The impact of vasopressin on pulmonary ventilation and arterial chemoreceptor reflex.

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Введение

Vasopressin (AVP) is a potent pressor neurohormone. Limited number of studies suggest that AVP may inhibit pulmonary ventilation. However, main stimuli for AVP – hypotension and hypovolemia – lead to increase in pulmonary ventilation. On the other hand, gaseous and acid-base homeostasis of arterial blood to large extend depends on arterial chemoreceptor reflex originating from carotid and aortic bodies. This reflex is triggered by hypoxemia, hypercapnia, acidity and drop of blood pressure. Stimulation of arterial chemoreceptors beside hyperventilation causes a marked increase in AVP blood concentration. Therefore, question arises if vasopressin is involved in any feedback mechanism on arterial chemoreflex.

Цель исследования

Our primarily goal was to investigate how intravenous AVP infusion affects ventilation. Furthermore, we assessed also the impact of AVP infusion on arterial chemoreceptor reflex.

Материалы и методы

The study was done in adult male Sprague Dawley rats anesthetized with urethane. The animals were divided into 3 groups, infused with: AVP (n=6) (AVP group), saline/KCN (n=6) (control group) and AVP/KCN (study group). Blood pressure was recorded from the catheter implanted into femoral artery. Another catheter, placed in femoral vein served for intravenous injections. In addition, a tracheal tube was inserted through tracheotomy for recording the airflow. In the first group mean arterial blood pressure (MABP), respiratory rate (RR) and minute ventilation (MV) were recorded after intravenous infusion of saline (0.9%NaCl, 100 microL) followed by intravenous infusion of pressor dose of AVP (10 ng/100 microL). In the following two groups the arterial chemoreflex was triggered pharmacologically with potassium cyanide (KCN) (30 microg/100 microL), which was administered after pretreatment with 0.9%NaCl (100 microL i.v.) or pretreatment with AVP infusion (0.6ng/min/20 microL for 5 min).

Результаты

Intravenous infusion of 0.9% NaCl had no effect on hemodynamic and ventilatory parameters, whereas AVP triggered a significant decrease in MV and RR and an increase in MABP (p less 0.05, paired Student t-test). Stimulation of the chemoreflex with KCN led to increase in MABP, RR, MV and a decrease in ETCO2. Peak response to KCN injection did not differ between control and study group. However, group pretreated with AVP had a significantly longer ventilatory response to pharmacological trigger (p less0.05, Student t-test). In the experimental group, MV was still increased above the baseline and ETCO2 was lower than baseline at 1 min after KCN-induced chemoreflex.

Выводы

We show that intravenous AVP administered at pressor dose inhibits pulmonary ventilation. Furthermore, our findings indicate that vasopressin sensitizes the arterial chemoreceptors which is manifested in the prolonged respiratory response to pharmacologically induced chemoreflex in rats. Together, our results provide the evidence for involvement of AVP in the control of respiratory system.