Role of Respiratory Impairment in Exercise Intolerance in COPD

莊銘隆

中山醫學大學附設醫院胸腔內科

Noxious agents such as tobacco smoke, pollutants, and occupational agents may damage airways, alveoli, interstitium and vasculature. The ensuing inflammatory mediators may damage further around the tissue. As such, the lung may progressively evolve to chronic bronchitis subsequently with obstruction and emphysema and eventually COPD ensues. Nevertheless, the damage is not just only in the lung but also in the thoracic cage and the extrapulmonary cardiovascular system and even in the locomotor muscles via a chronic systemic inflammatory process or disuse. The dysfunction of breathing center in COPD patients is complex and hard to differentiate the cause and effect. Cough, dyspnea and exercise intolerance are the most common symptoms that the COPD patients encounter.

Respiratory impairment is inevitable in COPD patients containing so many anatomical changes as aforementioned. Chronic inflammation in airways may cause airway lumen changed thus resistance elevated and flow impaired despite the thoracic pump generating higher force and pressure to overcome both in inspiratory and expiratory phases. The impairment may be mild but can be abnormal even at rest let alone while exertion. Lung volumes change as alveoli damaged augment. Total lung capacity increases as the lung parenchyma vanishes. Inspiratory capacity decreases as the functional residual capacity increases due to increase in dead space and/or airtrapping thereby restricting tidal volume expansion. Increase in the closing volume may also lead to maldistribution of gas in the lung. The high lung volume may put the lung in a disadvantageous posture as the inspiratory muscle may spend more energy to expand the lung. The reduced elastic recoil due to emphysema may impair the force for expiration. The diffusing capacity of lung may be reduced due to emphysema for a diminished total area of alveoli or pulmonary vasculopathy for an increased pulmonary vascular resistance, pulmonary hypertension and/or cor pulmonale.

All the impaired respiratory capacity can not adequately escalate to meet the respiratory demand during exercise thereby squeezing the breathing reserve capacity, ensuing flow limitation, dynamic hyperinflation, exercise limitation and exercise intolerance. The breathing reserve capacity as shown with the ratio of peak minute ventilation and maximum voluntary ventilation may reach 70% or more. Breathing frequency and tidal volume increase more rapidly with submaximal exercise as shown

with rapid shallow breathing index when compared to that performed in the normal subjects. Inspiratory time is shorter in response to exercise; however, the inspiratory duty cycle while approaching the peak exercise is lower as compared to that obtained from the normal subjects (0.5-0.55). This indicates that inspiratory muscle cannot sustain such breathing work incurred by exercise. Thus the breathing frequency at peak exercise is quite different from that obtained from patients with restrictive lung disease who usually perform the breathing frequency as high as 50 per minute or more. Airflow limitation and dynamic hyperinflation during exercise may be revealed as overlapping the tidal breathing with the maximum flow-volume loop measured at rest.

Ventilatory/respiratory muscle dysfunction is another common cause of pumping failure as measured with the product of the inspiratory effort and inspiratory duty cycle during exercise. Metabolic acidosis and high and/or low $\dot{V}/\dot{Q}$ mismatch (pulmonary gas exchange) during exercise are the other common causes of increased ventilatory demand. The demand may be more difficult to supply by an impaired respiratory system with lower ventilatory capacity.

In addition to imbalance between ventilatory capacity and ventilatory demand in COPD patients, the factors relating to circulation and neuroskeletomuscular system (including deconditioning), nutritional status, fitness, environmental condition, and psychological status are another potential causes of exercise limitation. Symptoms such as dyspnea, limb discomfort, and poor motivation in broad sense of physiologic function may also limit exercise performance.

Figure. Reduced Lung Function in Limiting Exercise Tolerance in COPD