates are measured, and volume is integrated from the flow signal

Spirometry **results** are reported as both absolute values (i.e., FEV1 = 2.5 L) as well as a percentage of what values would be predicted given certain individual characteristics (i.e., FEV1 = 90% predicted).

Predicted values for a given individual are derived from the person's height, race/ethnicity, age, and gender. Predictive equations have been derived from cross-sectional population studies.

the percent predicted values for FEV1 and FVC can also be used to **grade disease severity**. Deficits are typically described as mild, moderate, moderately severe, severe, or very severe depending on the degree of deviation from the predicted values

In addition to being characterized numerically, the measurements obtained while testing spirometry (e.g., FVC, PEFR) are typically displayed as a **flow-volume loop**. The flow-volume loop displays the expiratory effort with volume (in liters) on the *x* axis and low (in liters per second) on the *y* axis.



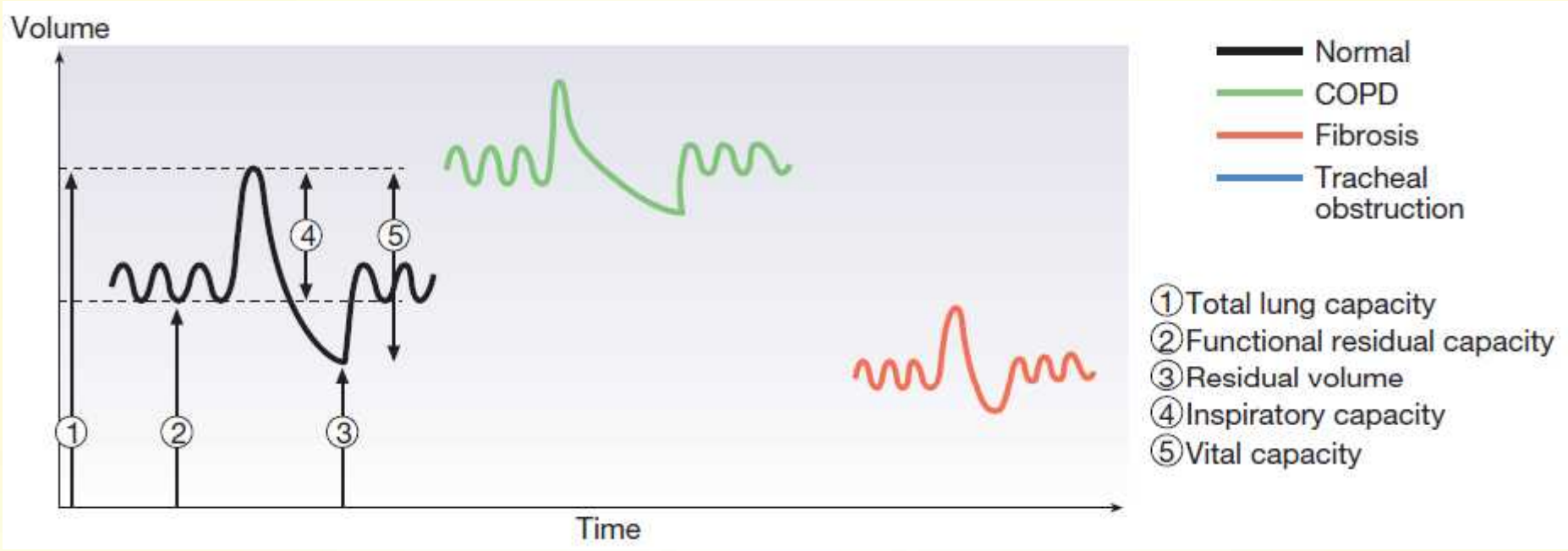
2- Lung Volumes: the lung volumes of most clinical interest include the **total lung capacity** (TLC, which is the volume of air in the lungs after maximal inhalation);
Residual volume (RV, which is the volume of air remaining in the lungs after maximal exhalation);
and **functional residual capacity** (FRC; the volume of air in the lung after a normal tidal volume breath is exhaled).

Lung volumes are typically measured by one of three techniques: helium dilution, nitrogen washout, or body Plethysmography

As with spirometry , lung volumes are reported as both measured values and as a percent predicted value.

Predicted values are generated from large cross-sectional studies.

Determinants of predicted lung volumes include height, gender, race/ethnicity, and age.


Volume
Time
①
②
③
④
⑤
Normal
COPD
Fibrosis
Tracheal obstruction
①Total lung capacity
②Functional residual capacity
③Residual volume
④Inspiratory capacity
⑤Vital capacity

3- Diffusion Capacity for Carbon Monoxide:

the diffusing capacity for carbon monoxide (DLco) is a test that quantifies the diffusing properties of the alveolar-capillary membrane.

the DLco provides information about the **efficiency of gas exchange** at the level of the alveolus and capillary.

Carbon monoxide is used as a surrogate , since its diffusion rate is similar to oxygen .

A low concentration of carbon monoxide is inhaled and the rate of absorption calculated.

the difference between the amount of CO inhaled and the amount present in the exhaled gas represents the uptake of CO across the alveolar-capillary membrane.

Obstructive Ventilatory Deficits

Obstructive ventilatory deficits are defined by a reduction of expiratory flow as measured by FEV1 or PEFR.

Pathophysiologically, obstructive ventilatory deficits are caused by increased airways resistance.

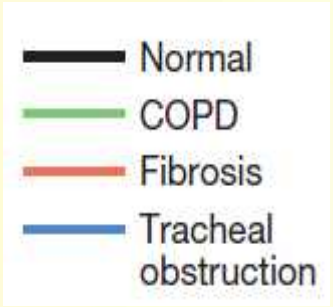
Increased airways resistance results in impaired expiratory air flow.

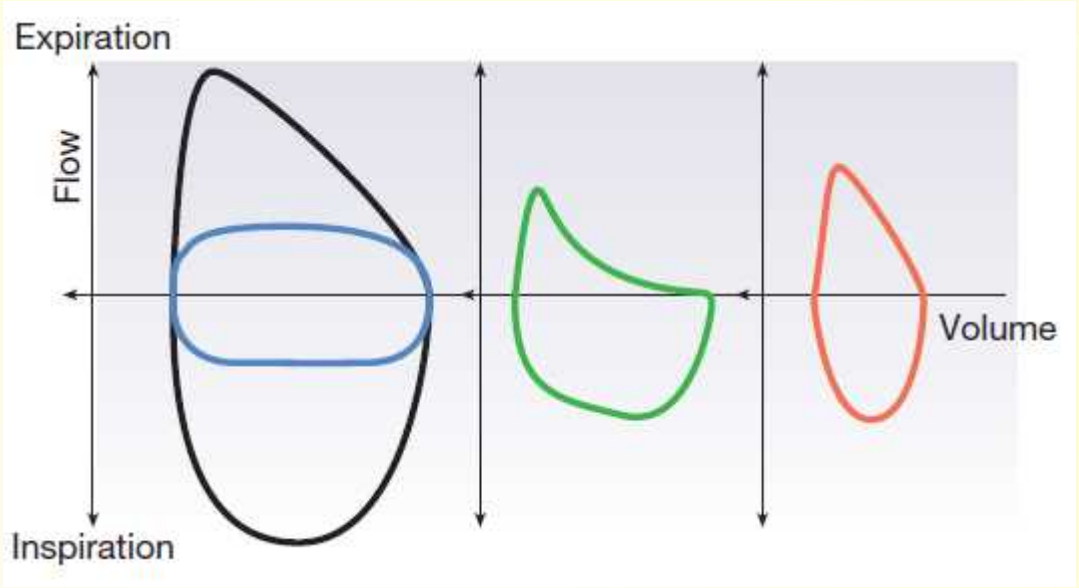
here are a variety of pathologic processes that can cause obstructive ventilatory deficits, ranging from reversible air flow obstruction (i.e., asthma) to permanent changes in the lung parenchyma and airways (i.e., emphysema).

Regardless of the underlying process, the primary manifestation of an obstructive ventilatory deficit is a decreased FEV1 as measured by spirometry.

Typically, FVC is relatively preserved in obstructive ventilatory diseases compared with the decrease in FEV1. he relative preservation of FVC occurs because the volume of air in the lungs is not decreased, and, given adequate time, a patient can exhale a relatively larger quantity of air during a complete exhalatory effort (delay) than is possible in 1 second of exhalation.

In the setting of a decreased FEV1 and a relatively preserved FVC, the FEV1/FVC ratio is decreased. this constellation of findings (decreased FEV1 as compared with FVC and decreased FEV1/FVC) is the hallmark of obstructive ventilatory deficits.





The classic coved appearance of a low-volume loop in a patient with an obstructive ventilatory deficit is caused by decreased expiratory low rates.

As depicted on a low-volume loop, the overall volume of air exhaled may not be significantly decreased compared with predicted values

Bronchodilator Responsiveness in obstructive ventilatory deficits:

Assessing for bronchodilator responsiveness is commonly done during the evaluation of obstructive ventilatory deficits.

Bronchodilator response can be assessed by administering a short-acting medicine (Inhaled albuterol).

Bronchodilator responsiveness is defined as an increase in the percentage predicted FEV_1 and/or FVC of at least 12% and 0.2 L above baseline

An interval of 15 to 20 minutes between measuring baseline spirometry and postbronchodilator spirometry values allows time for any effect of the medicine to occur.

Restrictive Ventilatory Deficits

Restrictive ventilatory deficits are defined by abnormally reduced lung volumes.

In restrictive diseases, lung volumes are decreased below their expected values.

FEV1 and FVC are reduced proportionately and the FEV1/FVC ratio remains normal or may even increase.

Restrictive ventilatory deicits can be caused by any process that results in decreased lung volumes. Fibrotic changes in the lung parenchyma can manifest as a restrictive deficit as the lung tissue becomes less compliant. the loss of compliance leads to lower lung volumes as a given inspiratory effort results in less inhaled volume. Restrictive deficits can also occur from extrapulmonary processes. Pleural diseases, such as pleural effusions or pleural fibrosis, can limit lung expansion and result in low lung volumes and a restrictive ventilatory pattern. The lungs themselves may have normal elastic properties, but the extrinsic compression from diseased pleura may limit the volume that can be inhaled.

Chest wall pathology can similarly result in a restrictive deficit. Kyphosis and/or scoliosis may result in restrictive physiology because the muscles of respiration are placed at a mechanical disadvantage. With severe curvature of the vertebral column, the diaphragm and accessory muscles may not be able to generate maximal inspiratory forces leading to decreased lung volumes

Chest wall trauma, including broken ribs or scarring (i.e., secondary to burns), may result in decreased mobility of the chest wall, extrinsic compression of the lungs, and a restrictive ventilatory deficit.
Similarly, extreme obesity can cause a restrictive ventilatory deficit by a similar mechanism; extrinsic compression by excess adipose tissue can limit maximal expansion of the respiratory system.

Finally, neuromuscular diseases may cause a restrictive ventilatory deficit.
Weakened or poorly functioning respiratory muscles (whether because of a myopathy and/ or neuropathy) can limit a patient's ability to ventilate, resulting in low lung volumes.

From the previous considerations, specific pathologic processes that may cause a restrictive ventilator deficit include pulmonary fibrosis, sarcoidosis, hypersensitivity pneumonitis, medication/toxic pneumonitis, collagen-vascular diseases (i.e., scleroderma, systemic lupus erythematosus, rheumatoid arthritis), pneumothorax, pleural effusion, pleural fibrosis, severe morbid obesity, scoliosis, kyphosis, chest wall trauma or scarring, and ankylosing spondylitis. Possible neuromuscular causes of restrictive ventilatory deicits include Guillain-Barré, amyotrophic lateral sclerosis, myasthenia gravis, and muscular dystrophies.

Reduced Diffusing Capacity for Carbon Monoxide

Decreased Dlco indicates impaired diffusion of CO across the alveolarcapillary membrane into the bloodstream and is thought to correlate with impaired diffusion of oxygen across the alveolarcapillary membrane.
However, CO uptake is dependent not only on diffusion across the alveolarcapillary membrane but also on binding to Hb.

Therefore the term *diffusing capacity* is inaccurate in that it implies that the transfer of CO from the alveolus to the bloodstream is dependent on diffusion alone.
As such, the term *transfer factor,* rather than Dlco, is used in many countries.

DLco may be increased by a number of processes that result in increased cardiac output through the pulmonary circulation like:

Obesity.

Polycythemic states (more Hb is available for CO binding).

Intraalveolar Hemorrhage.

left-to-right intracardiac shunts.

Decreased Dlco is typically caused by conditions in which the alveolar membrane is abnormal, cardiac output through the pulmonary circulation is decreased, or there is a decrease in Hb available for binding CO.

Decreased DLco may occur with restrictive lung deficits (i.e., pulmonary fibrosis) or obstructive ventilatory deicits (i.e., COPD).
In these conditions, lung parenchymal changes lead to perturbations in pulmonary capillary perfusion, which in turn result in a decrease in the Hb available to bind CO and thereby decrease CO uptake.

Decreased DLco with otherwise normal PFTs is associated with a limited differential diagnosis. Conditions that affect the pulmonary circulation to reduce functional pulmonary capillary circulation may cause a decreased DLco.

Specifically, pulmonary vascular disease (including both pulmonary arterial hypertension and thromboembolic disease) is a possible explanation for a reduced DLco and otherwise normal PFTs.

anemia can also result in a reduced unadjusted Dlco, although the DLco corrected for Hb concentration would be normal.

So when evaluating a PFT scenario , think in the terms of:

- **Expiratory flow (FEV1/FVC).**
- **Lung volume (TLC).**
- **Diffusion capacity (DLCO).**
- **Response to bronchodilators (salbutamol).**

1- look for restrictive disease:

- **Any TLC <80% is by definition , restrictive.**

Also restrictive disease is reflected in proportional decrease in FEV1 and FVC (FEV1/FVC=80% but FVC is <80%).

If restrictive , check the DLCO. This determines if it is "extrathoracic " or "intrathoracic".

If the decrease in DLCO is proportional to the decrease in TLC , it means that the restriction is not due to parenchymal lung disease – it is of extrathoracic origin. So label as extrathoracic and think of obesity and kyphosis.

If the decrease in DLCO is disproportionately low compared to the decrease in TLC , label it intrathoracic and think of interstitial lung disease.

2- look for obstructive disease :

Obstruction is defined by a disproportionately low FEV1 . So both FEV1 and FEV1/FVC are low (<70%) .
Label these as ”obstructive”.

If obstructive , check the TLC , DLCO , and reaction to beta2 agonist
”Emphysema” if the TLC is high but the DLCO is low, minimal to low response to beta2-agonist.

”Asthma” if the DLCO is normal or there is typically reaction to beta2-agonist.

3- combination of obstructive and restrictive (asthma +obesity).

CATEGORIES OF RESPIRATORY DISEASE

Category	Examples
Obstructive lung disease	Asthma
	Chronic obstructive pulmonary disease (COPD)
	Bronchiectasis
	Bronchiolitis
Restrictive pathophysiology—parenchymal disease	Idiopathic pulmonary fibrosis (IPF)
	Asbestosis
	Desquamative interstitial pneumonitis (DIP)
	Sarcoidosis
Restrictive pathophysiology—neuromuscular weakness	Amyotrophic lateral sclerosis (ALS)
	Guillain-Barré syndrome
Restrictive pathophysiology—chest wall/pleural disease	Kyphoscoliosis
	Ankylosing spondylitis
	Chronic pleural effusions
Pulmonary vascular disease	Pulmonary embolism
	Pulmonary arterial hypertension (PAH)
Malignancy	Bronchogenic carcinoma (non-small-cell and small-cell)
	Metastatic disease
Infectious diseases	Pneumonia
	Bronchitis
	Tracheitis