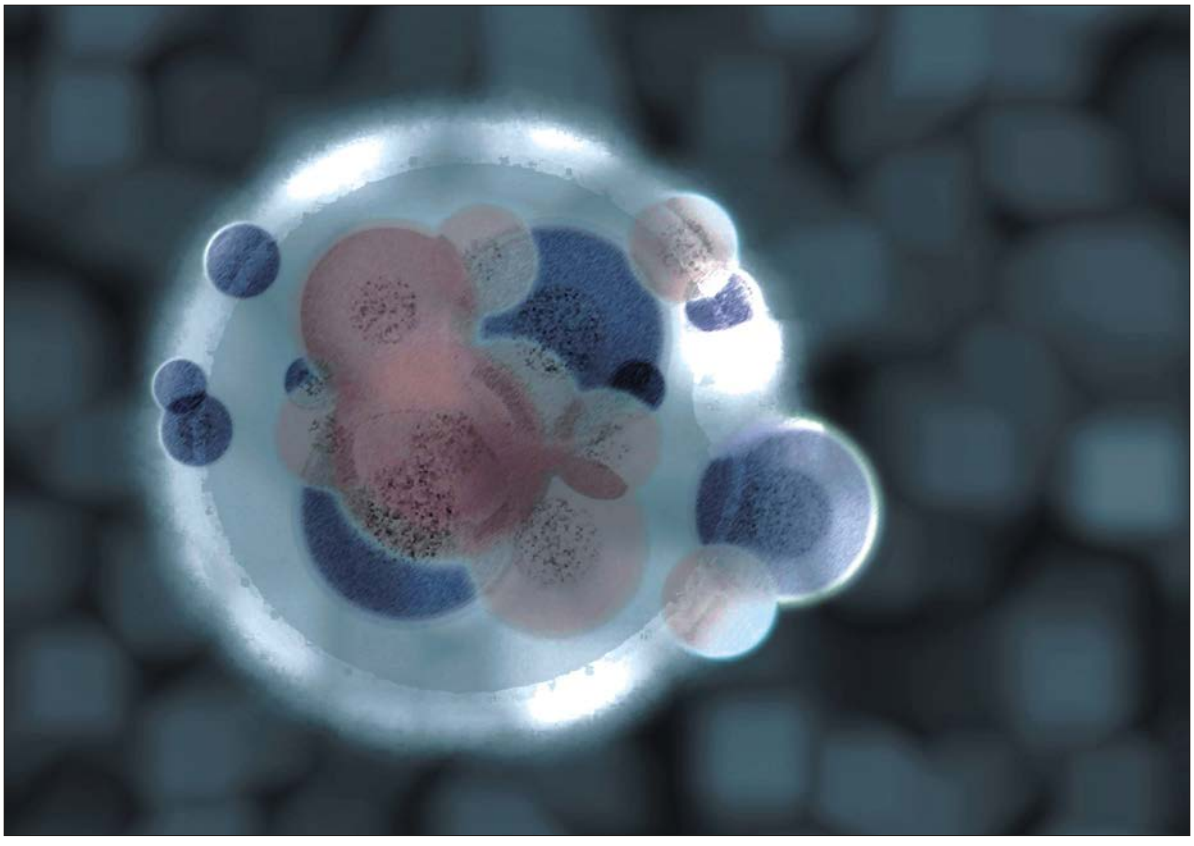

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Angiotensin and Arginine Vasopressin Receptor Subtypes of the Lateral Preoptic Area Effect on the Sodium Balance

[1-3]Wilson Abrão Saad, [2]Luiz Antonio de Arruda Camargo, [4]José Antunes-Rodrigues, [5]William Abrão Saad, [6]Ismael Franscisco Motta Sigueira Guarda and [6]Renata Saad Guarda
[1]Basic Institute of Biosciences, UNITAU, Taubaté SP, Brazil, [2]School of Dentistry, Department of Physiology and Pathology, Paulista State University, UNESP, Araraquara SP, Brazil, [3]Department of Natural and Exact Science, UNIARA, Araraquara SP, Brazil, [4]School of Medicine, Department of Physiology, São Paulo University, USP-Ribeirão Preto SP, Brazil [5]Clinic Hospital, University of São Paulo, Brazil [6]Department of Anesthesiology, Public Hospital of State of São Paulo, São Paulo, Brazil

Abstract: It was speculated whether the influence of lateral pvasopressin (AVP) and angiotensin (ANG II). The present study investigated the effects of central administration of specific AVP and ANG II antagonists ($d(CH_2)_5$-Tyr (Me)-AVP (AAVP) and [Adamanteanacetyl[1], 0-ET-$_D$-Tyr[2], Val[4], Aminobutyryl[6], Arg[8,9]]-AVP (ATAVP) antagonists of V_1 and V_2 receptors of AVP. Also the effects of losartan and CGP42112A (selective ligands of the AT_1 and AT_2 angiotensin receptors, respectively), was investigated on Na^+ uptake and renal fluid and electrolyte excretion. After an acclimatization period of 7 days, the animals were maintained under tribromoethanol (200 mg kg^{-1} body weight, intraperitoneal) anesthesia and placed in a Kopf stereotaxic instrument. Stainless guide cannula was implanted into the LPO. AAVP and ATAVP injected into the LPO prior to AVP produced a reduction in the sodium intake responses. Both the AT_1 and AT_2 ligands administered into the LPO elicited a decrease in the sodium intake induced by AVP injected into the LPO, but losartan was more effective than CGP 42112A. AVP injection into LPO increased sodium renal excretion, but this was reduced by prior AAVP administration. The ATAVP produced a decreased in the natriuretic effect of AVP. The losartan injected into LPO previous to AVP decreased the sodium excretion and the CGP 421122A also decreased the natriuretic effect of AVP with a high intensity. The AVP produced an antidiuresis effect that was inhibited by prior administration into LPO of the ATAVP. The AAVP produced no change in the antidiuretic effect of AVP. These results suggest that LPO are implicated in sodium balance that is mediated by V_1, V_2, AT_1 and AT_2 receptors.

Key words: Vasopressin, angiotensin receptor subtypes, sodium balance, lateral preoptic area

INTRODUCTION

The lateral pre-optic area of the hypothalamus (LPO) contains osmosensitivity receptors, which are involved in sodium balance. Blass and Epstein[1] demonstrated that the electrolyte ablation of the LPO bilaterally abolished the water intake in rat following dehydration induced by saline. Furthermore Saad and Camargo[2] demonstrated that electrolyte bilaterally lesion of the LPO abolished the water ingestion induced by cellular dehydration, hypotension, hypovolemia and deprivation. Recently it has been demonstrated that in the LPO has extracellular concentration of amino acid neurotransmitters. In the LPO, calcium and nerve impulse dependent increases of arginine, glutamate and aspartate[3].

The systemic application of ibersartan and losartan abolished the ANGII central physiologic responses water intake, sodium intake and increase arterial pressure[4]. The role of renin-angiotensin system in the control of arterial blood pressure and salt appetite in rats has been demonstrated[5]. The treatment with losartan reversed the blood pressure increase. Central angiotensin II AT_1 and AT_2 receptors mediated chronic intracerebroventricular (icv) ANGII-induced drinking in rats fed with high sodium chloride diet from weaning[6]. The increased release of atrium natriuretic factor (ANP) from the axons of neurons terminating on the neurons of the drinking response by stimulation of ANP receptors would inhibit the stimulatory response evoked by the action of ANGII on its receptors on these same effectors neurons[7]. AVP is

Corresponding Author: Dr. Wilson Abrão Saad, School of Dentistry, Department of Physiology, Paulista State University, UNESP, Rua Humaitá, 1680, Araraquara, SP 14801-903, Brazil
Tel/Fax: +55 16 201-6488

important in the renal regulation of fluid and electrolyte balance[8,9] including the septum[10,11]. The pharmacological properties of the AVP receptors in the lateral septal area (LSA) are similar to peripheral pressoric V_1-type receptors[12-14]. The central administration of the AVP antagonist, $d(CH_2)_5$-Tyr (Me)-AVP has been shown to be a potent antagonist of AVP at the V_1 receptors[15,16]. Central injection of angiotensin II (ANG II) elicits prompt and pronounced responses such as increased blood pressure, thirst, sodium appetite and the release of vasopressin[17]. Electrical stimulation of the basal forebrain causes the release of AVP and a prolonged thirst[18]. Application of the non-peptide type 1 angiotensin antagonist DuP753 (losartan) blocks the ANG II-induced depolarization in the Supraoptic Nucleus (SON). In contrast, application of the type 2 antagonist PD123177 was ineffective in blocking this response[19].

Systemic or central administration of Angiotensin II (ANG II) causes an increase in AVP secretion[20,21]. Multi neuronal pathways are involved the AVP-secretion in response to ANG II. There is no evidence in literature concerning the interaction of V_1, V_2 and AT_1; AT_2 receptors of the LPO influence the AVP effect on the sodium balance changes. Therefore it was investigated if a potent, selective V_1 and V_2 competitive AVP antagonist and selective antagonists AT_1 and AT_2 of ANG II receptors, respectively, influence AVP systems of the LPO on the central control of sodium balance.

MATERIALS AND METHODS

Subject: Male Holtzman rats weighing 250-300 g were used. The Medical Ethics Committee of the Universidade Estadual Paulista UNESP approved all protocols in this study. The animals were housed in individual stainless steel cages. Standard Purina pellets (Na^+ content 5 nM/100 g), tap water solution were available *ad libitum* unless otherwise noted. Temperature was maintained at a constant 23±2°C, with a 12:12 h light-dark cycle (light on at 0730). The experiments were performed between 0900 and 1000 h.

Surgical procedures

Cerebral cannula: After an acclimatization period of 7 days, the animals were maintained under tribromoethanol (200 mg kg^{-1} body weight, intraperitoneal) anesthesia and placed in a Kopf stereotaxic instrument. The skull was leveled between bregma and lambda. Stainless guide cannula (0.6 mm outer diameter, 0.33 mm inner diameter) was implanted into the LPO using the following coordinates: 0.4 mm caudal to bregma, 1.2 mm lateral to midline and 8.2 mm below the duramater. The cannula was secured to the top of the skull with dental cement and fastened with two screws. The insertion of a close fitting stylet kept the lumen free of debris and clots. A prophylactic dose of penicillin (30,000 IU) was administered three days before and three days after surgery.

Central drugs injection: Arginine vasopressin was purchased from Sigma (St. Louis, MO) and dissolved in saline (0.15 M NaCl) at 80 nM/0.2 μL CGP 42112A and losartan were purchased from DuPont Merck (Wilmington, DE) and dissolved in saline (0.15 M NaCl) at 160 nM/0.2 μL $d(CH_2)_5$-Tyr (Me)-AVP (AAVP) (160 nM/0.2 μL) and [Adamanteanacetyl^1, 0-ET-$_D$-Tyr^2, Val^4, Aminobutyryl^6, Arg^{8,9}]-AVP (ATAVP) (160 nM/0.5 μL and 220 nM/0.2 μL dissolved in 0.15M NaCl) purchased from Bachem (Torrance, CA). Injections into LPO were made using 10 μL Hamilton syringes connected by polyethylene tubing (PE-10) to 30-gauge injection cannulas. At the time of testing, the obturator was removed and the injection cannula introduced into the chronically implanted guide cannula. The injection cannula was 0.2 mm longer than the guide cannula. The injection volume was 0.2 μL delivered over 20 to 30 sec. After injection, the obturators were replaced and the rats were placed back into the cage.

Sodium chloride intake (0.5 M): AVP (80 nM/0.2 μL) or vehicle was injected into the LPO and 0.5 M NaCl was offered. AAVP and ATAVP, losartan and CGP 42112A, were injected into the LPO at the dose of 160 nM/0.2 μL, 15 min before AVP. The doses utilized in this experiment were based in previously experiment of our laboratories.

Sodium chloride intake (0.5 M) was recorded every 30 min over a 2 h period using burettes graduated in mL (0.1 mL divisions). Each animal is maintained in individual metabolic cages. Most of the sodium chloride drinking occurred within 15-30 min but we observed for a 2 h period.

Sodium excretion and urine volume recordings: Five days after brain surgery, catheters (PE-50 polyethylene tubing) was inserted under 2,2,2-tribromoethanol anesthesia (20 mg/100 g body weight) into the superior vena cava via the right external jugular vein and externalized between the scapulae. Two days after surgery, animals were submitted to the experimental session. After 12 h, the catheters were connected to 10 mL syringes driven by an apparatus infusion pump (Infusion Pump-BI 2000-Insight Equipments Ribeirão Preto SP Brazil). Intravenous infusion of hypotonic saline (0.08 M at 1.5 mL h^{-1}) was used to promote continuous

urine flow and 3 h were allowed for equilibration. Urine excretion was recorded at 30 min intervals for 2 h after the injection of the AVP and ANG II antagonists (160 nM) and AVP (80 nM). Urine volume was gravimetrically measured.

Experimental procedures

Effects of AAVP, ATAVP, losartan and PD 123319, injected into the LPO before AVP injection into the LPO on 0.5 M NaCl intake, sodium excretion and urine output: Following a recovery period of at least 5 days, Holtzman rats with cannulae implanted into the LPO unilaterally were randomly assigned to one of eight treatment conditions: vehicle+vehicle, vehicle+AVP, AAVP+AVP, ATAVP+AVP, AAVP+ATAVP+AVP, losartan+AVP, CGP 42112A+AVP, LOS+CGP+AVP. Antagonists or vehicle pretreatment were administered into the LPO, fifteen minutes before AVP injections into LPO. Sodium chloride intake (0.5 M), urine volume and sodium renal excretion were recorded at every 15 min for 120 min following AVP administration.

Histology: At the end of the experiments, the animals were anesthetized with ether. The brains were removed, fixed in 10% formalin, frozen to -25°C and cut into 20-30 μm sections. Only animals in which the injection was placed in the intermediate part of LPO were used in this study.

Statistical analysis: The results are reported as means±SEM. The results were statistically examined by using ANOVA, followed by comparisons between individual means using Student-Newmans-Keuls post-hoc test. Differences at the 5% level ($p<0.05$) were considered significant.

RESULTS

Effects of AAVP, ATAVP, losartan and CGP42112A, injected into the LPO before AVP injection into the LPO on 0.5 M NaCl intake: AVP stimulation of the LPO increased the 0.5 M NaCl intake compared to controls values ($p<0.01$). AAVP and ATAVP injected into the LPO produced reduction of the 0.5 M NaCl intake responses elicited by LPO administration of AVP ($p<0.01$ and $p<0.05$, respectively). The 0.5 M NaCl drinking behavior was blocked when a combination of AVPA+ATAVP was injected into LPO previously to AVP ($p<0.001$). Both the AT_1 and AT_2 ligands administered into the LPO decreased in 0.5 M NaCl intake induced by AVP injected into the LPO, but losartan ($p<0.01$), was more effective than CGP 42112A ($p<0.05$) in the AVP response. Losartan+CGP injected into the LPO previously to AVP, blocked the increases in 0.5 M NaCl induced by AVP (Fig. 1).

Effects of AAVP, ATAVP, losartan and CGP42112A, injected into the LPO before AVP injection into the LPO on sodium output: AVP injection into LPO increased sodium renal excretion vs control ($p<0.001$), which was reduced by previous injection of AAVP and ATAVP ($p<0.001$) and ($p<0.05$), respectively. Prior administration of a combination of V_1 and V_2 antagonists abolished the effect of AVP on sodium output ($p<0.001$). Previous injection of losartan and CGP42112A also decreased the natriuretic effect of AVP ($p<0.05$). The injection of losartan with CGP42112A abolished the effect of AVP on sodium output ($p<0.001$) (Fig. 2).

Effects of AAVP, ATAVP, losartan and CGP42112A, injected into the LPO before AVP injection into the LPO on urine volume excretion: The AVP injected into LPO produced an antidiuretic effect vs control ($p<0.05$), which was decreased by prior administration into LPO of the ATAVP ($p<0.05$). The AAVP produced no change in the antidiuretic effect of AVP ($p>0.05$). Prior administration of losartan and CGP 42112A into LPO produced a decreased in the inhibitory effect of AVP on urinary volume ($p<0.05$). Prior administration of losartan in combination with CGP 42112A into LPO abolished the inhibitory effect of AVP on urinary volume ($p<0.001$) (Fig. 3).

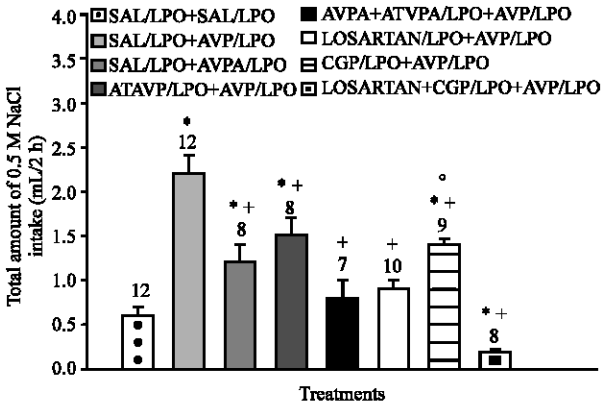

Fig. 1: Sodium chloride intake (0.5 M) (mL/2h), in rats treated with injections of vehicle (0.15 M NaCl) or AVP Effect of previous injection of AAVP, ATAVP, AT_1 and AT_2 receptor antagonists). The results are expressed as mean±SEM. *Different from vehicle. +Different from AVP. °Different from Losartan ($p<0.05$) tested by Newman-Keuls test). In parenthesis the number of rats

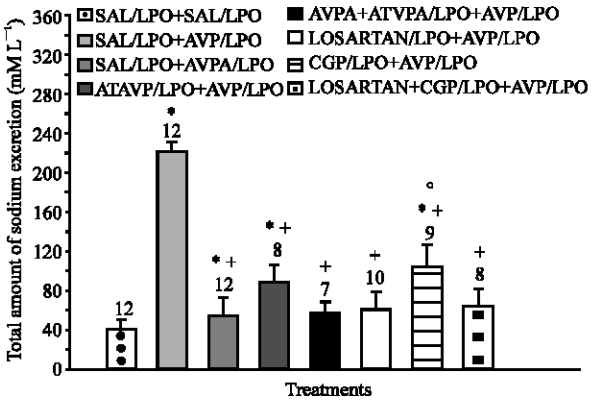

Fig. 2: Sodium renal excretion (mM/L/2h) in rats treated with injections of vehicle (0.15 M NaCl) or AVP. Effect of previous injection of AAVP, ATAVP, AT_1 and AT_2 receptor antagonists). The results are expressed as mean±SEM. *Different from vehicle. +Different from AVP. °Different from Losartan ($p<0.05$) tested by Newman-Keuls test). In parenthesis the number of rats

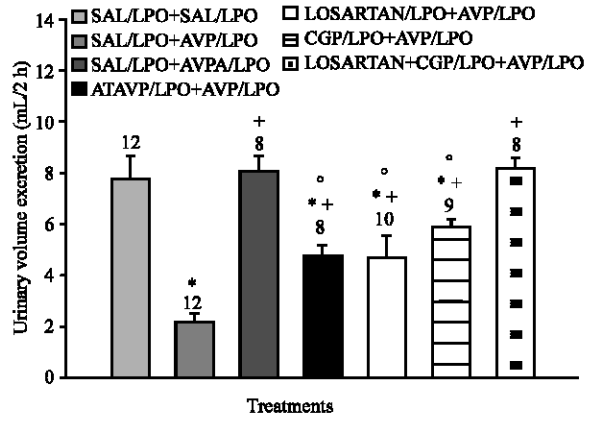

Fig. 3: Urinary volume excretion (mL/2h) in rats treated with injections of vehicle (0.15 M NaCl) or AVP. Effect of previous injection of AAVP, ATAVP, AT_1 and AT_2 receptor antagonists). The results are expressed as mean±SEM. *Different from vehicle. +Different from AVP. °Different from AVPA ($p<0.05$) tested by Newman-Keuls test). In parenthesis the number of rats

DISCUSSION

In the present experiments the natriogenic effects induced by AVP are mediated primarily by V_1 receptors of the LPO however; the V_2 receptors also decreased de effect of AVP in sodium intake with a less intensity. The sodium drinking behavior was blocked when a combination of AAVP+ATAVP was injected into LPO previously to AVP. AVP injected into LPO increased sodium renal excretion, which was inhibited by previous injection of AAVP. The AVP-induced increase in urinary sodium loss was abolished in animals that received a combination of AVP and AAVP, although the antidiuretic effect persisted[22]. Both the AT_1 and AT_2 antagonists administered into the LPO elicited a dependent-concentration inhibition in the sodium intake induced by AVP injected into the LPO, but losartan was more effective than CGP 42112A in the AVP response. The injection of AT_1 and AT_2 ANG II antagonists-receptors also decreased the natriuretic effect of AVP. Prior administration of losartan and CGP 42112A into LPO produced a decreased in the inhibitory effect of AVP on urinary volume. Prior administration of losartan in combination with CGP 42112A into LPO abolished the inhibitory effect of AVP on urinary volume. LPO influences sodium intake and sodium output[23]. The V_1 receptors of the LPO are implicated in the central control of sodium output[24]. Other important finding is that the V_1 receptors of LPO are implicated most in regulation of sodium ingestion and sodium excretion and that the V_2 receptors of the LPO are most implicated in the antidiuretic effect of vasopressin. The precise mechanism of AVP action has not been defined precisely yet. By these results we can postulated that the mechanism of the control of the one of the most important ion of the body (sodium), is that AVP stimulated the osmoreceptors of the LPO by utilizing the V_1 and the V_2 receptors, acting first in the V_1 inducing sodium intake and sodium excretion. The V_2 receptors are implicated in water loss.

Several studies have suggested that either a small number of losartan-sensitive binding sites are revealed when AT_2 receptors are bound, or that PD123319 and CGP42112A might act to increase the affinity of losartan at AT_1 receptors[25,26]. AVP can also influence the central cathecholamines metabolism, as well as angiotensin on specific brain regions including limbic system and hypothalamus[27]. The data of the present experiments showed that AT_1 and AT_2 receptors of the LPO are implicated in the effect of AVP. The AT_1 receptors are more sensitive than AT_2 in the control of sodium balance induced by AVP injected into LPO. When the body needs an emergency mechanism to establish the equilibrium of the extra cellular fluid both the AT_1 and AT_2 receptors are putted in action by AVP. Recently it has been demonstrated that in the LPO has extra cellular concentration of amino acid neurotransmitters. In the LPO calcium and nerve impulse dependent increases of arginine, glutamate and aspartate[3]. Localization and pharmacological characterization of high affinity sites for vasopressin and oxytocin in the rat brain by light

microscopic autoradiography has been demonstrated[28]. We recently demonstrated the participation of the AVP receptors in the brain areas in the regulation of water intake[27,29]. Biological bases of salt appetite have not been understood completely yet. Many papers suggest that NaCl consume cannot be due to excitatory stimulus associated with sodium deficiency, but due a complex interaction between excitatory and inhibitory stimulus[17]. The ablation of the LPO in rats abolished completely the water intake induced by cellular dehydration induced by administration of hypertonic saline as demonstrated first of all by Blass and Epstein[1], (Blass and Epstein animal's). We demonstrated that electrolyte lesion of the LPO abolished completely the water ingestion induced by cellular dehydration, hypotension, hypovolemia and deprivation[2]. Reflex and behavior answer are necessary to correct the balance and maintenance of corporal fluid homeostasis. Reflex mechanism use the autonomic nervous system and endocrine answers (as angiotensin and AVP) to change the water and sodium loss on dehydrating situations. Behavior answers include water and sodium ingestion from food and liquids. The present data showed that AVP administration into LPO of satiated rats increases sodium ingestion. AVP administration into ventral region of third ventricle at proptic area also shows an influence in sodium ingestion[30]. A new finding is that AVP, at administered dose at this work injected into the LPO play a role in the natriuretic effect via V_2receptors (central effect) also as in the kidney. It is suggested that combination of V_1 and V_2 antagonist can have therapeutic effects in certain types of chronic renal failure[10]. The contribution of V_2 receptor of the LPO in these effects can bee postulated.

The present experiments demonstrated that natriorexigenic effects induced by AVP are mediated primarily by LPO AT_1 receptors. However, doses of losartan were more effective when combined with CGP42112A than when given alone. AVP injected into the LPO induces sodium intake. Sodium intake is an important ingestive behavior to the control of body fluid homeostasis. This behavior is critical in correcting the reduction of extracellular fluid volume. Sodium intake is also important to the correction of intracellular dehydration that ensues during a decrease in the osmolality body fluid. The activation and coordination of reflexive and behavioral responses that maintain body fluid homeostasis require the integrated action of the central nervous system (CNS)[30,31]. Dehydration stimulates thirst and water intake. This event stimulates secretion of AVP, which leads to limitation of further renal water loss, in order to replace water deficit. After dehydration the thirst and AVP secretion are inhibited[32].

We conclude that the LPO is important area of the central nervous system in regulation of fluid and salt balance utilizing an interaction between vasopressinergic and angiotensinergic pathways.

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