Stimulatory effect of CO2 on vagal bronchopulmonary C-fiber afferents during airway inflammation

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摘要

Abstract

This study investigated: 1) whether pulmonary C-fibers are activated by a transient increase in the CO2 concentration of alveolar gas; 2) if the CO2 sensitivity of these afferents is altered during airway inflammation. Single-unit pulmonary C-fiber activity was recorded in anesthetized, open-chest rats. Transient alveolar hypercapnia (HPC) was induced by administering a CO2-enriched gas mixture (25 – 30% CO2, 21% O2, balance N2) for 5 – 8 breaths, which increased alveolar CO2 concentration progressively to near or above 13% for 3 – 5 s and lowered the arterial pH transiently to 7.10 ± 0.05. Our results showed: 1) HPC evoked only a mild stimulation in a small fraction (4/47) of pulmonary C-fibers, and there was no significant change in fiber activity (ΔFA = 0.22 ± 0.16 impulses/s; $P > 0.1$, n = 47); 2) in sharp contrast, after airway exposure to poly-l-lysine (PLL), a cationic protein known to induce mucosal injury, the same challenge of transient HPC activated 87.5% of the pulmonary C-fibers tested and evoked a distinct stimulatory effect on these afferents (ΔFA = 6.59 ± 1.78 impulses/s; $P < 0.01$, n = 8); 3) similar potentiation of the C-fiber response to HPC was also observed after acute exposure to ozone (n = 6), and during a constant infusion of inflammatory mediators such as adenosine (n = 15) or prostaglandin E2 (n = 12); and 4) The enhanced C-fiber sensitivity to CO2 after PLL was completely abrogated by infusion of NaHCO3 (1.82 mmol/kg/min) that prevented the reduction in pH during HPC (n = 6). In conclusion, only a small percentage (< 10%) of the bronchopulmonary C-fibers exhibit CO2 sensitivity under control condition, but alveolar HPC exerts a consistent and pronounced stimulatory effect on the C-fiber endings during airway inflammation. This effect of CO2 is probably mediated through the action of hydrogen ions.