

Vasopressin: Its current role in anesthetic practice

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Abstract

Vasopressin or antidiuretic hormone is a potent endogenous hormone, which is responsible for regulating plasma osmolality and volume. In high concentrations, it also raises blood pressure by inducing moderate vasoconstriction. It acts as a neurotransmitter in the brain to control circadian rhythm, thermoregulation and adrenocorticotrophic hormone release. The therapeutic use of vasopressin has become increasingly important in the critical care environment in the management of cranial diabetes insipidus, bleeding abnormalities, esophageal variceal hemorrhage, asystolic cardiac arrest and septic shock. After 10 years of ongoing research, vasopressin has grown to a potential component as a vasopressor agent of the anesthesiologist's armamentarium in the treatment of cardiac arrest and severe shock states.

Keywords: Cardiopulmonary resuscitation, hemorrhagic shock, operating room, septic shock, vasopressin

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Introduction

Vasopressin is a potent vasopressor that may be a useful therapeutic agent in the treatment of cardiac arrest, septic and several other shock states and esophageal variceal hemorrhage. Studies indicate that the use of vasopressin during cardiopulmonary resuscitation may improve the survival of patients with asystolic cardiac arrest.^[1] Vasopressin deficiency can contribute to refractory shock states associated with sepsis, hypovolemia, cardiogenic shock and cardiac arrest. Low doses of vasopressin can restore vasomotor tone in conditions that are resistant to catecholamines, with preservation of renal blood flow and urine output. It may also be useful in reducing bleeding and mortality associated with esophageal variceal hemorrhage. Terlipressin is a useful vasopressin analog that produces sustained increases in blood pressure in septic patients.

Physiology

Vasopressin is a nonapeptide synthesized as a prohormone in magnocellular neuron cell bodies of the paraventricular and supraoptic nuclei of the posterior hypothalamus. It is bound to a carrier protein, neurohypophysin, and transported along the supraoptic hypophyseal tract to the axonal terminals of magnocellular neurons located in the posterior pituitary. Synthesis, transport and storage take 1-2 hours. Normal plasma concentrations are < 4 pg/ml. It has a half-life of 10-35 minutes, being metabolised by vasopressinases. Vasopressin acts on V1, V2, V3 and oxytocin-type receptors (OTR).

V1 vascular receptors mediate vasoconstriction and are located on vascular smooth muscle. V1 receptors are found in the kidney, myometrium, bladder, hepatocytes, platelets, adipocytes and spleen. These G-protein coupled receptors activate phospholipase-C via Gq G-protein, which ultimately leads to an increase in intracellular calcium.

V2 receptors are predominantly located in the distal tubule and collecting ducts of the kidney. These G-protein coupled receptors activate adenylyl cyclase to increase cyclic adenosine monophosphate. This

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mobilizes aquaporin channels, which are inserted into the apical membrane of the renal collecting duct cells and endothelial cells. V2 receptors are responsible for antidiuretic effects of vasopressin. Their presence on endothelial cells induces the release of Von Willebrand Factor (VWF) and Factor VII: Coagulant (FVII: c). VWF protects FVII from breakdown in plasma and is important in binding platelets to the site of bleeding.

V3 receptors are found mainly in the pituitary. They are Gq-coupled G-protein receptors, which increase intracellular calcium when activated. They are thought to be involved in adrenocorticotrophic hormone release, responsible for the actions of vasopressin on the central nervous system, and may act as a neurotransmitter or mediator involved with memory consolidation or retrieval and body temperature regulation.^[2]

Vasopressin has equal affinity for OTR as oxytocin. Activation of these receptors raises intracellular calcium via the phospholipase C and phosphoinositide pathway. They are found predominantly on myometrium and vascular smooth muscle. In addition, they are located on vascular endothelial cells where they increase endothelial nitric oxide synthase activity, increasing nitric oxide, which is a potent vasodilator. It is postulated that OTR placement on vascular endothelium and their subsequent activation may account for vasopressin's selective response on different vascular beds.

The most potent stimulus for vasopressin release is an increased plasma osmolarity and severe hypovolemia.^[3] Central nervous input mediated by pain, stress, nausea, hypoxia, pharyngeal stimuli and endogenous and exogenous chemical mediators also increase vasopressin release.

Hyperosmolarity is sensed by both central and peripheral osmoreceptors. Central osmoreceptors (subfornical organ nuclei) located outside the blood-brain barrier monitor systemic plasma osmolality. Peripheral osmoreceptors are found in the portal veins and give early warning of ingested food and fluid osmolality. Signals are transmitted via the vagus to the nucleus tractus solitaries, area postrema and ventrolateral medulla and finally to the paraventricular nuclei and supraoptic nuclei where vasopressin is manufactured in the magnocellular neuron cell bodies. Osmolarity is finely controlled in the range of 275-290 mOsm/kg.

Plasma volume and the resultant change in arterial pressure are less sensitive controllers of vasopressin release. A 20-30% reduction in mean arterial pressure

(MAP) is needed to induce a response. An 8-10% reduction in plasma volume detected by arterial stretch receptors is required to induce an exponential increase in vasopressin release. A reduction in plasma volume increases the sensitivity of the osmoreceptors and vice versa. A decrease causes an increase in plasma norepinephrine and renin concentrations whereas the plasma vasopressin concentration does not increase until MAP decreases.

Acetylcholine, histamine, dopamine, prostaglandins, angiotensin and other catecholamines directly stimulate vasopressin release. High PaCO₂ stimulate carotid body chemoreceptors and thus increase vasopressin levels. Opioids, γ -aminobutyric acid and atrial natriuretic peptide inhibit vasopressin by α 1-adrenoreceptors whereas the α 2-adrenoreceptors and β -adrenergic receptors inhibit vasopressin and oxytocin release.

A very similar substance, lysine vasopressin (LVP) or lypressin, has the same function in pigs and is often used in human therapy. Terlipressin (triglycyl-lysine vasopressin) became popular in 1990s because it has a prolonged duration of action. It is a prodrug and is converted to the lysine vasopressin in the circulation after the N-triglycyl residue is cleaved by endothelial peptidases. This results in a slow release of the vasoactive LVP. Ornipressin is a derivative of arginine vasopressin acting specifically on the V1 subtype receptors. In other words, it is a derivative that mimics the effects of arginine vasopressin on vascular smooth muscle; however, it has no antidiuretic effect. Desmopressin acetate (1-deamino-8-D-arginine vasopressin or dDAVP) is a synthetic analog of the antidiuretic hormone arginine vasopressin that was initially devoted to the treatment of central diabetes insipidus. Through a direct action on endothelial V2 receptors, desmopressin raises factor VIII and VWF plasma concentrations in healthy volunteers. The increase in VWF concentrations is related to a release from endothelial intracellular storage sites and not to *de novo* synthesis. Thus, this effect is rapid, transient and diminishes with repeated injections at short intervals (tachyphylaxis). This agent also transiently increases t-PA and prostacyclin plasma levels. It has additional but unclear effects on hemostasis.^[4]

Pharmacology

Exogenous vasopressin (8 arginine vasopressin) is presented as sterile aqueous solution of synthetic vasopressin for intravenous, intramuscular and subcutaneous administration. It is not protein-bound and has a volume of distribution of 140 ml/kg. The plasma half-life of vasopressin is 24 minute. It is cleared by renal elimination (65%) and metabolism (35%) by tissue peptidase.

Therapeutic Use

Septic shock

In septic shock there are biphasic changes in vasopressin concentration with high concentration in the early part to maintain organ perfusion, but as the shock state progresses the concentrations decrease. Possible explanations include exhaustion of stores and autonomic nervous system dysfunction. Large doses of norepinephrine are inhibitory to vasopressin release. Dünser and colleagues published the largest randomized prospective controlled study in 2003. In this study 48 patients with catecholamine resistant vasodilatory shock were prospectively randomized to receive a combined infusion of vasopressin 4 IU/hour and norepinephrine or norepinephrine alone to maintain a MAP above 70 mmHg. The vasopressin group showed a significant increase in MAP, cardiac index, systemic vascular resistance index and left ventricular stroke work index as well as reduced norepinephrine requirements and heart rates. Compared with norepinephrine group, there was better preservation of gut mucosal blood flow and a significantly less incidence of tachyarrhythmias.^[5] The vasopressin and septic shock trial (VASST) was the first multicenter, blinded, randomized trial comparing low-dose vasopressin with norepinephrine in 778 patients with septic shock.^[6] The use of vasopressin did not reduce mortality but was shown to be as safe as norepinephrine.

After cardiovascular function has stabilized and norepinephrine support could be withdrawn to dosages <0.2–0.3 µg/kg/min, vasopressin was slowly withdrawn in most studies. Administered as a supplementary vasopressor agent, vasopressin seems to be capable of bridging the phase of advanced cardiovascular failure and preventing a vicious cycle of high-dose catecholamine therapy that may develop. The pivotal issue of clinical research on the use of vasopressin in septic shock must therefore not be the question 'Can vasopressin replace norepinephrine therapy?' but be the question 'Can the supplementary infusion of vasopressin in addition to norepinephrine improve the quantitative and qualitative outcome of advanced septic shock?'

Since low vasopressin plasma concentrations have been found in septic shock patients, vasopressin infusion was first proposed as hormone replacement therapy rather than vasopressor therapy.^[7] A recent study demonstrated that plasma vasopressin levels were almost always increased in the initial phase of septic shock, and decreased thereafter. Accordingly, the relative vasopressin deficiency and consequently the suggested indication for vasopressin hormone replacement was found only in one-third of late septic shock patients.^[8]

Additionally, the increase in arterial pressure during vasopressin infusion occurs independently of plasma vasopressin concentrations.^[9] Vasopressin therapy at dosages from 0.01 to 0.1 U/min increases plasma concentrations to 100–250 pg/mL,^[9] which is 50-fold to 100-fold higher than the vasopressin levels reported in patients with cardiogenic shock and septic shock states who still respond to conventional therapy.^[10] Therefore, institution of vasopressin infusion in advanced septic shock should not be guided by endocrinological, but by hemodynamic indications.

Whether the possibility of 'bridging' vasopressin with catecholamine therapy in advanced septic shock patients may have a benefit/risk ratio within a tolerable clinical range that may improve quantitative and qualitative patient outcome, can only be determined by a large, prospective, randomized study. Such a study will finally also answer the question of whether positive effects of vasopressin on macro circulatory parameters are outweighed by possible adverse effects on the microcirculation system, the hepatosplanchnic system or the coagulation system. While no data of supplementary vasopressin infusion in advanced septic shock on patient outcome exists, the infusion of vasopressin in addition to catecholamine vasopressor agents in order to reduce high, potentially toxic adrenergic vasopressor dosages can only be recommended as a last-resort therapy.^[11]

Hemorrhagic shock

Vasopressin should be used at a dosage of 0.2–0.4 IU/kg bolus injection followed by 0.8 IU/kg/min continuous infusions depending on the severity of shock stage. Morales *et al* successfully treated two patients suffering from gastrointestinal hemorrhage and refractory hypovolemic shock with a vasopressin infusion of 0.001 and 0.004 IU/kg/min, respectively.^[12]

Vasodilatory shock after cardiac surgery

Comparable to septic shock vasodilatory shock after cardiac surgery is characterized by poor response of vascular smooth muscles to circulating catecholamines. A supplementary vasopressin infusion 0.6–6 U/hour has been repeatedly observed to reverse advanced vasodilatory shock in post cardiectomy patients, although some authors have advised caution with the use of vasopressin in cardiac surgery because of possible reductions in cardiac output.^[13]

A retrospective analysis in 41 postcardiotomy shock patients found improved myocardial performance during vasopressin therapy. Vasopressin given as a continuous infusion not exceeding 6 U/h proved to be

devoid of adverse effects on the heart in these patients with catecholamine-resistant postcardiotomy shock. The observation of a significant reduction in heart rate, vasopressor and inotropic support even suggests a substantial improvement in myocardial performance. These findings are supported by the observation of a significant decrease of cardiac enzymes and the spontaneous cardioversion of tachyarrhythmia into sinus rhythm in nearly half of patients with new-onset tachyarrhythmia. Therefore, vasopressin may provide an important additional treatment option in patients receiving high vasopressor and inotropic support after cardiac surgery.^[14]

Other vasodilatory shock states

Anaphylactic shock

The predominant pathophysiological mechanisms of cardiovascular failure in anaphylactic shock are vasodilatation and hypovolemia resulting from increased endothelial permeability. All authors observed rapid hemodynamic stabilization after vasopressin bolus of 2-10 IU, followed by a continuous infusion of 40 IU over 60 min, in one report.^[15] Tsuda *et al.*^[16] investigated the reversal of vascular relaxation induced by histamine in human internal mammary arteries, and found that epinephrine could only partly reverse histamine-induced vasodilatation, whereas vasopressin, methylene blue and drugs involved in the inhibition of nitric oxide and prostaglandin generation caused by a complete reversal of vascular relaxation.

Hypotension during spinal or epidural anesthesia

Supplementary opioids and α_2 -receptor agonist's drugs are being frequently used during the administration of spinal/epidural block. These drugs inhibit endogenous vasopressin release. It was hypothesized that the administration of a vasopressin analog may mimic the physiological adaptation to sympathetic block induced by epidural anesthesia.^[17]

Acute brain injury

In view of systemic vasoconstrictive and local vasodilatory effects on arteries of the circle of Willis, vasopressin has also been used to ensure adequate cerebral perfusion pressure in the perioperative setting. Yeh *et al* reported on the successful administration of vasopressin (2.4IU/h) in a patient suffering from spontaneous cerebellar and subarachnoid hemorrhage with advanced vasodilatory shock, neurogenic pulmonary oedema, and myocardial dysfunction.^[18] Similarly, Bradley *et al*^[19] administered terlipressin (0.25 mg), a vasopressin analog with a pressor effect for approximately 4-6 h, in a hemodynamically unstable patient with subarachnoid hemorrhage.

Vasopressin is a safe adjunct vasopressor to phenylephrine for hypervolemic, hemodilutional, and hypertensive treatment in patients with subarachnoid hemorrhage. Vasopressin does not induce vasospasm or aggravate electrolyte disorders.^[20]

Hormone replacement in brain-dead organ donors

Vasopressin has been administered in brain-dead organ donors since 1986. The 1 IU bolus followed by 0.5-4 IU/hr dose of vasopressin, not only treated diabetes insipidus, but resulted in significant reductions in vasopressor and inotropic drug requirements.^[21]

Perioperative hypotension in pts chronically treated with ACE inhibitors

As angiotensin II is a physiological stimulator of vasopressin release, perioperative hypotension in pts chronically treated with ACE inhibitors has been speculated to be caused by vasopressin deficiency. Studies showed that 1 mg terlipressin was effective in rapidly correcting refractory hypotension without major side-effects.^[22]

Vasodilatory shock after pheochromocytoma extirpation

In view of extensive adrenergic receptor downregulation, vasopressin therapy seems to be particularly beneficial if catecholamine resistant vasodilatory shock develops after pheochromocytoma surgery. Augoustides *et al* successfully applied AVP in patients with catecholamine resistant vasoplegic shock after the urgent resection of a massive pheochromocytoma.^[23]

Hypotension caused by carcinoid crisis

Vasopressin infusion has been reported to be an alternative to sympathomimetic vasoconstrictors, which are considered relatively contraindicated in pts with carcinoid tumors because they may exaggerate further peptide release via adrenergic receptor stimulation.^[24]

Cardiac arrest (CPR)

The use of epinephrine in resuscitation is universal, but there is little evidence to show that vasopressin improves survival in humans. Theoretically vasopressin is an attractive alternative to epinephrine during cardiopulmonary resuscitation because it significantly improves total cerebral and myocardial blood flow and causes a sustained increase in MAP compared with maximal dose of epinephrine.^[25] Wenzel and colleagues performed a multicenter randomized double-blinded trial in 1186 patients who had an out-of-hospital cardiac arrest.^[26] They were randomly

assigned to receive either 40 IU of vasopressin or 1 mg of epinephrine during resuscitation. In the asystolic group, significantly more patients reached hospital who received vasopressin, compared with those who received epinephrine. 4.7% of the vasopressin group were discharged from the hospital compared with 1.5% in the epinephrine group. No difference was found between the groups in patients with pulseless electrical activity or ventricular fibrillation cardiac arrests. There is a suggestion that vasopressin may work better than epinephrine in hypoxic, acidotic conditions. It also suggests a better outcome in the vasopressin groups if there was delayed or prolonged resuscitation. In the contrary, another randomized control trial found that repeated doses of vasopressin during cardiac arrest did not improve survival rates compared with repeated doses of epinephrine.^[27]

Because the effects of vasopressin have not been shown to differ from those of epinephrine in cardiac arrest, the current international guidelines for cardiopulmonary resuscitation (2010) recommended that 1 dose of vasopressin 40 units IV/IO may replace either the first or second dose of epinephrine in the treatment of cardiac arrest.

Esophageal variceal bleeding

Vasopressin acting via V1 receptors reduces portal blood flow, portal systemic collateral flow and variceal pressure. Its side-effects include increased peripheral resistance; reduced cardiac output and decreased coronary blood flow, which is reduced if combined with glyceryl trinitrate. A Cochrane review found that terlipressin; a prodrug of vasopressin produced a relative risk reduction in mortality from variceal hemorrhage of 34% compared with placebo.^[28] The i.v. dose is 2 mg 4 hourly.

Bleeding abnormalities

Vasopressin acts via extrarenal V2 receptors to increase predominantly FVII: c and VWF. These actions are useful in certain types of Von Willebrand disease and mild forms of hemophilia. It can also be used in pts with impaired platelet function due to drugs such as aspirin or renal failure. The exact mechanism of its effect in these situations is not fully understood, but the increase in FVIII levels which allows activation of FX and the more efficient activation of platelets are all important.^[29]

Central diabetes insipidus

Clinically, the patient produces vast quantities of dilute urine. Key feature is that urine osmolality is inappropriately low compared with plasma osmolality.

Desmopressin (DDAVP) can reduce polyurea, nocturia and polydypsia in these patients.^[30] It is given nasally, sublingually, i.m, or in critical care setting i.v.

Adverse Effects/Safety

Severe skin necrosis after extravasation of low-dose vasopressin administered for catecholamine resistant shock has been reported and peripheral administration of low-dose vasopressin infusions should be discouraged.^[31] It is thrombogenic, acting via V2 receptors. Anaphylaxis, bronchospasm, urticaria and ischemia of the gastrointestinal tract have been reported with clinical use. Reduction in cardiac output and systemic oxygen delivery, impairment in gut mucosal and hepatic oxygenation, increases in aminotransferases activities and bilirubin concentrations and reduction in platelet count have also been observed. In addition, sodium concentration and plasma osmolality should be regularly evaluated, although to date there is no evidence that vasopressin analogs are linked to antidiuresis, water retention or renal impairment in septic shock patients. Conversely, several studies reported an improvement in renal function as well as advantages over sole catecholamine therapy in patients at risk for acute renal failure.

Cost-effectiveness

Cost is an issue, which might have limitations into ongoing studies on the use of vasopressin as an alternative to adrenaline as 1 ampoule (20 U) of vasopressin costs almost 70 times more than 1 ampoule of adrenaline. However, since vasopressin and epinephrine combined therapy improves the quality of life in patients of cardiopulmonary resuscitation,^[32] it can be assumed that this would help in lowering the overall long-term cost in patient care in the combined therapy group compared against the hidden cost that would be needed for treatment and rehabilitation of long-term morbidity in the adrenaline group.

In a recent study by Stiell *et al* of in-hospital CPR, 87% of the patients in the vasopressin group also received epinephrine^[32] and the usefulness of the combination regimen was demonstrated by clinical observations such as significantly improved coronary perfusion pressure,^[33] increased likelihood of restoration of spontaneous circulation^[34] and 24-hour survival rates.^[35] No randomized control trials have yet been done to determine the cost-effectiveness of vasopressin.

Future

Clinical and experimental studies certainly support

the beneficial effects of low-dose vasopressin infusion in vasodilatory shock. Nevertheless, is an increase in arterial pressure, and perhaps in urine output, sufficient to support the use of vasopressin in all pts with septic shock? Although recent animal studies have suggested improved outcomes in animals treated with vasopressin, no clinical study including the VASST, has yet demonstrated reduced mortality in patients treated with vasopressin. Now, should vasopressin be considered as a vasopressor therapy, as endocrine support or both? In hypotensive septic shock, the catecholamine α 1-adrenergic receptors may be desensitized or downregulated to standard catecholamine vasopressors, limiting their vasopressor activity. Because vasopressin binds to its own V1 vascular receptor, it can still act to restore vascular tone even if α 1-receptors are downregulated. Vasopressin reduces catecholamine requirement, but there is no suggestion to use it as a conventional agent and titrated to arterial pressure. Rather it should be used at low fixed dosages. So, if we are using vasopressin more as endocrinological support, should we only be giving it to patients with low vasopressin concentrations? Sharshar and colleagues suggested that relative vasopressin deficiency occurred in only one-third of late septic shock patients.^[36] In addition, the effects of vasopressin on arterial pressure seem to occur regardless of the endogenous plasma vasopressin concentration. This observation suggests that vasopressin therapy may be beneficial in all patients with septic shock rather than only in those with low vasopressin concentration. Lin and colleagues recently proposed that a low vasopressin/norepinephrine ratio could predict the development of septic shock in emergency department patients with sepsis or severe sepsis, suggesting that the changes in vasopressin occur before shock develops, so vasopressin should perhaps be given early rather than as a last resort.^[37]

Vasopressin in combination with epinephrine can significantly increase hospital discharge rates in cardiac arrest victims. Vasopressin-catecholamine combination can improve hemodynamics in vasodilatory and hemorrhagic shock. However, its effects on patient outcome are yet to be established across the board. Nonetheless, in the perioperative setting vasopressin is being considered to be a potent adjunct vasopressor agent in advanced shock states that are unresponsive to conventional therapeutic strategies. More research is needed on the use of vasopressin, particularly in humans, to enable its full benefits as an alternative to adrenaline in cardiac arrest to be realized.

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