Vasopressin (Argipressin)

**Applicable areas**

**This section will be left blank for each hospital to complete in accordance with local practice. Examples: ICU, ED, OR, Ward 2B**

## Mechanism of action/pharmacology

Endogenous vasopressin is a hormone secreted by the posterior pituitary gland that acts as a non-adrenergic vasopressor. Actions on vascular smooth muscle V1 receptors cause peripheral vasoconstriction and increase systemic vascular resistance and blood pressure. Less potent actions on V2 receptors in the kidneys cause an antidiuretic effect by promoting water reabsorption by the renal tubules.1 Vasopressin also increases adrenocorticotropic hormone (ACTH) and aldosterone levels.

Vasopressin plays a minimal role in blood pressure regulation in normotensive states. During vasodilatory shock, administration may correct a relative endogenous vasopressin deficiency that develops when endogenous secretory stores become depleted.2

Vasopressin augments the response to catecholamine therapy, reducing the required catecholamine dose,3,4 and may be a more effective vasopressor in patients with severe acidosis, when the efficacy of adrenergic agents is potentially reduced due to receptor down-regulation.5

Onset of action: 1–2 minutes.6

Duration of action: up to 20 minutes.6

Half-life: 10–20 minutes.1

## Indications

To increase blood pressure in refractory vasodilatory shock when low systemic vascular resistance persists despite adequate fluid resuscitation and first-line vasopressor support with noradrenaline.7

The optimal timing for initiating vasopressin therapy remains controversial. Under conventional management, the introduction of vasopressin is delayed until the patient’s noradrenaline requirement is greater than 20 to 30microg/min. Limited studies have investigated the role of vasopressin as a first-line agent in treating septic shock, and the benefits of this approach remain uncertain.8

Vasopressin provides a component of physiological support in brain dead potential organ donors.9

## Precautions

* Hypersensitivity to vasopressin or components – anaphylaxis has been reported1
* Hypotension due to uncorrected hypovolaemia1
* Conditions exacerbated by fluid overload or water intoxication including asthma, epilepsy and heart failure.1

## Medication presentation

20 units/1 mL vial.

## Medication storage

Store vials below 25°C. Do not freeze.10

Infusion solutions are only stable for 18 hours at room temperature1 or 24 hours at 2–8°C.10

Stock not registered in Australia will require completion of a Special Access Scheme Category A form.

## Preparation

|  |  |
| --- | --- |
|  | Syringe driver |
| Prescribe | 20 units in 20 mL | 40 units in 40 mL |
| Make up infusion in | Glucose 5%\* | Glucose 5%\* |
| Volume to be drawn up into the syringe | 19 mL | 38 mL |
| Drug dose to be added | 20 units (1 mL) | 40 units (2 mL) |
| Final volume | 20 mL | 40 mL |
| Final concentration | 1 unit/mL | 1 unit/mL |
| 1 mL/hr = | 1 unit/hr | 1 unit/hr |

\*Glucose 5% is preferred for diluting all inotropes and vasopressors. However, Vasopressin is also compatible with sodium chloride 0.9%.10

## Administration – this guideline is intended for central access only

Administer continuous intravenous infusion through a central access line.10

Infusions should be administered via a syringe driver, preferably with medication error reduction software enabled.

Avoid administration via lines where other drugs or fluids may be bolused or flushed.11

## Dosing

Starting dose: 0.6 units/hr.

Titrate in accordance with prescribed blood pressure parameters – for example, in increments of 0.6 units/hr.

Usual dose range for vasodilatory shock: 0.6 to 2.4 units/hr.3,4,12

Maximum dose: up to 3.6 units/hr has been used, but higher doses may increase the risk of ischaemic side effects.8

As a general rule, consider commencing vasopressin wean when the patient’s noradrenaline requirement is below 20microg/min, and wean no more rapidly than in increments of 0.6 units/hr every 15 minutes.

Usual dose range for physiological support for brain dead potential organ donors: 0.5 to 2.4 units/hr.9

## Monitoring

* Continuous blood pressure and cardiac monitoring for the duration of the infusion
* Monitor fluid balance and electrolytes
* Assess for organ ischaemia (including myocardium, kidneys, gastrointestinal tract and peripheral extremities) – see ‘Side effects’ for more information.

## Side effects

* Decreased cardiac output, cardiac dysrhythmia and cardiac arrest6
* Myocardial, mesenteric or peripheral (digital) ischaemia – can manifest as acute myocardial infarction, gastrointestinal infarction, decreased urine output/creatinine clearance or gangrene6
* Hyponatraemia – due to water retention.6

## Compatibilities

Consult the following references, which are available online through the [Clinicians Health Channel](https://www2.health.vic.gov.au/clinicianshealthchannel):

* Australian injectable drugs handbook
* Trissel’s™ in IV compatibility (Micromedex) – from the site homepage, select the ‘IV Compatibility’ tab.

## Important drug interactions

There are no known significant drug interactions.

## References

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9. Australian and New Zealand Intensive Care Society. The ANZICS statement on death and organ donation (Edition 3.2). ANZICS, Melbourne, 2013.
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