**Abstract**

Nasal high flow (NHF) reduces minute ventilation and ventilatory loads during sleep but the mechanisms are not clear. We hypothesised NHF reduces ventilation in proportion to physiological but not anatomical dead space. 11 subjects (five controls and six chronic obstructive pulmonary disease (COPD) patients) underwent polysomnography with transcutaneous carbon dioxide (CO$_2$) monitoring under a metabolic hood. During stable non-rapid eye movement stage 2 sleep, subjects received NHF (20 L·min$^{-1}$) intermittently for periods of 5-10 min. We measured CO$_2$ production and calculated dead space ventilation. Controls and COPD patients responded similarly to NHF. NHF reduced minute ventilation (from 5.6±0.4 to 4.8±0.4 L·min$^{-1}$; p<0.05) and tidal volume (from 0.34±0.03 to 0.3±0.03 L; p<0.05) without a change in energy expenditure, transcutaneous CO$_2$ or alveolar ventilation. There was a significant decrease in dead space ventilation (from 2.5±0.4 to 1.6±0.4 L·min$^{-1}$; p<0.05), but not in respiratory rate. The reduction in dead space ventilation correlated with baseline physiological dead space fraction ($r^2=0.36$; p<0.05), but not with respiratory rate or anatomical dead space volume. During sleep, NHF decreases minute ventilation due to an overall reduction in dead space ventilation in proportion to the extent of baseline physiological dead space fraction.