Attenuation of the Hering-Breuer Reflex: Yet Another Adverse Consequence of COPD?

Erik van Lunteren

Pulmonary Division, Department of Medicine, Case Western Reserve University and Cleveland VA Medical Center, Cleveland, Ohio, USA

Receptors in the airways and parenchyma of the lung send afferent information to the brainstem via the vagus nerves, thereby exerting strong influences on the rate and pattern of breathing. For example, airway irritant receptors can be excited by a variety of inhaled particulates and irritant gasses, whereas parenchymal J receptors can be stimulated by perturbations of the pulmonary vasculature, such as pulmonary edema. Their activation is responsible for many of the alterations in the motor output to the respiratory muscles seen in human cardiopulmonary diseases, including tachypnea, apnea and coughing.

Pulmonary slowly adapting stretch receptors, on the other hand, respond mainly to changes in lung volume rather than to exogenous stimuli. When activated by sustained lung inflation, they reflexly inhibit the onset of the subsequent inspiration, as noted by Hering and Breuer [1] almost 150 years ago. When activated during the course of a spontaneous inspiratory effort, they reflexly terminate inspiration [2] and alter the pattern and distribution of motor output to various thoracic and upper airway respiratory muscles [3]. The role these receptors play in the regulation of breathing is felt to be generally greater in neonates than in adults, and generally greater in several subprimate mammalian species than in humans.

In the present issue of *Respiration*, Tryfon et al. [4] examined the strength of the Hering-Breuer reflex in humans without and with lung disease. The group with

normal pulmonary function was comprised of subjects undergoing mechanical ventilation in the intensive care unit following various surgical procedures. The diseased subjects consisted of two groups, those with interstitial fibrosis and those with chronic obstructive pulmonary disease (COPD), all of whom were undergoing mechanical ventilation for respiratory failure. The strength of the Hering-Breuer reflex was tested by determining the degree to which end-inspiratory airway occlusion delayed the onset of the subsequent inspiration beyond the range seen during unoccluded breaths as well as the extent to which this was augmented further by the prior brief application of positive end expiratory pressure (PEEP). The Hering-Breuer reflex was demonstrable in all three groups, but was attenuated significantly in subjects with COPD relative to both normals and those with interstitial fibrosis.

Several mechanisms could potentially account for the diminution of the Hering-Breuer reflex in subjects with COPD. These include a reduction in the number of stretch receptors as a direct consequence of the disease, a flattening of the response of the receptors to stretch resulting from adaptation to the chronic hyperinflation, and an adaptation of central respiratory neural network responsiveness to continued afferent traffic. The relative importance of these possibilities cannot be ascertained from the data presented by Patakas et al. [4].

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Erik van Lunteren, MD
Pulmonary Section, 111J(W), Cleveland VA Medical Center
10701 East Boulevard
Cleveland, OH 44106 (USA)
Tel. +1 216 791 3800 (ext. 4616), Fax +1 216 231 3420, E-Mail exv4@po.cwru.edu

These findings are notable for several reasons. First, is that they demonstrate that the Hering-Breuer reflex is in fact alive and well in the adult human. Second, they indicate that lung disease, in particular COPD, can modulate the strength of this reflex. A possible consequence of the attenuation of the Hering-Breuer reflex in this disease is a slowing of respiratory rate by prolonging inspiration. On the other hand, patients with COPD may undergo dynamic hyperinflation (auto-PEEP) when acutely ill. Based

on the data by Patakas et al. [4], this should cause a compensatory strengthening of the Hering-Breuer reflex, minimizing any slowing of respiratory rate. Third, these findings indicate that use of PEEP during mechanical ventilation will strengthen the Hering-Breuer reflex in subjects with both healthy and diseased lungs, suggesting a mechanism by which PEEP may directly modulate spontaneous respiratory frequency in the critically ill.

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