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Detection of Dynamic Lung Hyperinflation

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Abstract

Chronic obstructive pulmonary disease (COPD) is the general term for what has traditionally been referred to as chronic bronchitis and emphysema. COPD is commonly caused by inhaling toxic substances, harmful gases, and air pollution. COPD is an irreversible pathology that causes airflow obstruction and ventilation impairment. Until now, there is no complete treatment for COPD, and the main focus is on symptomatic treatment to improve symptoms and delay pathological progression. Airflow obstruction in COPD is caused by a decrease in pulmonary elastic contractile pressure due to peripheral airway involvement and emphysematous lesions, resulting in collapsed airways and the trapping of air in the lungs during forced expiration (referred to as unk or unk air trapping). Air trapping caused by collapsed peripheral airways strengthens with exertion or exercise, causing further lung hyperinflation. In COPD, dynamic lung hyperinflation (DLH) is one of the most significant determinants of short of breath (SOB) on exertion and exercise capacity. It is also an essential determinant in patients with significant lung hyperinflation and emphysema-dominant COPD. Measurement of DLH requires expensive equipment and instruments and is rarely evaluated in routine clinical practice. This review provides a summary of methods for measuring DLH, reaffirms how DLH and its therapeutic effects can be evaluated in routine practice, and outlines new methods that are being proposed.

Keywords: Dynamic lung hyperinflation; Measurement; detection; Chronic obstructive pulmonary disease; Hyperventilation; Exercise testing

Abbreviations: COPD: Chronic Obstructive Pulmonary Disease; DLH: Dynamic Lung Hyperinflation; FEV: Forced Expiratory Volume; FEV1.0: Forced Expiratory Volume in 1 Second; FEV1.0%: FEV as Percent of Forced Vital Capacity; VC: Vital Capacity; IC: Inspiratory Capacity; EELV: End-Expiratory Lung Volume; SOB: Short of Breath

What is COPD?

Chronic obstructive pulmonary disease (COPD) is the general term for what has traditionally been referred to as chronic bronchitis and emphysema. COPD is commonly caused by inhaling toxic substances, harmful gases, and air pollution [1,2]. The most common cause is smoking, and it is estimated that more than 90% of COPD cases in Japan are caused by smoking [3]. It is also found that 15-20% of smokers develop COPD [4]. The epidemiological data estimates that 8.6% of people over 40 years old, or about 5.3 million people, have the disease, yet many of them are still not diagnosed with COPD and do not receive proper treatment [5].

In the world, although the incidence rate has not changed over the past decade or so, it is estimated that 7.6 to 10.3%, or 292.0 to 391.9 million of the population over 30 years old are affected by COPD [1]. COPD causes inflammation of the lungs and bronchi, airflow obstruction due to bronchial stenosis, as well as irreversible alveolar wall destruction. These changes are thought to occur in COPD in different individuals. Due to the irreversible pathology of COPD, there is no complete treatment for COPD, and the main focus is on symptomatic treatment to improve symptoms and delay pathological progression [2]. Therefore, it is essential to prevent the early onset of COPD or to diagnose COPD at an early stage and to inhibit the pathological progression of the disease.

Dynamic Lung Hyperinflation and Its Effects

Airflow obstruction in COPD is caused by a decrease in pulmonary elastic contractile pressure due to peripheral airway involvement and emphysematous lesions, resulting in collapsed airways and trapped air in the lungs during forced expiration (this is called “air trapping”). This collapsed peripheral airway also occurs during resting breathing as the disease progresses, contributing to lung hyperinflation [6]. This air trapping caused by collapsed peripheral airways strengthens with exertion or exercise, causing further lung hyperinflation. This is called dynamic lung hyperinflation (DLH) and is an important factor in patients with COPD, contributing to increased respiratory workload, SOB on exertion, and reduced exercise tolerance [7,8].

Forced expiratory volume in 1 second (FEV1.0) determines the stage of COPD, but does not necessarily correlate with perceived symptoms, exercise tolerance, or quality of life [9]. Diaz et al. reported that inspiratory capacity (IC) correlates with maximal oxygen uptake and is a predictive factor of exercise tolerance in COPD patients with significant lung hyperinflation and low IC [10]. As a result of DLH, the distended lungs compress the intrathoracic organs (e.g., heart and inferior vena cava) due to increased intrathoracic pressure [11]. Thereby the circulating blood volume and cardiac output is reduced. A similar situation is ventilator management, where increased positive end expiratory pressure has been shown to decrease circulating blood volume [12,13]. On the other hand, they reported that airflow obstruction is a determinant of exercise tolerance in COPD patients with less pronounced lung hyperinflation [10].

Clinical Evaluation of COPD Patients

In patients with COPD, clinical evaluation parameters in recent years have emphasized not only forced expiratory volume (FEV) in 1 second (FEV1.0) as percent of forced vital capacity (FEV 1.0%) and % vital capacity (% VC), but also exercise tolerance and quality of life [11,14-17]. Furthermore, in addition to improvement in these parameters, improvements in lung hyperinflation, perceived symptoms, number of exacerbations and prevention of severe exacerbations, decline in respiratory function over time, and improvement in life expectancy are now being incorporated as primary endpoints in clinical studies [18-22].

In several epidemiological studies, there have been many potential patients with COPD previously [1,5]. However, if a patient has already been diagnosed with COPD after spirometry testing based on subjective symptoms, smoking history, and other screening factors, or is determined to have DLH, the patient can promptly receive the necessary treatment. However, DLH in COPD patients is often empirically measured (i.e., SOB in COPD patients is probably due to DLH) and is rarely quantitatively assessed by testing. In addition, there is no widespread method of measuring DLH to determine what type of assessment can be used to quantitatively evaluate DLH. The following methods for detecting DLH have been reported to date. If DLH occurs due to mild or unrecognized COPD onset, it may pass without screening and without any preventive treatment.

Metronome-Paced Incremental Hyperventilation

Since DLH occurs in a respiratory rate-dependent manner [23], a method for quantitative evaluation by hyperventilation without exercise testing has been presented [24]. In the resting state, end-expiratory lung volume (EELV) is measured using the body plethysmograph method, followed by maximal inspiration, and IC is measured. The subject then hyperventilates for 30 seconds at a respiratory rate of 20 breaths per minute in time with metronome, and EELV and IC are measured at the end of the hyperventilation. After a short break, the respiratory rate is increased in steps of 30 breaths/min and 40 breaths/min, and EELV and IC are measured in the same manner. DLH is evaluated by decreasing IC, and IC and EELV remain unchanged even if the respiratory rate is increased step by step in healthy never-smoker subjects. The degree of DLH is said to be stronger in the emphysema-dominant type of COPD than in the peripheral airway lesion-dominant type, in which emphysema is less prominent [25].

The conventional hyperventilation method requires an expensive body box to measure EELV and IC using the body plethysmograph method. Since the measurement of IC following hyperventilation is taken from the EELV during resting breathing and not from the EELV during hyperventilation, general spirometers cannot accurately measure IC after hyperventilation. Recently, a spirometer was improved and a new device was developed to easily measure DLH by the hyperventilation method [26]. In the new method, zero adjustments of flow can be made sequentially at atmospheric pressure by switching the circuit without removing the mouth from the mouthpiece. Furthermore, the measurement of IC following hyperventilation was improved so that it could be measured as the amount from the average EELV level of the last three breaths of hyperventilation. In normal subjects, IC remained unchanged with stepwise increases in respiratory rate, but in COPD patients, IC showed a decrease with increasing respiratory rate [26]. Assessment of DLH by a new method was able to predict exercise capacity and showed a significant correlation with 6-minute walking distance. It has also been reported that treatment with bronchodilator, significantly reduces DLH [27].

Exercise Testing

DLH is evaluated by measuring the IC of the patient by performing an exercise testing, and then measuring the decrease in IC every 1 to 2 minutes [28]. The exercise testing is performed by using a treadmill or bicycle ergometer to perform an incremental exercise testing, followed by a steady exercise testing in which exercise is sustained at 70 to 80% of peak VO_2. The use of portable breath gas analyzers allows IC evaluation in the walking test, and similarly, it has been reported that IC decreases over time in the 6-minute walk test [29,30]. This method requires a breath gas analyzer and an exercise testing equipment, and requires maximal IC measurement during exercise testing, which may increase the dyspnea. In addition, evaluation is difficult in critically ill or severe condition patients.

The evaluation of DLH with the hyperventilation method correlates well with DLH assessed with conventional exercise testing [31]. Good correlations have been obtained between the minimum IC during exercise testing and the minimum IC obtained by the hyperventilation method or the exercise duration. DLH by the hyperventilation method can predict DLH during exercise testing and exercise tolerance. Although the hyperventilation method correlates well with the IC measured by exercise testing, it is often difficult to evaluate it as easily in daily practice as spirometry.

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Computed Tomography

The method of evaluating DLH using computed tomography (CT) is similar to the method of measuring hyperventilation using spirometry [32]. Evaluation of dynamic pulmonary hyperinflation with CT showed significant increases in emphysema volume and emphysema index. However, CT measurements are taken in the supine position, which differs from general activity and breathing patterns [33]. In addition, total lung volume did not increase and IC could not be assessed, so it is unclear whether DLH occurred. Furthermore, the problem of exposure to radiation with CT makes it difficult to introduce an aggressive examination.

A possibility for a Novel Detection of Dynamic Lung Hyperinflation

Although IC reduction by measuring maximal IC during exercise testing has been a common quantitative test for DLH, recently, a new device-based hyperventilation method has been proposed from the body plethysmography-based hyperventilation method. However, the new devices are not widespread enough. There is also a method of evaluating DLH using CT, but it is not as common due to concerns about radiation exposure. Neither of these methods can be said to be easily measured or evaluated. In addition, several epidemiological studies have shown that the number of potential COPD patients has been high for some time [34]. In our specialty cardiac patients, a history of smoking is a risk factor for cardiovascular disease, and many patients have a history of smoking or comorbid COPD [35,36].

However, the development of mild or unrecognized COPD and DLH may pass without screening and without any preventive measures being taken. Most previous studies of DLH evaluation have been in patients with moderate to severe COPD, it is possible that mild or unrecognized COPD has been overlooked. Puente-Maestu et al. [37] measured end-inspiratory lung volume (EILV) and EELV during exercise testing in patients with severe COPD and found the increase in EELV was slower than the increase in EILV. Vogiatzis et al. [38] also reported that EELV increased less than EILV during incremental exercise testing in patients with stable COPD. This suggests that DLH may result in less expiration than inspiration during exercise. The tidal volume during exercise testing is also increased in patients with COPD [39]. However, there are few reports of DLH during cardiopulmonary exercise testing (CPET), the most standard method of assessing exercise tolerance [29,40-44].

CPET is a very good tool to examine SOB. VE/VCO2 slope and minimum VE/VCO2, which are used as indices of SOB in CPET, are indicators of ventilatory efficiency [45]. It has been reported that divergence between minimum VE/VCO2 and VE/VCO2 slope or flattening of VE/VCO2 slope and high Y-intercept may occur with the progression of COPD [46]. However, neither is typical of DLH. Existing cardiopulmonary exercise stress test-only indices may indicate the possibility of obstructive ventilatory impairment, but they cannot demonstrate that DLH is caused during exercise. A very recent study has demonstrated the possibility of using expiratory gas analysis data from CPET as an indicator of DLH by examining the difference between inspiratory and expiratory tidal volume [47].

In this method, as ventilation volume increases during incremental exercise testing, expiratory tidal volume decreases relative to inspiratory tidal volume, and DLH can be visually assessed by calculating the difference. This method differs from the spirometry-based hyperventilation method and IC measurement during exercise testing, and since it can be depicted using data from CPET, it may be a good indication for milder or asymptomatic patients, rather than for those with already diagnosed moderate to severe COPD. However, this method is an observational study of a very small number of cases, and the validity and efficacy of the index has not yet been examined. When the validity and efficacy of this detection method is clarified, it may be a convenient way to evaluate and diagnose potential airway and bronchial stenosis or mild COPD patients, such as cardiac patients and smokers, and may lead to preventive involvement.

Conclusion

Despite a large number of potential COPD patients, methods for their diagnosis and evaluation of DLH are not widely available. As a result, many potential COPD patients may be missing opportunities to prevent exacerbations or treat COPD. This leads to a decline in the quality of life of COPD patients and a shortened prognosis for life. The methods for assessing DLH that have been reported to date have been based on patients with moderate to severe COPD. Screening methods for DLH in mild or asymptomatic COPD patients, such as those presented at the end of this review, need to be thoroughly examined for validity and effectiveness and then disseminated. As methods for early detection become more widely known, preventive interventions can be implemented at an early stage, leading to improved quality of life and life expectancy for patients with COPD.

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