Commentary

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Inducing Hyponatremia through Vasopressin Administration

Martin Fronius *

Department of Physiology, University of Otago, Dunedin, Aotearoa New Zealand

***Corresponding author:** Martin Fronius. Department of Physiology, University of Otago, Dunedin, Aotearoa New Zealand, E-mail: frnusmartin@otago.ac.nz

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Description

Vasopressin is an analog of the hormone diuretic that is frequently used to raise blood pressure in children who are thermodynamically unstable. If norepinephrine failed to maintain the Mean Blood Pressure (MAP) goal, current guidelines recommend vasopressin as a second line treatment or as an adjuvant therapy to reduce the need for norepinephrine dosing in vasoplegic shock patients. Vasopressin lowers blood pressure by activating V1 receptors, which raises arterial blood pressure and systemic vascular resistance. It has an antidiuretic effect at lower doses by stimulating the V2 receptors. Vasopressin acts as a procoagulant by stimulating V1a receptors, which result in platelet aggregation, and V2 receptors, which result in the release of coagulation factors. It is interesting to note that administration of vasopressin may cause hyponatremia through an unidentified mechanism. Free water retention caused by stimulation of the V2 receptors in the kidneys may account for this phenomenon. According to Choong et al., hospital-acquired hyponatremia affects 17% to 45% of children admitted to hospitals.2011. Children who are critically ill and have certain comorbidities, like a congenital heart defect, are more likely to have neurodevelopmental abnormalities, necessitating close monitoring of hyponatremia while in the hospital.

Vasopressin Infusion for Hemodynamic Instability

The occurrence of hyponatremia in pediatric patients undergoing vasopressin therapy following cardiac surgery is poorly documented in the literature. Davalos, others reported that nearly half of pediatric patients who received a vasopressin infusion for hemodynamic instability after complex cardiac surgery experienced hyponatremia. In another study, neonates with complex heart disease who received vasopressin had lower serum sodium concentrations in the first 48 hours than those who did not. There was no increase in the incidence of hyponatremia in some studies that looked at the use of vasopressin in adult and pediatric patients who were in vasodilator shock. When vasopressin was administered to non-cardiac patients, numerous studies have documented the prevalence of hyponatremia. A randomized trial that looked at the use of low-dose vasopressin in non-septic critically ill children found a high rate of hyponatremia with the low-dose vasopressin infusion. In addition, the administration of a high vasopressin dose (0.4 units/min) for the treatment of patients with acute variceal hemorrhage was associated with a higher rate of hyponatremia. During pregnancy and lactation, renal water reabsorption increases to meet the cardiovascular demands of the developing fetus and newborn baby. Vasopressin, an antidiuretic hormone, helps the kidney reabsorb water, and body fluid osmolality is the primary factor that drives its secretion. As a result, vasopressin secretion typically decreases when osmolality is reduced. However, vasopressin levels are maintained to drive blood volume expansion despite the fact that water retention significantly reduces osmolality during pregnancy and lactation. The cellular mechanisms that maintain vasopressin secretion despite decreased osmolality during pregnancy and lactation are unknown, despite its significance for successful reproduction.

Activity of Vasopressin on Neurons

Through the expression of the N-terminal truncated-transient receptor potential vanilloid-1 channel (N-TRPV1), which is mechanically activated by osmotically-induced cell shrinkage to increase vasopressin neuron activity, vasopressin is secreted by neurons that are intrinsically osmosensitive.TRPV4 is expressed by vasopressin neurons as well, but its function in vasopressin neurons is unclear. The novel evidence that TRPV4 forms functional channels with N-TRPV1 that have a higher single-channel conductance than channels formed with N-TRPV1 alone is summarized here. In order to maintain vasopressin secretion during pregnancy and lactation and increase blood volume necessary for successful reproduction, we propose that upregulation of TRPV4 heteromerization with N-TRPV1 might be necessary. Our daily interactions with others can have a significant, positive or negative impact on our health. We focused on neural circuits involving serotonin neurons of the dorsal raphe and vasopressin neurons of the bed nucleus of the stria terminalis in order to gain a deeper comprehension of the neural circuitry that governs social behavior. Previous research indicates that male mice interact with a female to activate BNST vasopressin neurons and that vasopressin indirectly excites serotonin neurons. We tested the hypothesis that certain social interactions would also activate neurons in the DR, particularly neurons that express the vasopressin 1A receptor (Avpr1a),

which may be the BNST vasopressin neurons' direct targets. We found that male and female subjects exposed to a female conspecific showed activation in the DR in separate experiments using immunohistochemistry and in situ hybridization. The activated neurons included populations of Avpr1a-expressing and other non-serotonergic, non-Avpr1a neurons in roughly equal numbers. The population of Avpr1a neurons in the DR is largely unknown. The majority of DR Avpr1a neurons, according to electrophysiological data, behave similarly to fast-spiking interneurons found in other brain regions. In the DR, there are glutamatergic, GABAergic, and serotonergic subtypes of Avpr1a neurons, according to RNAseq and in situ hybridization results. The sum of our findings lends credence to the hypothesis that a subset of vasopressin-responsive interneurons in the DR may transmit prosaically stimulus-specific social signals from the forebrain BNST to the serotonergic DR system. We investigated the effects of administering oxytocin and vasopressin on neural reactivity to infant cry sounds in a randomized double-blind within-subject control study on 70 first-time fathers in their first year of fatherhood. Additionally, we investigated whether fathers' early childhood experiences moderated the effects of oxytocin and vasopressin administration on neural reactivity. Using functional magnetic resonance imaging (fMRI), neural reactivity to infant cry sounds was measured in comparison to that of control sounds. In addition, participants shared stories about how their parents withdrew their love and disciplined them harshly in childhood. Analyses of the whole brain revealed that the administration of vasopressin or oxytocin had no significant effect on the neural activation in response to infant cry sounds. Amygdala activation was less pronounced in the vasopressin and oxytocin groups than in the placebo group, according to region of interest analyses. Fathers' early experiences had no moderating effect, according to our findings. According to our findings, administering oxytocin may lessen feelings of anxiety or aversion to a crying infant. Further research is needed to determine whether decreased amygdala activation following vasopressin administration is an affiliated response to infant distress or if it can be explained by contextual factors like the infant's unfamiliarity or lack of high levels of threat. The present study revealed that important hormones like oxytocin and vasopressin are involved in neural models of infant cry perception in fatherhood.