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RESPIRATORY CONTROL DURING EXPOSURE TO SIMULATED MICROGRAVITY

Abstract

Background. Recent studies have shown that there is a marked redistribution of fluid from lower to upper half of the body during the early stages of space flight. To increase thoracic blood volume due to fluid shifts within the entire body might result in changes in the control of ventilation in humans. However early adaptive response of respiratory system to zero gravity have not been well characterized. Head-down tilt (HDT) is commonly used as model for simulating weightlessness. Our previous study has shown increase of central venous pressure (CVP), decrease of functional residual capacity (FRC) and lung compliance (C) at postural change from supine to HDT-30 in anesthetized cats.

Aim. To examine the role of vagal afferents in the control mechanism of ventilation and compensatory capacity of the respiratory system during short-term exposure at head-down tilt position.

Methods. The experiments were performed in vagaly intact or vagotomized, anesthetized (uretan, 1000 mg/kg), tracheostomized rats (310 g). The response of tidal volume (VT), breathing frequency (f), minute lung ventilation (VE), esophageal pressure (Poes), inspiratory occlusion pressure (P0.1) were examined at supine (baseline) and after 30 min HDT-30 exposure.

Results. There were significant decreases in VT by 18%, VE by 14% after 30 min exposure in HDT-30 relative to supine. Increases both resistive by 50% ($P<0.05$) and elastic component by 90% ($P<0.05$) of respiratory resistance were found. On the other hand rising Poes by 116% was seen which reflects an increase in inspiratory muscle output occurs when respiratory load increases. After bilateral vagotomy Poes rose only to 65% in comparison to vagal intact animals. Inspiratory occlusion pressure evoked increase P0.1 in both supine and HDT. In supine this value rose to 536% ($P<0.01$), whereas in HDT by 320% ($P<0.05$).

Conclusion. HDT in anesthetized animals induces ventilatory augmentation accompanied with the rise of airway resistance due to increased intrathoracic blood volume. A compensatory capacity of the respiratory system is diminishing during short-term exposure at HDT-30. One of the possible mechanism of compensatory response respiratory system is the vagal afferent, presumably originating from slowly adapting lung stretch receptors due to decreased lung volume on HDT.