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Impacts of Aging and Opiates on Interactions between Brain, Lungs and Chest

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Introduction

In this Commentary, I briefly call attention to how the respiratory neural network perceives and reacts to aging and the effects of opiate medications. These two issues are of increasing relevance to the practicing pulmonologist.

In the first instance, because the fastest growing population throughout the world is over 65 years of age, there are increasing social and ethical reasons to understand how the aging process affects the respiratory system. In parallel with structural changes in the chest wall and diaphragm [1,2], aging in humans slows responsiveness and dulls sensitivity to action potential frequency at the neuromuscular junction of the diaphragm [3]. Consequently, there is lower maximal inspiratory pressure in the elderly.In neural networks of the mammalianbrainstem and inthe carotid bodies, irrespective of age,the CO_2/pH chemoreflex maintains respiratory homeostasis by matching ventilation to metabolic CO_2 production [4]. In the elderly, however, the ventilatory response to CO_2 is greater than in young adults, while the CO_2 threshold for increased inspiratory effort is reduced. Elderly subjects also have a lessened perception of added resistive loading, such as in COPD. The underlying mechanisms are poorly understood, but seem to be associated with reduced neural integration within the CNS [5]. Cough reflexes are also less forceful and productive in the elderly. One reason is that cough sensation is suppressed [6], in part by reduced sensitivity of slow- and rapidly-adapting vagal laryngeal afferents and by impaired cortical perception [7]. Weakening of the cough reflex could be a factor in the higher incidence of aspiration pneumonia in older subjects.

Opiate respiratory effects are important because synthetic opiate drugs have respiratory side effects that in some situations pose health risks and limit their therapeutic usefulness, and incidence of respiratory fatalities and near-fatalities are on the increase [8]. Opiates depress breathing depth and rate, blunt respiratory responsiveness to CO_2 and hypoxia, increase upper airway resistance and reduce pulmonary compliance. The opiate respiratory disturbances are mainly due to agonist activation of μ- and δ-subtypes of receptor and involve specific types of respiratory-related neurons in the ventrolateral medulla and the dorsolateral pons [9]. Synthetic opiates with affinity for either the μ- or the δ- type of receptor suppress all parameters of effective breathing. They depress rate and depth of respiration, induce chest and abdominal wall rigidity, reduce upper airway patency and blunt respiratory responsiveness to carbon dioxide and hypoxia [10-13]. Elderly subjects are notably more sensitive to respiratory depression by opioids [14]. One reason, among others, is that decreased glomerular filtration and reduced total and functional hepatic blood flow reduce the capacity to excrete hydrophilic opioids. In addition, pharmacodynamic changes related to drug-receptor interactions and intracellular second messenger efficacy increase the intrinsic activities of opiates such as fentanyl and its congeners, andpromote longer duration of drug effect [15-17].

Neural regulation of the airways, diaphragm and chest wall, whether during aging or affected by opiate medications or affected in other manners, is complicated, often misunderstood and needs further studies and exchange of ideas and information that will aidthe pulmonologist in the diagnosis and treatment of respiratory dysfunction.

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Competing Interests

The authors declare that they have no competing interests.

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