Safer Care Victoria

Dopamine

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

Dopamine is an immediate catecholamine precursor of noradrenaline that directly stimulates alpha, beta and peripheral dopaminergic receptors in a dose-dependent manner, as well as acting indirectly by releasing endogenous noradrenaline from storage sites in sympathetic nerve endings.

At low doses, dopamine receptor stimulation predominates, causing renal, mesenteric and coronary blood vessel vasodilation without any significant effect on cardiac output or blood pressure.

At infusion rates of 3 to 10 microg/kg/min, β_1-receptor activation promotes positive inotropic and some chronotropic effects, increasing stroke volume, heart rate and cardiac output.

At high infusion rates, above 10 microg/kg/min, α-receptor stimulation predominates causing vasoconstriction and an increase in blood pressure.

Due to interpatient variation, these effects may occur at doses above or below the ranges stated.

The overall α-adrenergic receptor effect of dopamine is weaker than that of noradrenaline, and the β_1-adrenergic receptor stimulation of dopamine at doses greater than 2 microg/kg/min can result in dose-limiting dysrhythmias.[1]

Onset of action: 5 minutes.[2]

Duration of action: less than 10 minutes.[2]

Half-life: 2 minutes.[2]

INDICATIONS

Post-cardiac surgery, dopamine may be used short-term in low cardiac output states.

Dopamine is no longer recommended as a first-line treatment for cardiogenic or septic shock. Its use is associated with increased mortality, tachycardia and arrhythmias.[3,4]

Low-dose dopamine infusions should not be used for renal protection.[3,5]

PRECAUTIONS

- Hypersensitivity to dopamine or sulfites (vial contains sodium metabisulfite)[2]

- Hypotension due to uncorrected hypovolaemia[2]
- Tachyarrhythmias[2]
- Hypertrophic obstructive cardiomyopathy (HOCM) and severe aortic stenosis – potential for outflow obstruction
- Pulmonary hypertension – may worsen pulmonary vasoconstriction[2]
- Phaeochromocytoma.[2]

MEDICATION PRESENTATION

200 mg/5mL ampoule (Dopamine Concentrate DBL®).

MEDICATION STORAGE

Store below 30°C. Protect from light.[6]

Infusion solution: stable for 24 hours at 25°C.[6]

PREPARATION

	Infusion pump		Syringe driver
Prescribe	300 mg in 50 mL	600 mg in 100mL	300 mg in 50mL
Make up infusion in	50 mL bag of glucose 5%*	100 mL bag of glucose 5%*	Glucose 5%*
Volume to be removed from IV bag	7.5 mL	15 mL	Not applicable Draw up 42.5 mL in the syringe
Drug dose to be added	300 mg (7.5 mL)	600 mg (15 mL)	300 mg (7.5 mL)
Final volume	50 mL	100 mL	50 mL
Final concentration	6 mg/mL	6 mg/mL	6 mg/mL
1mL/hr =	100 microg/min	100 microg/min	100 microg/min

* Glucose 5% is preferred for diluting all inotropes and vasopressors. However, dopamine is also compatible with glucose in sodium chloride solutions, Hartmann's and sodium chloride 0.9%.[6]

ADMINISTRATION – THIS GUIDELINE IS INTENDED FOR CENTRAL ACCESS ONLY

Administer continuous intravenous infusion through a central access line.

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

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

DOSING

Starting dose: 100 to 400 microg/min.[2,7,8]

Titrate in accordance with prescribed parameters – for example, by increments of 50 to 100 microg/min.[9] Effects on end-organ perfusion may not occur immediately.

Usual dose range: 100 to 1,500 microg/min (2 to 10 microg/kg/min).[2]

Doses above 10 microg/kg/min may be required but are associated with increased adverse effects. Maximum dose 20 microg/kg/min.[9]

If weight-based dosing methods are employed, use ideal body weight.[10]

Dopamine infusions should not be ceased abruptly.[6]

MONITORING

- Continuous blood pressure and cardiac monitoring for the duration of the infusion[6]
- Daily 12-lead ECG
- Monitor fluid balance and electrolytes at least daily, especially magnesium and potassium.

SIDE EFFECTS

- Angina, tachycardia, arrythmias and palpitations[1]
- Tissue ischaemia or necrosis due to vasoconstriction[3]
- Hyperglycaemia[3]
- Lactic acidaemia.

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

- **Monoamine oxidase inhibitors (MAOIs)** (including reversible, non-selective agents such as linezolid) inhibit the metabolism of dopamine and prolong its duration of action. Patients who have been treated with a MAOI in the past 2–3 weeks will require a substantially reduced dopamine dose and may experience an exaggerated hypertensive response. Reduce the dopamine starting dose to 0.2 to 0.5microg/kg/min and titrate cautiously.[2,11]
- **Tricyclic antidepressants (TCAs)** may potentiate the cardiovascular effects of dopamine, increasing the risk of arrhythmias, tachycardia, hypertension and hyperpyrexia. Dose dopamine conservatively if the combination cannot be avoided.[2,11]
- **Halogenated anaesthetics** sensitise the myocardium to the effects of dopamine, increasing the risk of ventricular arrhythmias and hypotension. Avoid dopamine use.[2,11]
- **Ergot derivatives** enhance the vasoconstrictive effects of dopamine and increase the risk of severe hypertension and gangrene. Avoid dopamine use.[2,11]
- **Oxytocin** enhances the vasopressor effects of dopamine and increases the risk of severe hypertension. Avoid dopamine use.[2,11]
- **Digoxin** may increase the risk of cardiac arrhythmias when used in conjunction with dopamine. Dose dopamine cautiously with close ECG monitoring.[2,11]
- **Intravenous phenytoin** (but not enteral phenytoin) may cause dose-dependent, sudden hypotension in patients receiving dopamine infusions. Consider alternative antiepileptics.[2,11]
- **α or β-antagonists**: concurrent administration with dopamine will reduce the efficacy of both drugs.[2,11] Patients taking non-selective β-blockers may experience severe hypertension.[12]

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Email criticalcare.clinicalnetwork@safercare.vic.gov.au

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