Vasopressin

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Introduction

Vasopressin, also known as arginine vasopressin or antidiuretic hormone, is an endogenous peptide hormone traditionally used to treat diabetes insipidus and acute oesophageal variceal haemorrhage. Recent reports describe its use in two clinical situations directly applicable to emergency medicine: in cardiac arrest as an alternative to epinephrine and as a vasopressor in treatment-resistant vasodilatory (septic) shock. This review describes the physiology of vasopressin and briefly outlines its use historically. It will also focus upon the use of vasopressin in cardiac arrest and vasodilatory shock and will discuss recent American Heart Association guidelines. Consideration will be given to its use in the ED.

Physiology

Vasopressin is a circulating endogenous nonapeptide hormone synthesized as a prohormone in the magnocellular neurones in the paraventricular and supraoptic nuclei of the hypothalamus. The hormone is stored in granules in the pars nervosa of the posterior pituitary. Release is tightly regulated and is influenced by many osmotic and non-osmotic stimuli (Table 1).

Key words: advanced cardiac life support, heart arrest, septic shock, vasoconstrictor agents, vasopressins.
Vasopressin

Only 10–20% of the vasopressin store in the posterior pituitary can be released immediately into the circulation. Further release occurs more slowly in response to appropriate stimuli.

Plasma vasopressin levels in fasted, hydrated humans are normally < 4 pg/mL. Small increases in plasma osmolality result in rapid increase in vasopressin release, and subsequent renal water retention, to return plasma osmolality to normal. Physiological regulation of osmolality and renal collecting-duct permeability occurs at plasma vasopressin concentrations in the range of 1–7 pg/mL. Vasoconstrictor effects occur at much higher concentrations, in the range of 9–187 pg/mL.

The rapid metabolism of vasopressin by vasopressinases in the liver and kidney results in a plasma half-life of 10–35 min. The physiologic effects of vasopressin are mediated by ligand binding to specific vasopressin receptors. These are serpentine receptors with seven transmembrane domains coupled to G protein-mediated second messenger systems. Major receptor subtypes, the tissues in which they are found and the principal effect of vasopressin acting at these receptors are summarized in Table 2. The principal physiologic effects of vasopressin are direct vasoconstriction of the systemic circulation, mediated by V1 vascular smooth muscle receptors, and as an osmotic regulator, mediated via renal V2 receptors.

Vasopressin has little effect on BP at physiologic concentrations but plays an important role in maintaining BP during hypovolaemia or in conditions of hypovolaemia causing hypotension. In contrast to most shock states in humans, in which appropriately high levels of vasopressin have been found, some patients with late vasodilatory septic shock have low plasma vasopressin levels. These patients demonstrate an increased sensitivity to low-dose vasopressin, and reinforce the importance of this potent vasoressor in maintaining perfusion pressure during shock states.

### Historical use of vasopressin

Vasopressin has been used over the past 50 years to treat neurogenic (central) diabetes insipidus. A newer agent, desmopressin (a longer duration of action, fewer side-effects and potentially better efficacy) has led to a decline in vasopressin usage in this condition. Vasopressin is used to prevent and treat postoperative abdominal distension, and is also used in abdominal radiography to remove interfering gas shadows.

Its use in the initial management of bleeding esophageal varices is well documented, mostly to control bleeding until sclerotherapy can be performed. However, the route of administration (peripheral venous line vs central venous line vs intra-arterial injection into superior mesenteric artery) and dosage is variable and controversial. Systemic vasoconstrictive effects are problematic. The morbidity associated with vasopressin use has led to the development of analogues with fewer adverse effects, such as terlipressin. Studies comparing the efficacy of intravenous somatostatin with vasopressin in controlling variceal bleeding demonstrate somatostatin to be at least as effective with fewer complications.

Other uses of vasopressin described include:

1. reduction in bleeding and blood transfusions during burn wound excision
2. treatment of upper gastrointestinal haemorrhage secondary to haemorrhagic gastritis
3. treatment of severe haematuria due to cyclophosphamide-induced haemorrhagic cystitis
4. reduction of blood loss and prevention of intra-operative hypotension during liver transplant
5. treatment of intractable placental bleeding in patients undergoing caesarian sections, and
6. treatment of refractory bleeding after uterine myoma resection

### Table 1. Stimuli of vasopressin release

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<td>Hormones/mediators</td>
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With late vasodilatory septic shock have low plasma vasopressin levels. These patients demonstrate an increased sensitivity to low-dose vasopressin, and reinforce the importance of this potent vasoressor in maintaining perfusion pressure during shock states.
Interest has been shown in its role to treat memory disorders.44

**Use of vasopressin in cardiac arrest**

The 2000 American Heart Association (AHA) Advanced Cardiac Life Support (ACLS) Guidelines on the use of pressors in the treatment of cardiac arrest recommend that ‘vasopressin should be considered an alternative pressor to epinephrine for the treatment of shock-refractory, VF-induced cardiac arrest in adults.’10 These guidelines and two recent reviews3 present the evidence supporting its use in this way.

Research in the last decade has identified endogenous vasopressin as an important hormone in cardiac arrest and CPR.10 Circulating vasopressin concentrations were raised in patients undergoing CPR, and levels in...
patients who had been successfully resuscitated were significantly higher than in non-survivors. A clinical study of 60 out-of-hospital cardiac arrest patients found significantly higher concentrations of vasopressin during CPR in those who survived, whilst plasma epinephrine and norepinephrine concentrations (both before and after epinephrine administration) were significantly higher in patients who died. Researchers concluded that endogenously released vasopressin may function as an adjunct vasopressor during cardiac arrest and CPR, and that plasma concentrations of vasopressin may play an important role in the outcome of CPR. Theoretically vasopressin is a desirable vasopressor for use in cardiac arrest and CPR, producing selective vasoconstriction of resistance vessels in non-vital tissues whilst preserving blood flow to vital organs (heart + brain).

### Vasopressin and laboratory models of CPR

Animal studies comparing the use of vasopressin with epinephrine during CPR in porcine models of VF cardiac arrest suggest that vasopressin produces better outcomes than epinephrine, both in terms of return of spontaneous circulation (ROSC) and neurological outcome. Vasopressin is more effective than epinephrine in increasing vital organ blood flow (myocardial and cerebral blood flow) during CPR and significantly increases coronary perfusion pressure. The effects of vasopressin on vital organ blood flow are of longer duration than those of epinephrine (4 min vs. 1.5 min) and significantly more vasopressin animals can be resuscitated. Laboratory studies indicate that the same dose of vasopressin can be given intravenously, endobronchially and intraosseously with similar plasma vasopressin levels, haemodynamic effects, coronary perfusion pressure and ROSC rates.

In animal studies of prolonged CPR, repeat doses of vasopressin result in higher arterial pressures and greater coronary perfusion pressures than epinephrine. The importance of coronary perfusion pressure is reflected in the finding that coronary perfusion pressure correlates with return of spontaneous circulation in human cardiac arrest. In a study using a porcine model to simulate prolonged advanced cardiac life support, all vasopressin animals had ROSC and full neurologic recovery (assessed clinically and by cerebral MRI), whilst all pigs in the epinephrine and saline group died. Vasopressin has greater efficacy than epinephrine in porcine models of cardiac arrest in the settings of epidural anaesthesia and hypothermia and hypovolaemic shock.

### Clinical investigations: vasopressin in cardiac arrest

There are to date only four published clinical studies comparing the use of vasopressin with epinephrine in cardiac arrest. They describe the outcome of 258 adult victims of cardiac arrest.

In a case series of eight patients with refractory cardiac arrest (six with ventricular fibrillation and two with pulseless electrical activity [PEA]) intravenous administration of 40 units of vasopressin led to increased arterial blood pressure and return of spontaneous circulation in all patients. Three patients survived and were discharged from hospital neurologically intact (38%). Of particular note was the use of vasopressin as rescue therapy; in all patients standard therapy with chest compressions, ventilation, defibrillation, and epinephrine had failed. The authors suggest that vasopressin can be more effective than epinephrine because of greater vasoconstrictor effect in the presence of hypoxia and acidosis, and because of longer duration of effect.

Another small case series of 10 patients describes response to vasopressin after prolonged (approximately 40 min) unsuccessful advanced cardiac life support. Four patients had a mean increase in coronary perfusion pressure of 28 mmHg, but no return of spontaneous circulation. Despite no improvement in survival, the increase in coronary perfusion pressure after such a prolonged period of unsuccessful resuscitation is surprising, and has not previously been seen with any other drug.

In a small prospective, randomized study of 40 patients with out-of-hospital, shock-refractory VF arrest, a significantly larger proportion of patients treated with vasopressin were resuscitated successfully and survived to 24 h compared with patients treated with epinephrine. There was a non-significant trend towards increased survival to hospital discharge in the vasopressin group (more than twice as many patients in the vasopressin group survived to hospital discharge).

The second, and largest, prospective randomized trial of vasopressin in cardiac arrest provides results that appear to conflict with those of the earlier clinical studies, however. Intravenous vasopressin (40 µg) is compared with epinephrine (1 mg) as the initial vasopressor in 200 patients with in-hospital cardiac...
Controversy: should vasopressin be used as an alternative to epinephrine?

The most striking difference between the two trials is the location of the patients when they had a cardiac arrest and the impact this has upon response times. In the larger study of in-hospital cardiac arrest patients the average time elapsed to initiation of CPR is 1.6 min and to ACLS measures is 2.8 min, at least 3 min earlier than in the out-of-hospital study. Among the in-hospital patients more than three times as many patients receive early ‘bystander’ basic life support. Vasopressin is used as the initial vasopressor in the in-hospital study, but as rescue therapy in the other studies. It is possible that the different response times and different timing of vasopressin administration influenced the benefits of the drugs. Vasopressin and epinephrine may be equally effective when used as the initial vasopressor in CPR commenced rapidly after the arrest.2

A second fundamental difference between the two studies is the mode of cardiac arrest. All of the patients studied in the out-of-hospital setting had VF as the initial rhythm.5 The cause of cardiac arrest in the in-hospital patients was VF in 18%, VT in 3%, pulseless electrical activity in 46%, and asystole in 30%.5 The in-hospital patients also had greater comorbidity from chronic diseases.5 It is possible that significant differences in the study patients contributed to the conflicting results, although the in-hospital study did not detect any trend in favour of vasopressin even in the VF/VT subgroup.4

The authors of the in-hospital study, Stiell and colleagues, strongly disagree with the AHA guidelines recommending vasopressin as an alternative to epinephrine.5 A closer look at the classes of recommendations within the AHA guidelines may help resolve this debate.5 The recommendations are that ‘vasopressin should be considered an alternative pressor to epinephrine for the treatment of shock-refractory, VF induced cardiac arrest in adults (Class IIb)’ and that ‘vasopressin is a promising adjunct or alternative pressor to epinephrine for the treatment of cardiac arrest (Class Indeterminate).’10

A Class IIb recommendation requires fair-to-good evidence with a majority of experts considering it to be an ‘optional or alternative intervention’.4 There is a small quantity of published data supporting the use of vasopressin in shock-refractory VF10 and the study by Stiell et al. shows that patients in VF given vasopressin as an alternative to epinephrine do not have a worse outcome or suffer additional harm.4

The Class Indeterminate recommendation implies that there is insufficient evidence or data from human prospective randomized trials to support the use of vasopressin in other forms of cardiac arrest, such as pulseless electrical activity or asystole.4 Vasopressin may be effective in these forms of cardiac arrest, but there is no evidence. There are also little human data to suggest that vasopressin harms.4 The results of Stiell et al. seem to support this conclusion.

Details of the discussions that led to the new AHA recommendations are recently published, and provide an insight into other areas of the vasopressin controversy.4,10 Although the human studies demonstrate improvement in haemodynamics, early return of spontaneous circulation, and survival to hospital admission in patients given vasopressin, there is no significant benefit in long-term survival or neurologic outcome demonstrated.10 This, however, highlights a more fundamental controversy in cardiac arrest epinephrine, the gold standard vasopressor in advanced cardiac life support, is no better than placebo for cardiac resuscitation in humans.4 Detrimental long-term survival or neurological effects are difficult to detect because baseline intact survival rate in the cardiac arrest population is only 3–5%.10 However, none of the existing preclinical or clinical studies comparing epinephrine with vasopressin favour epinephrine therapy.10

The beneficial effect of vasopressin in increasing cerebral blood flow during CPR may result in an increased risk of cerebral oedema or haemorrhage after return of spontaneous circulation.10 Vasopressin has a relatively long half-life (at least 5 min during CPR) and may cause persistent vasoconstriction.13 This may be advantageous in the resuscitation setting, with one dose of vasopressin easier to administer.
The future

The European vasopressor study is investigating the effects of 40 U vasopressin intravenously versus 1 mg epinephrine (given up to two times) in 1500 out-of-hospital cardiac arrest patients with ventricular fibrillation, asystole and pulseless electrical activity.\(^2\) The results of this study may better define the role of vasopressin in the management of cardiac arrests other than shock-refractory VF. The efficiency of a CPR intervention is measured by its effects on hospital discharge rate, long-term survival and neurologic outcome.\(^2\) Determining the effect of a drug given during CPR is, however, very difficult.\(^1\) A given patient population differs significantly due to variables such as age, race, standard of living, past medical history, rhythm at collapse and location of cardiac arrest.\(^2\) Healthcare services differ in terms of emergency medical services, response times and time to hospital resuscitation facilities.\(^2\) For a study to detect a significant increase in hospital discharge rates and/or neurological recovery it would require 15–20 000 patients, several years and millions of US dollars.\(^2,3\) Healthcare and research funding is decreasing worldwide, and commercial manufacturers of vasopressin have little interest in this drug as its patent expired 50 years ago. Therefore the primary endpoint of this study are the effects of vasopressin versus epinephrine on short-term survival measured as hospital admission.\(^2,3\)

The ideal vasopressor for CPR is a drug that is able to significantly increase myocardial and cerebral perfusion during CPR but which can, if necessary, be rapidly and titratably reversed immediately post-resuscitation.\(^2,3\) It is possible that the ideal vasopressor and the ideal dosing regimen for CPR is yet to be discovered, meaning that a combination of drugs may be necessary.\(^2,3\) Vasopressin looks promising, but more clinical data in human cardiac arrest are needed and the large European clinical trial ‘would have to come out significantly in favour of vasopressin in order to further change international CPR guidelines’.\(^2\)

Use of vasopressin in vasodilatory shock

Vasodilatory shock is characterized by hypotension secondary to peripheral vasodilation, and a poor response to vasopressor therapy.\(^7\) Whilst sepsis is the most frequent cause,\(^7,57,58\) vasodilatory shock can be the final common pathway in all forms of profound and long-lasting shock, including severe hypovolemic or cardiogenic shock.\(^7\) In all forms of vasodilatory shock that have been studied, plasma concentrations of catecholamines are markedly...
increased\textsuperscript{7,69,70} and the renin–angiotensin system is activated\textsuperscript{7,71}. The vascular smooth muscle, however, fails to constrict resulting in vasodilation and hypotension. Recent research has uncovered three common mechanisms that are responsible for the vasodilation and resistance to vasopressors seen in most types of vasodilatory shock: activation of ATP-sensitive potassium channels in the plasma membrane of vascular smooth muscle, activation of the inducible form of nitric oxide synthase, and deficiency of vasopressin.\textsuperscript{7}

**Vasopressin deficiency in vasodilatory shock**

In the initial phase of haemorrhagic or septic shock, vasopressin secretion increases, resulting in appropriately high plasma concentrations of vasopressin, which contribute to the maintenance of arterial pressure.\textsuperscript{7} Indeed most forms of hypotension are associated with appropriately high levels of plasma vasopressin.\textsuperscript{1,72–75} As shock worsens the initial very high plasma concentrations of vasopressin fall.\textsuperscript{7,76–78} This is illustrated by a recent study of haemorrhagic shock in dogs in which the average plasma vasopressin concentrations during the acute phase of hypotensive haemorrhage were > 300 pg/mL, but decreased to < 30 pg/mL after 1 h of sustained hypotension (normal < 5 pg/mL).\textsuperscript{7,78} Human studies have demonstrated that patients with advanced vasodilatory septic shock,\textsuperscript{79} late-phase haemorrhagic shock\textsuperscript{79} or vasodilatory shock after cardiopulmonary bypass\textsuperscript{80} had inappropriately low plasma vasopressin concentrations for the degree of hypotension.\textsuperscript{7}

Potential mechanisms for the vasopressin deficiency include:

1. impaired vasopressin secretion due to depletion of pituitary vasopressin stores after exhaustive release in early shock.\textsuperscript{7} It is known that posterior pituitary stores of vasopressin may be depleted after profound osmotic stimulation.\textsuperscript{7,46}
2. autonomic dysfunction in patients with advanced septic shock.\textsuperscript{1,80,84}
3. increased vascular endothelial release of nitric oxide in the posterior pituitary, which is known to reduce vasopressin production.\textsuperscript{1,85}

**Increased sensitivity to vasopressin in vasodilatory shock**

Clinical studies of low-dose vasopressin in human vasodilatory shock reveal a marked sensitivity to exogenously administered vasopressin.\textsuperscript{1,70,86} When exogenous vasopressin is given to such patients at doses designed to correct the low plasma vasopressin concentrations, significant increases in arterial pressure (in the range of 25–50 mmHg) are observed.\textsuperscript{7,72,84,87} This effect occurs at plasma concentrations of vasopressin similar to those found in acute hypotension, i.e. at ‘physiologic’ levels.\textsuperscript{7} This pressor response to ‘physiologic’ doses of vasopressin\textsuperscript{1} is seen in patients with severe septic shock,\textsuperscript{7} haemorrhagic shock unresponsive to volume replacement and catecholamine administration\textsuperscript{79} and vasodilatory shock after cardiopulmonary bypass and placement of a left ventricular assist device.\textsuperscript{80} It is also seen in organ donors with haemodynamic instability.\textsuperscript{88} This effect of vasopressin in patients with vasodilatory shock is remarkable as it occurs with doses that have no effect in normal subjects\textsuperscript{7,86–89} and because vasodilatory shock is characterized by resistance to other vasoconstrictors such as norepinephrine,\textsuperscript{82} angiotensin II\textsuperscript{83} and endothelin.\textsuperscript{7,94}

The reasons for this increased sensitivity are likely to be multifactorial:\textsuperscript{1}

1. As plasma concentrations of vasopressin are relatively low, there will be a significant proportion of unoccupied vasopressin receptors at which exogenous hormones may act. This is in contrast to norepinephrine and angiotensin II, where high endogenous concentrations reduce available receptors and may result in desensitization.\textsuperscript{7}
2. Patients in septic shock have an impaired sympathetic nervous system.\textsuperscript{7,81} The vasopressor action of vasopressin increases in patients with autonomic failure\textsuperscript{7,80} and also in dogs with baroreceptor denervation.\textsuperscript{7,19} The pressor sensitivity of humans with idiopathic orthostatic hypotension to physiologic doses of vasopressin increases 1000-fold.\textsuperscript{1,95}
3. Vasopressin potentiates the vasoconstrictor effect of norepinephrine\textsuperscript{2,64} and plasma norepinephrine concentrations are markedly elevated in vasodilatory shock.\textsuperscript{7,69,70}
4. Vasopressin directly inactivates the ATP-sensitive K channels\textsuperscript{7,97} whose activation in vascular smooth muscle contributes to vasodilation.
5. Vasopressin decreases the synthesis of inducible nitric oxide synthase that is stimulated by lipopolysaccharide in septic shock\textsuperscript{7,38} and blunts the increase in cyclic guanosine monophosphate (cGMP) that occurs in nitric oxide\textsuperscript{2,38} and atrial natriuretic peptide-mediated vasodilation.\textsuperscript{7,38}
Randomized controlled trials of vasopressin in vasodilatory shock

Several small studies and case reports demonstrate the efficacy of low-dose vasopressin as pressor support in patients with septic shock, cardiogenic shock, and vasodilatory shock after cardiopulmonary bypass, cardiac transplantation, organ donation and left ventricular assist device implantation. Vasopressin is effective in shock states refractory to other vasopressor therapies (including epinephrine, nor-epinephrine, dopamine and dobutamine) with end points that include increase in arterial blood pressure, reduction or discontinuation of catecholamine infusions, and increase in urine output.

However to date there are only two small randomized controlled trials of vasopressin in humans. The first trial, involving only 10 patients, investigated the use of vasopressin in the treatment of vasodilatory shock following cardiopulmonary bypass and left ventricular assist device placement. Patients who had mean arterial pressure < 70 mmHg despite norepinephrine infusion of 8 µg/min were randomized to receive vasopressin infusion (0.10 U/min) or placebo. Vasopressin infusion significantly increased mean arterial pressure (from 57 to 84 mmHg) and allowed reduction in epinephrine infusion by > 50%, the authors concluding that vasopressin is an effective vasopressor in vasodilatory shock after cardiopulmonary bypass.

The second trial involved using vasopressin in patients with septic shock. Ten patients admitted to the trauma ICU with vasodilatory septic shock (need for pressor agents to maintain mean arterial pressure > 70 mmHg) were randomized to receive vasopressin infusion (0.04 U/min) or placebo. Vasopressin infusion significantly increased mean arterial pressure (from 57 to 84 mmHg) and allowed reduction in epinephrine infusion by > 50%, the authors concluding that vasopressin is an effective vasopressor in vasodilatory shock after cardiopulmonary bypass.

Conclusions from recent reviews

Two recent reviews give insight into the current state of knowledge regarding the use of vasopressin in vasodilatory shock patients. In refractory vasodilatory shock, vasopressin infusion results in an effective pressor response and allows reduction in conventional exogenous catecholamine infusion rate. In ‘physiologic’ (low) doses (0.01–0.04 U/min giving plasma levels of 20–30 pg/mL) vasopressin is an effective pressor without causing organ hypoperfusion. ‘Pharmacologic’ (high) dose vasopressin (> 0.04 U/min, plasma levels of > 100 pg/mL) also creates an effective pressor response but at the cost of potentially harmful vasoconstriction of renal, mesenteric, pulmonary, and coronary vasculature. Use of low-dose vasopressin in severe vasodilatory shock provides potential benefits of restoration of vasomotor tone, preservation of renal blood flow and urine output, whilst avoiding renal, mesenteric, pulmonary and coronary ischaemia.

However, none of the studies to date can demonstrate whether vasopressin has any effect on organ dysfunction or survival. Randomized clinical trials designed to determine whether use of vasopressin can alter the outcome of vasodilatory shock are needed before vasopressin is used clinically. Studies to determine the optimum or safe dose, or comparing the use of low-dose versus high-dose vasopressin infusions are also needed. Similarly, the lack of studies addressing the use of vasopressin in vasodilatory shock in the postcardiac arrest population is reflected in the Class Indeterminate recommendation in the 2000 AHA guidelines for post-resuscitation management.

The guidelines state, in the postcardiac arrest patient, ‘If vasodilatory shock is refractory to adrenergic vasopressor agents, a continuous infusion of vasopressin may be beneficial’. The indeterminate recommendation recognizes that data from existing studies of vasopressin use in vasodilatory shock is only indirectly applicable to patients who have had a cardiac arrest. Recognition of the lack of studies of vasopressin treatment during the post-resuscitation period prompted the panelists to ‘strongly suggest’ that ‘more studies be performed to study the most effective therapies for this vulnerable period’.

Vasopressin use in the ED

Current international guidelines suggest that vasopressin has two potential uses in the ED setting.

1. In adult patients with shock-refractory VF, 40 U of vasopressin i.v. as a single dose may be used as an alternative to 1 mg epinephrine, repeated every 2–5 min. Use of vasopressin in adult patients with asystole or pulseless electrical activity, or in paediatric cardiac arrest is not recommended.

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2. Patients with severe vasodilatory (septic) shock refractory to adrenergic vaspressors may benefit from low-dose vasopressin infusion (0.01–0.04 U/min) in order to stabilize cardiorespiratory function.²

Conclusion

Vasopressin is an endogenous peptide hormone with potent vasoconstrictor and antidiuretic properties. It has traditionally been used in the treatment of diabetes insipidus and in the initial management of bleeding esophageal varices. The finding that endogenous vasopressin is critically important in the maintenance of arterial blood pressure in hypotension and shock states has led to interest in the use of vasopressin in these clinical settings. Recent international advanced cardiac life support guidelines recommend a single dose of vasopressin 40 U i.v. as an alternative to epinephrine in adult shock-refractory life-saving vasopressor. Vasopressin is emerging as a rational therapy for haemodynamic support of patients in severe vasopressor-refractory vasodilatory shock.¹ Large scale multicentre randomized controlled trials demonstrating the efficacy of vasopressin in this setting are required before routine use of vasopressin in refractory vasodilatory shock can be recommended.

References


70. Argenziano M, Choudhri AF, Oz MC et al. Vasopressin deficiency and pressure hypersensitivity in haemodynamically unstable organ donors. Circulation 1999; 100 (Suppl. II): 244-6.


