INFLUENCE OF CENTRAL HYPOVOLEMIA ON RESPIRATORY FUNCTION

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It has been shown that pathologically high negative pressure evokes central hypervolemia during episodes of obstructive sleep apnea. Also, forced breathing during an asthmatic attack induces increased venous return and overflow of the lung by blood. It is known that central hypervolemia facilitates cardiovascular deconditioning. The aim of the present study was to investigate whether central hypervolemia influences respiratory function and compensatory capability of the respiratory system.

Material and methods: The experiments were performed on the anaesthetized rats. Central hypervolemia was induced by head-down tilt on -30 degree rotation (HDT). HDT increased negative value of central venous pressure (from -2.0 ±0.4 cm H_2O to 3.9 ±0.8 cm H_2O, $P<0.05$). Central venous pressure was registered in v. cava superior. Pneumotachography had been used to measure peak flow, tidal volume, and parameters of respiratory cycles. Intrathoracic pressure was measured as the esophageal pressure inspiratory swings (Poes). Maximal mechanical load was created by occlusion of airway at the end of expiration. Load compensatory response was measured as Poes in the first occlusion inspiration and expressed as the percentage change of the unloaded Poes.

Results: After 30 min of central hypervolemia, resistive and elastic component of airway resistance significantly increased (by 50% and 90%, respectively), negative intrathoracic pressure (indirect measure of respiratory effort) rosed (by 116 %), inspiratory flow, and tidal volume and minute ventilation decreased (by 28%, 18%, and 14%, respectively) ($P<0.05$). After vagotomy the same central hypervolemia had a smaller effect on the respiratory effort which increased just by 65% and as a result, both tidal volume and minute ventilation decreased (by 30% $P<0.05$). Moreover, central hypervolemia weakened the load compensatory response.

Conclusion: Central hypervolemia increases mechanical load on the respiratory system and decreases its compensatory capabilities. Afferent traffic from pulmonary mechanoreceptors mediates the respiratory response to central hypervolemia.