Chapter 17

Inspiratory Capacity and Exercise Tolerance in Chronic Obstructive Pulmonary Disease

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Introduction

During the last half-century many studies have investigated the correlation of exercise tolerance with routine pulmonary function testing in patients with obstructive pulmonary disease. In virtually all of these studies the degree of airway obstruction was assessed in terms of forced expired volume in 1 s (FEV₁) and forced vital capacity (FVC). Since, only a weak correlation was found between exercise tolerance and degree of airway obstruction, it has been concluded that factors other than lung function impairment (e.g., deconditioning and peripheral muscle dysfunction) play a predominant role in limiting exercise capacity in patients with chronic airway obstruction. Recent work, however, shows that in moderate-to-severe chronic obstructive pulmonary disease (COPD) patients, the inspiratory capacity is a more powerful predictor of exercise tolerance than FEV₁ and FVC, suggesting that the main cause of exercise intolerance in these patients is dynamic pulmonary hyperinflation due to concurrent expiratory flow limitation.

Dynamic Hyperinflation

In normal subjects at rest, the end-expiratory lung volume (functional residual capacity, FRC), corresponds to the relaxation volume (Vr) of the respiratory system, i.e., the lung volume at which the elastic recoil pressure of the respiratory system is zero [1]. Pulmonary hyperinflation is defined as an increase in FRC above the predictable normal value. This may be due to increased Vr, as a result of the loss of recoil (e.g., emphysema) and/or to dynamic pulmonary hyperinflation, which is said to be present when the FRC exceeds Vr. Dynamic hyperinflation exists whenever the duration of expiration is insufficient to allow the lungs to deflate to Vr prior to the next inspiration. This tends to occur under conditions in which expiratory flow is impeded (e.g., increased airway resistance) or when the expiratory time is shortened (e.g., increased breathing frequency). Expiratory flow may also be retarded by other mechanisms, such as persistent contraction of the inspiratory muscles during expiration and expiratory narrowing of the glottal aperture. In COPD patients, dynamic hyperinflation is common and is due to expiratory flow limitation [2, 3].
Expiratory Flow Limitation

The term flow limitation should be used only for describing a condition in which flow cannot augment at a given lung volume. Thus, expiratory flow limitation (FL) simply reflects the incapacity to increase expiratory flow by increasing pleural and, therefore, alveolar pressure at that lung volume.

FL characteristically develops in normal subjects, as well as in patients with respiratory disorders, during a correctly performed maximal forced expiratory maneuver, in which, after the peak expiratory flow, isovolumic expiratory flow rates cannot be increased by increasing expiratory effort and, thus, are maximal. Conversely, FL never occurs during tidal breathing in normal subjects in either the supine or sitting position. In respiratory disease, however, FL may be present during tidal breathing; hence, tidal FL is said to occur when expiratory flow is maximal under the prevailing tidal volume, either at rest or during exercise [2, 3].

Pathophysiological Factors

Several factors may contribute to the occurrence of tidal FL. These include airway obstruction, lung volume, expiratory flow rate, and body posture. Airway obstruction limits maximal expiratory flow rates, reducing expiratory flow reserve. Similarly, the marked reduction of maximal expiratory flow rates at low lung volumes is crucial in promoting FL during tidal breathing. Reduced absolute lung volumes, as observed in gross obesity, restrictive lung and chest wall disorders, congestive heart failure, etc., are automatically associated with decreased maximal expiratory flow rates.

Increased ventilatory requirements augment the expiratory flow requirements because of greater tidal volume and faster respiratory frequency, predisposing to FL [3]. In the supine position, the Vr is lower as a result of gravitational forces, and normally the end-expiratory lung volume decreases with recumbency. Since the maximal flow-volume curve shows little variation on assuming the supine position, this posture facilitates FL because tidal breathing occurs at a lower lung volume, at which maximal expiratory flow rates are necessarily smaller [2].

Methods for Assessing Flow Limitation

Comparison between full (or partial) maximal and resting tidal flow-volume loops has been widely used to detect FL, which is assumed to be present when tidal expiratory flow impinges on or exceeds the maximal expiratory flows at the same lung volume [4]. This method, which should be performed by means of body plethysmography in order to avoid artifacts due to thoracic gas compression [5], is fatally flawed by the different volume and time history of the lung and airways prior to the maximal and tidal expirations, which influence the expiratory flows that are compared in the two maneuvers [6].