

---

# 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  $V_1$  receptors cause peripheral vasoconstriction and increase systemic vascular resistance and blood pressure. Less potent actions on  $V_2$  receptors in the kidneys cause an antidiuretic effect by promoting water reabsorption by the renal tubules.<sup>1</sup> Vasopressin also increases adrenocorticotrophic 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.<sup>2</sup>

Vasopressin augments the response to catecholamine therapy, reducing the required catecholamine dose,<sup>3,4</sup> 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.<sup>5</sup>

Onset of action: 1–2 minutes.<sup>6</sup>

Duration of action: up to 20 minutes.<sup>6</sup>

Half-life: 10–20 minutes.<sup>1</sup>

## 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.<sup>7</sup>

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 30 microg/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.<sup>8</sup>

Vasopressin provides a component of physiological support in brain dead potential organ donors.<sup>9</sup>

## PRECAUTIONS

- Hypersensitivity to vasopressin or components – anaphylaxis has been reported<sup>1</sup>
- Hypotension due to uncorrected hypovolaemia<sup>1</sup>

- Conditions exacerbated by fluid overload or water intoxication including asthma, epilepsy and heart failure.<sup>1</sup>

## MEDICATION PRESENTATION

20 units/1 mL vial.

## MEDICATION STORAGE

Store vials below 25°C. Do not freeze.<sup>10</sup>

Infusion solutions are only stable for 18 hours at room temperature<sup>1</sup> or 24 hours at 2–8°C.<sup>10</sup>

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

## PREPARATION

	Syringe driver	
<b>Prescribe</b>	20 units in 20 mL	40 units in 40 mL
<b>Make up infusion in</b>	Glucose 5%*	Glucose 5%*
<b>Volume to be drawn up into the syringe</b>	19 mL	38 mL
<b>Drug dose to be added</b>	20 units (1 mL)	40 units (2 mL)
<b>Final volume</b>	20 mL	40 mL
<b>Final concentration</b>	1 unit/mL	1 unit/mL
<b>1 mL/hr =</b>	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%.<sup>10</sup>

## ADMINISTRATION – THIS GUIDELINE IS INTENDED FOR CENTRAL ACCESS ONLY

Administer continuous intravenous infusion through a central access line.<sup>10</sup>

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.<sup>11</sup>

## 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.<sup>3,4,12</sup>

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

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.<sup>9</sup>

## 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 arrest<sup>6</sup>
- Myocardial, mesenteric or peripheral (digital) ischaemia – can manifest as acute myocardial infarction, gastrointestinal infarction, decreased urine output/creatinine clearance or gangrene<sup>6</sup>
- Hyponatraemia – due to water retention.<sup>6</sup>

## COMPATIBILITIES

Consult the following references, which are available online through the Clinicians Health Channel:

- 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

1. MIMS [online] (accessed 4 April 2017)
2. Landry DW, Levin HR, Gallant EM, et al. Vasopressin deficiency contributes to the vasodilation of septic shock. *Circulation* 1997; 95(5): 1122–1125
3. Tsuneyoshi I, Yamada H, Kakihana Y, et al. Hemodynamic and metabolic effects of low-dose vasopressin infusions in vasodilatory septic shock. *Critical Care Medicine* 2001; 29(3): 487–493
4. Dellinger RP, Levy MM, Rhodes A, et al. Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock, 2012. *Critical Care Medicine* 2013; 41(2): 580–637
5. Sharman A, Low J. Vasopressin and its role in critical care. *Continuing Education in Anaesthesia Critical Care and Pain* 2008; 8(4): 134–137
6. Micromedex [online] (accessed 5 April 2017).
7. Rhodes A, Evans LE, Alhazzani W, et al. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock: 2016. *Intensive Care Medicine* 2017; 43(3): 304–377
8. Gordon AC, Mason AJ, Thirunavukkarasu N, et al. Effect of early vasopressin vs norepinephrine on kidney failure in patients with septic shock: the VANISH randomized clinical trial. *JAMA* 2016; 316(5):509–518
9. Australian and New Zealand Intensive Care Society. The ANZICS statement on death and organ donation (Edition 3.2). ANZICS, Melbourne, 2013.
10. Australian injectable drugs handbook (AIDH) [online] (accessed 2 April 2016).
11. University College London Hospitals (UCL). UCL hospitals injectable medicines administration guide: pharmacy department, 3rd edn. Wiley-Blackwell, Chichester, 2013.
12. Russell JA, Walley KR, Singer J, et al. Vasopressin versus norepinephrine infusion in patients with septic shock. *New England Journal of Medicine* 2008; 358(9):877–887

## ACKNOWLEDGEMENTS

We would like to thank the pharmacists involved in writing the guidelines: Melissa Ankravs, Melanie Kowalski, Rachel Fyfe, Robyn Ingram, Annalie Jones, Susan Trevillian, and Lucy Sharrock.

To receive this publication in an accessible format phone 9096 1384, using the National Relay Service 13 36 77 if required, or email [info@safercare.vic.gov.au](mailto:info@safercare.vic.gov.au)

Printed copies of this document may not be the most recent version.

Authorised and published by the Victorian Government, 1 Treasury Place, Melbourne.

© State of Victoria, Australia, Safer Care Victoria, December 2018

ISBN 978-1-76069-711-2 (online/print)

Available at [www.safercare.vic.gov.au](http://www.safercare.vic.gov.au)

Email [criticalcare.clinicalnetwork@safercare.vic.gov.au](mailto:criticalcare.clinicalnetwork@safercare.vic.gov.au)

