

## 慢性阻塞性肺疾病 (COPD) 患者的膈肌适应和损伤改变

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## Diaphragm adaptations and muscle fiber dysfunctions in patients with chronic obstructive pulmonary disease (COPD)

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## 摘要

慢性阻塞性肺疾病 (chronic obstructive pulmonary disease, COPD) 由于气流受限和持续过度充气导致膈肌负荷过重,从而使膈肌结构、代谢和收缩功能发生改变,如肌卫星细胞活化,肌纤维向I类氧化纤维移行及有氧代谢增强等改变。但是由于慢性缺氧和细胞因子的影响,膈肌组织中乳酸和氧自由基生成增多,使参与膈肌能量代谢的蛋白酶功能受损,横桥循环动力不足。同时由于膈肌损伤和氧化应激激活蛋白水解途径,导致收缩蛋白的丢失,膈肌收缩力下降。最终,COPD患者呼吸困难,活动耐力下降并向恶性病理发展。本文就COPD患者膈肌所发生的适应和损伤改变做一综述。

**关键词** : 慢性阻塞性肺疾病(COPD), 膈肌损伤, 代谢, 收缩功能障碍

## Abstract :

Long-term airflow limitation and continuing hyperinflation lead to overloaded diaphragm in chronic obstructive pulmonary disease (COPD), so that diaphragm structure, metabolism and contractile function have altered, such as the activation of muscle satellite cell, muscle fiber shift towards fibers of oxidative type I, enhancement of aerobic metabolism. However, due to the impact of chronic hypoxia and cytokines, lactate and oxygen free radicals increased in diaphragm. Diaphragm metabolism protease dysfunction leads to underpowered cross-bridge circulation. In addition, the sarcomeric injury and oxidative stress activate proteolysis result in the loss of contractile protein and diaphragmatic contractility. Ultimately, the disease causing dyspnea, endurance intolerance, and then develops to cachexia. We reviewed recent studies and researches on the diaphragmatic adaptations and muscle fiber dysfunctions in patients with COPD.

**Key words** : chronic obstructive pulmonary disease (COPD) diaphragm injury metabolism contractile dysfunction

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