Arginine vasopressin (AVP)

see also Syndrome of inappropriate antidiuretic hormone secretion.

Arginine vasopressin (AVP) is an antidiuretic hormone which is synthesized in the magnocellular neurons of the Supraoptic nucleus (SON) and paraventricular nucleus (PVN) in the hypothalamus. AVP is transported to the posterior pituitary through the axons, released into the systemic circulation, and plays a pivotal role in water balance by promoting reabsorption of free water through the V2 receptor in kidney. The release and synthesis of AVP are mainly regulated by plasma osmolality (or serum Na) in physiological conditions. The osmoregulation of the AVP neuron system is so precise that only 1–2% increases in serum Na levels significantly stimulate its release as well as the transcription of AVP gene in the SON and PVN [1].

Arginine vasopressin (AVP) is a common second-line or third-line vasopressor used in critically ill neurosurgical patients. Neurosurgical indications include hyperdynamic therapy for vasospasm, maintenance of cerebral perfusion pressure in patients with intracranial hypertension, and prevention of hypotension in patients with sepsis

The actions of vasopressin are mediated by stimulation of tissue-specific G protein-coupled receptors (GPCRs) called vasopressin receptors.

Plays an important part in circulatory and water homoeostasis and is important in renal hemodynamic alterations, water retention, and cardiac remodeling in congestive heart failure (CHF). There are three AVP receptor subtypes V1a, V1b, and V2. All belong to the large rhodopsin-like G-protein-coupled receptor family. V(1a) antagonists improve water balance and cardiac hypertrophy in CHF and might be beneficial for the treatment of water retention and cardiac remodeling in CHF.

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Acting synergistically with CRH, vasopressin causes significant secretion of ACTH [2].

The pituitary stalk (also known as the infundibular stalk or simply the infundibulum), is the connection between the hypothalamus and the posterior pituitary.

The floor of the third ventricle is prolonged downward as a funnel-shaped recess, the infundibular recess, into the infundibulum, and to the apex of the latter the hypophysis or pituitary is attached.

It passes through the dura mater of the diaphragma sellae as it carries axons from the magnocellular neurosecretory cells of the hypothalamus down to the posterior pituitary where they release their neurohypophysial hormones, oxytocin and vasopressin, into the blood.

This connection is called the hypothalamo-hypophyseal tract or hypothalamo-neurohypophyseal tract.

A series of six neurosurgical patients receiving AVP infusions developed severe but transient diabetes insipidus (tDI) after cessation of AVP. No previous reports of this phenomenon in neurosurgical patients have been published. We reviewed the clinical histories, intensive care unit treatment,

medication administration records, and laboratory values of these patients and found recurrent elevated serum sodium and urine output and decreased urine specific gravity after discontinuation of AVP. Resolution of tDI occurred upon resumption of AVP or administration of desmopressin. Elevated serum sodium levels were often severe, resulting in worsened clinical outcomes. When AVP was resumed, tDI typically recurred if AVP was again tapered and discontinued. Routine administration of desmopressin was useful in controlling sodium levels until the tDI resolved.

Recognition of this phenomenon has caused us to change our clinical management of neurosurgical patients on AVP. We hypothesize that tDI is caused by downregulation of the V2 receptor mass in the renal distal convoluted tubule and collecting duct cells. When AVP is discontinued, patients develop nephrogenic tDI secondary to decreased V2 receptor binding, which explains why desmopressin is effective in correcting tDI. Future research includes a large prospective study to determine risk factors for tDI, its incidence, and its pathophysiology [3].

Carbamazepine release ADH or potentiate it.

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