RESPIRATORY HEALTH IN THE ELDERLY

M. Pokorski
Department of Respiratory Research,
Medical Research Center, Polish Academy of Sciences,
Warsaw, Poland

This study intends to tackle some of the aspects of aging of the respiratory system’s components at both structural and functional levels. The respiratory system is directly exposed to the environmental air, pumping ca 20,000 liters of air through the lungs daily, which poses a formidable functional challenge. No wonder that respiratory health is linked to the quality of life and longevity of the elderly. All components of the respiratory system, aside from being affected by individuals’ clinical history and habits, for instance smoking, are subject to the aging process.

The lung is characterized by an extensive air-liquid interface of alveoli. Normal lung function is determined by the integrity of the pulmonary surfactant. Surfactant is a mixture of phospholipids, neutral lipids such as cholesterol, and proteins. It is assembled in the endoplasmic reticulum and the Golgi apparatus of alveolar type II pneumocytes and is stored in lamellar bodies until exocytosis. Surfactant forms the alveolar extracellular lining layer and is key for the maintenance of alveoli in the open state, by decreasing surface tension, and for the proper gas exchange (1). With age, subtle but distinct degenerative changes develop in the lamellar bodies of type II pneumocytes. In the old rat, >2 years of age, these changes consist of defects of phospholipid arrangement, which, in turn, causes disruptions or blurredness in the continuity of inner lamellae, or their partial disappearance. The surfactant formed and secreted into the alveolar hypophase is in part disordered structurally, frayed off, dysfunctional, and at times separated from the epithelial cell membrane by a layer of exudative fluid. All this is accompanied by an enhanced activity of alveolar macrophages performing the cleaning up role.

Surfactant defects are importunate at a time when alveolar sacs are enlarged in old age. The enlargement concerns airspaces in general due to decreasing alveolar wall tissue accompanied by the loss of elastic recoil (2). Such changes show gender differences, as they are more pronounced in men than in women, and have functional implications in that residual lung volume, closing volume, and functional residual capacity increase, whereas vital capacity and FEV1 decrease (3). Stiffening of the rib cage, decreased respiratory muscle strength, and redistribution of blood flow to the upper parts of lungs all lead to increased ventilation-perfusion mismatch with advancing age. Accordingly, one
could expect a progressively declining partial pressure of oxygen (PaO₂) with age. However, such a decline, reported in older studies, seems at present an open issue. More precise, contemporary measurements of PaO₂ failed to substantiate the presence of an inverse relationship between age and PaO₂ in healthy elderly people. Nor could expiratory flow limitation be substantiated, although expiratory flow rate, particularly at lower lung volumes, may be lower in old age (4).

Even though the PaO₂ does not decrease appreciably with advancing age, older adults are definitely prone to develop periods of relative hypoxia due to general atherosclerotic changes, slower blood circulation from the heart to peripheral organs, and consequently hindered O₂ diffusion to tissue. When the respiratory system is under strain such as exercise, fever, and other occurrences, the possible hypoxic consequences come easier to sight. The ability to adapt to periods of hypoxia is crucial to disease free functioning and survival. This ability has to do with the performance of the hypoxic chemoreflex mediated by the carotid body. Carotid body chemoreceptors are excited by reductions in PaO₂. The signal consisting of increased impulse rate generated by chemoreceptors is conveyed to the brain stem respiratory areas, giving a driving force to pulmonary hyperventilation aimed at warding off hypoxia by increasing O₂ delivery to the lungs (5). The stimulatory ventilatory response to hypoxia is a measure of functional sensitivity of carotid body chemoreceptors. Whether the hypoxic chemoreflex weakens with advancing age is another contentious issue. There are older reports that that might be the case to even as much as 50% of norm (6). Indeed, the decreased hypoxic ventilatory response could be a result of any combination of respiratory changes with age.

Morphologically, chemoreceptor cells undergo degenerative changes and the surrounding connective tissue becomes enlarged in the old carotid body (7). That may hinder O₂ diffusion from capillaries to chemoreceptor cells and render them less responsive to PaO₂ reductions. Slower synaptic transmission makes the central respiratory controller less efficient. Moreover, the effector part, that is respiratory motoneurons and muscles, become less efficient with age. There are, however, studies showing that the carotid body-mediated hypoxic ventilatory response is undiminished in old age. In one of our recent studies, we have shown that hypoxic ventilatory sensitivity, as measured from the linear slope of minute ventilation on arterial blood oxygen saturation, did not decrease during progressive hypoxia in healthy old women compared with the young ones. Nor was hypoxic sensitivity dampened within the 20-year time frame in women between 60 and 80 years of age (8). The ventilatory response to hypoxia would just be independent of age, even though the lung volumes, assessed in routine spirometric tests, were nearly halved in this age group. Thus, elderly subject’s pulmonary ventilation is able to compensate for natural lowering of lung capacity with age to meet the hyperventilatory challenge.

Hypoxic episodes are liable to appear more often in the elderly, particularly connected with breathing disorders during sleep. Increasing the hypoxic ventilatory reactivity seems desirable for respiratory health, as it would help deliver O₂ to tissues to meet metabolic
needs and counteract periodic desaturations. There are no good pharmacological ways to rejuvenate or invigorate the carotid hypoxic chemoreflex in old age. The only one drug available to his end, almitrine, has but modest effects and its use is limited due to the side effects consisting of peripheral neuropathy. The search for ways to improve carotid body function is hampered by the unresolved mechanisms of the organ’s detection of hypoxia. It is now known that the redox state of chemoreceptor cells plays into these mechanisms.

The presence of ascorbate, a leading water-soluble antioxidant, has been unraveled in the carotid body (9). Asorbate supplementation seems to offer promise in enhancement of hypoxic reactivity in humans (10). Further research is warranted aimed at improving carotid body function. Such an improvement may foster respiratory health and mitigate the natural process of aging.

LITERATURE