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THE ROLE OF VASOPRESSIN V2 RECEPTOR IN THE FUNCTIONING OF CAROTID BODY AND ARTERIAL CHEMOREFLEX.

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The carotid body (CB) is the main peripheral chemoreceptor which detects chemical and hormonal signals in bloodstream. What's more it's involved in triggering arterial chemoreflex including increasing ventilation, sympathoexcitation and rise in blood pressure. Vasopressin (AVP) it's well known as a neurohormone involved in regulation of cardiovascular and respiratory system. V2 receptors for vasopressin are present mostly in renals but also in extra-renal tissue, such as lungs and vessles. It has been already proved that AVP affects ventilation via CBs, but the role of V2 receptors has not been determined so far.

Aim:

Our goal was to find out whether CBs contain V2 receptors for AVP and how the receptors influence arterial chemoreflex.

Methods:

The study was performed on adult male Sprague-Dawley rats (n=12). Under urethane terminal anesthesia animals were implanted with the catheters in both femoral artery and femoral vein for recording hemodynamic parameters (mean arterial blood pressure - MABP; heart rate - HR) and for intravenous infusions. Tracheotomy was also made and was followed by insertion of the tracheal tube for recording of ventilatory parameters (minute ventilation - MV; respiratory rate - RR). The control group of animals (n=6) was pretreated with 0.9%NaCl (100 µl i.v.) followed by pharmacological testing of arterial chemoreflex with potassium cyanide (KCN) (30 µg/100 µl i.v.). The experimental group (n=6) was pretreated with V2 receptor antagonist (Tolvaptan) (0,5 mg/100 µl) followed by pharmacological testing of the arterial chemoreflex. After euthanasia, carotid body bifurcations were collected and thin (30 µm) sections were cut on a cryostat. CBs were immunostained with primary antibodies against tyrosine hydroxylase (TH) to detect chemoreceptor cells (glomus cells) and V2 receptor. After incubation with secondary antibodies, sections were visualised with confocal microscopy.

Results:

Infusions of KCN indicated significant increase in MABP, MV and RR in control group. Pretreatment with V2 receptor antagonist had insignificant effect on arterial chemoreflex. Immunostaing confirmed presence of V2 receptors in the CB. However, they were not located on the chemoreceptive glomus cells.

Conclusions:

Our results show that V2 receptors are expressed in the carotid bodies, but not on glomus cells.

In addition, inhibition of V2 receptors seems to have insignificant effect on the arterial chemoreflex.