SEMINAR

V | LF Spiro3D

Low and very-low-field 3D magnetic resonance spirometry for advanced regional exploration of respiratory diseases

The Physiologic Basis of Spirometry

Pierantonio Laveneziana – APHP

Service d'Explorations Fonctionnelles de la Respiration, de l'Exercice et de la Dyspnée
Neurophysiologie respiratoire expérimentale et Clinique
Hôpital Pitié-Salpêtrière – Sorbonne Université

Tuesday, 25 June 2024 – 10:30

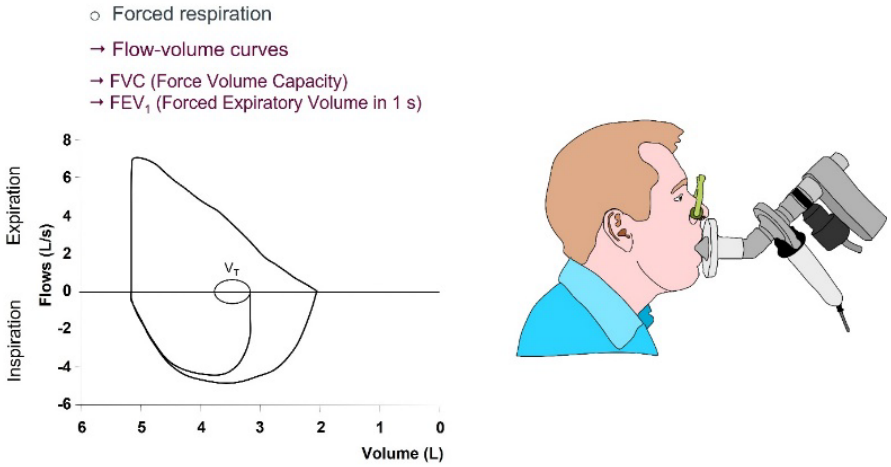
Physical Salle de conferences – UPSaclay
BioMaps – SHFJ – 4 place du general Leclerc, Orsay, France
Digital Conference Room here

Spirometry is one of the most useful and commonly available tests of pulmonary function. It is a physiological test that measures individual inhalation and exhalation volumes of air as a function of time (Figure). The simplicity of spirometry hides the subtleties of the physiologic mechanisms at work, especially the processes that result in expiratory flow limitation, in which the magnitude of the flow leaving the lungs does not increase indefinitely with the expiratory pressure generated by the respiratory muscles. Rather, flow approaches a maximum value that cannot be exceeded regardless of how much extra effort is exerted. The maximum expiratory flow that can be achieved decreases markedly with lung volume and, in fact, is beyond the reach of the respiratory muscles in a healthy subject near total lung capacity. Over about 80% of the vital capacity range, however, the muscles of a healthy individual can generate sufficient pressure to achieve maximal expiratory flow. This maximal flow is influenced by the elastic recoil pressure of the lung and by airway diameter and is decreased in diseases such as asthma and bronchitis. Consequently, the shape and position of the plot of flow (V') versus volume (V) during a maximal expiratory effort is diagnostic of lung disease. The most commonly used parameter derived from the phenomenon of flow limitation is the volume of air expired in the first second of a forced expiration following a maximal inspiration, known as FEV1. Maximal flow decreases with decreasing airway radius, which explains why individuals with narrowed airways (as in asthma or chronic obstructive pulmonary disease) have a reduced FEV1 compared with normal individuals. Maximal flow also decreases as the airway wall becomes less stiff. Consequently, maximal flow decreases with decreasing lung volume as a result of the decreased tension in the alveolar walls that tether the pulmonary airways. FEV1 is thus reduced in subjects with abnormally compliant airways, such as in emphysema. FEV1 is a sensitive variable for the diagnosis of lung disease, but its specificity is limited by the fact that a lower than expected value can indicate either that the airways are not as patent as they should be (obstructive pulmonary disease) or that there was a problem in expanding the lungs properly in the preceding maximal inspiration (restrictive pulmonary disease). When FEV1 is coupled with a measure of the total volume of gas expired during an entire forced expiration, known as forced vital capacity (FVC), one can differentiate between obstructive disease (both FEV1 and the ratio FEV1/FVC are lower than normal) and restrictive disease (FVC is lower than normal but FEV1/FVC is normal).

The benchmark studies that described the physiology of forced expiratory maneuvers used for spirometry were published in the 1950s and 1960s. The flow of a forced expiration can be seen as a consequence of the subject's muscle strength and airway resistance, as is the case with a mechanical

This project has received funding from the European Union's Horizon Europe research and innovation programme under grant agreement N° 101099934. This communication reflects only the author's view and the Commission is not responsible for any use that may be made of the information it contains.

bellows, but that analogy is seriously incomplete because of 2 fundamental structural features of the respiratory system. First, the lungs are elastic, constantly exerting an inward force against the stiffer chest wall and respiratory muscles. Second, the intrathoracic airways are compressible and subject to intrathoracic pressure generated by forced expiration. The result of this is that, under forced expiration, airway resistance is dynamic and changes with lung volume, effort (transpulmonary pressure), and air flow. This airway resistance is not evenly distributed, but occurs at certain points in the airways, because, as air flows through the airways during forced expiration, the intraluminal pressure diminishes with distance from the alveolus, due to the distributed resistance of the airways. At some point the intraluminal pressure equals the pressure surrounding the airway (the equal pressure point [EPP]). Beyond the EPP or the segment upstream (ie, from the alveoli to the EPP) the pressure falls below the surrounding pressure, and the affected airway segment is compressed, limiting flow. Thereafter, increased effort raises both the driving pressure and the airway resistance, and the result is that flow becomes subject only to pulmonary recoil pressure that is itself dependent on lung volume.



This lecture will summarise the complex respiratory physiology of spirometry, which is dependent on the elasticity of the lungs and airway resistance and is determined by the degree of expiratory force exerted. Understanding this complicated physiology is very important in the field of respiratory care and the interpretation of spirometry.

More information: www.v-lf-spiro3d.eu

This project has received funding from the European Union's Horizon Europe research and innovation programme under grant agreement N° 101099934. This communication reflects only the author's view and the Commission is not responsible for any use that may be made of the information it contains.