DOPAMINE HYDROCHLORIDE

℞

40 mg/mL (200 mg/ 5 mL) Concentrate Solution for I.V. Infusion

1.0 PHARMACOLOGIC CATEGORY

Cardiac Stimulant (Adrenergic and Dopaminergic agent)

2.0 DESCRIPTION

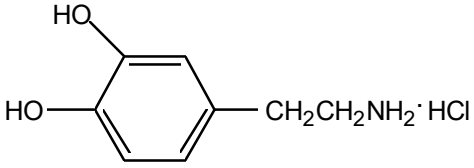
Dopamine hydrochloride is a naturally occurring biochemical catecholamine precursor of noradrenaline and adrenaline. The chemical name of dopamine hydrochloride is 4-(2-aminoethyl) benzene-1,2-diol hydrochloride and has the chemical formula $C_8H_{11}NO_2$.HCl.

Excipient with known effect

Sodium metabisulfite

Chemical structure

The chemical structure is:



DOPAMINE HYDROCHLORIDE

MW 189.6

CAS number

CAS 51-61-6

3.0 FORMULATION/ COMPOSITION

Sterile Dopamine Concentrate is a sterile solution of Dopamine Hydrochloride BP in water for injection, containing 1% sodium metabisulfite. The strength supplied is 200 milligrams/5 mL in a clear glass ampoule.

4.0 CLINICAL PARTICULARS

4.1 Therapeutic Indications

For the correction of hemodynamic imbalance present in:

Acute hypotension or shock associated with myocardial infarction, endotoxic septicemia, trauma and renal failure.

As an adjunct after open heart surgery, where there is persistent hypotension after correction of hypovolemia.

In chronic cardiac decompensation as in congestive failure.

4.2 Dosage and Method of Administration

WARNING: Dopamine is a potent drug. It must be diluted before administration. Do not add to alkaline solutions such as sodium bicarbonate, as these inactivate dopamine.

In appropriate cases, restoration of blood volume with plasma, whole blood, or a suitable plasma expander, should be instituted prior to administration: central venous pressure should be 10 to 15 cm H_2O, or pulmonary wedge pressure 14 to 18 mm Hg.

Dosage

Adult

When appropriate, increase blood volume with a suitable plasma expander, whole blood or plasma until central venous pressure is 10 to 15 cm H_2O or pulmonary wedge pressure is 14 to 18 mm Hg. Begin administration of diluted solution at doses of 2 to 5 micrograms/kg/min in patients who are likely to respond to modest increments of heart force and renal perfusion (see table under **Section 5.0 Pharmacological properties** for physiological effects at various doses).

In more seriously ill patients, begin administration of diluted solution at doses of 5 micrograms/kg/min and increase gradually using 5 to 10 micrograms/kg/min increments up to 20 to 50 micrograms/kg/min as needed. In patients who do not respond to these doses with adequate arterial pressures or urine flow, additional increments may be employed in an effort to produce an appropriate arterial pressure and central perfusion. If doses of dopamine in excess of 50 micrograms/kg/min are required, it is suggested that urine output be checked frequently. Should urine flow begin to decrease in the absence of hypotension, reduction of dosage should be considered. Once optimal hemodynamic effects have been achieved, the lowest dose that maintains these effects should be used. Multiclinic trials have shown that more than 50% of the patients were satisfactorily maintained on doses of dopamine less than 20 micrograms/kg/min.

Treatment of all patients requires constant evaluation of therapy in terms of the blood volume, augmentation of myocardial contractility, distribution of peripheral perfusion and urinary output. Dosage should be adjusted according to the patient's response with particular attention to diminution of established urine flow rate, increasing tachycardia or development of new dysrhythmias as indices for decreasing or temporarily suspending the dosage.

Care must be taken in patients with cardiac decompensation to avoid alpha-adrenoceptor induced vasoconstriction and increased afterload. These patients should be started on a dose of 1 to 2 micrograms/kg/minute and the rate of infusion increased with caution. Patients with occlusive vascular disease should also be commenced on a similar low dose.

For patients with severe, refractory, chronic congestive heart failure who are to be treated for a short period of time with dopamine, it is recommended that the infusion rate be commenced at a rate of 0.5 to 2 micrograms/kg/min, increasing the dose as the urine flow increases to a usual maintenance dose of 1 to 3 micrograms/kg/min. The infusion rate should be reduced if the diastolic blood pressure or heart rate increases.

Pediatric

It is not recommended for use in children as safety and efficacy in this age group has not been established.

Geriatric

No variation in dosage is suggested for geriatric patients.

Method of administration

The rate of administration should be controlled in order to prevent inadvertent bolus administration: constant evaluation of therapy should be undertaken (ie. blood volume, ECG, arterial blood pressure, urine output, augmentation of myocardial contractility and distribution of peripheral perfusion. Measurement of central venous pressure and pulmonary wedge pressure and cardiac output are also helpful). Dopamine should be administered into a large vein (preferably of the antecubital fossa) to reduce the risk of extravasation into surrounding tissue which may cause necrosis.

Antidote for peripheral ischemia following extravasation

To prevent sloughing and necrosis in ischemic areas, the area should be infiltrated as soon as possible with 10 to 15 mL of sodium chloride intravenous infusion 0.9% containing from 5 to 10 milligrams of phentolamine, an adrenergic blocking agent. A syringe with a fine hypodermic needle should be used, and the solution liberally infiltrated throughout the ischemic area. Sympathetic blockade with phentolamine causes immediate and conspicuous local hyperemic changes if the area is infiltrated within 12 hours. Therefore, phentolamine should be given as soon as possible after the extravasation is noted.

Suggested dilution

Aseptically transfer Sterile Dopamine Concentrate into the intravenous solution as per the table below:

Strength	**Volume**	**Intravenous**	**Final**
milligrams/5 mL	**mL**	**solution**	**concentration**
		volume	**micrograms/mL**
		mL	
200	5	250	800
200	5	500	400

In patients in whom greater fluid load is undesirable, an alternative regimen is suggested:

Strength	Volume	Intravenous	Final
milligrams/5 mL	mL	solution	concentration
		volume	micrograms/mL
		mL	
200	10	250	1600
200	20	500	1600

Rate of administration

Dopamine, after dilution, is administered intravenously through a suitable intravenous catheter or needle. An intravenous drip chamber or other suitable metering device is essential for controlling the rate of flow in drops per minute. Each patient must be individually titrated to the desired hemodynamic and/or renal response with dopamine. In titrating to the desired increase in systolic blood pressure, the optimum dosage rate for renal response may be exceeded, thus, necessitating a reduction in rate after the hemodynamic condition is stabilized.

Administration at rates greater than 50 micrograms/kg/minute has safely been used in advanced circulatory decompensation states. If unnecessary fluid expansion is of concern, adjustment of drug concentration may be preferred over increasing the flow rate of a less concentrated dilution.

Compatibilities

Dopamine has been reported to be compatible with the following: sodium chloride 0.9%, glucose 5%, glucose 5% and sodium chloride 0.9%, glucose 5% in sodium chloride 0.45%, glucose 5% in lactated Ringer's solution, sodium lactate 1/6 M injection, lactated Ringer's injection.

It is recommended that, if dopamine is administered with other drugs, a "piggyback" administration set or administration into a second injection site is used to avoid mixing of potent drugs with dopamine.

IV fluids

Dopamine injection has been shown to be stable for 24 hours when 200 milligrams is diluted in 250 mL or 500 mL of the following intravenous fluids:

sodium chloride infusion 0.9%
glucose 5% injection
glucose 5% and sodium chloride infusion 0.9%
5% glucose in lactated Ringer's solution
1/6 M sodium lactate injection
lactated Ringer's injection

However, as with all intravenous admixtures, dilution should be made just prior to administration.

Antibiotics

Dopamine has been found to be chemically and physically stable for 24 hours (at 23° to 25°C and exposed to light) with the following antibiotics:

kanamycin sulfate (500 milligrams/250 mL 5% glucose admixture)
tetracycline hydrochloride (250 milligrams/250 mL 5% glucose admixture)
carbenicillin disodium (1.0 g/250 mL 5% glucose admixture)
chloramphenicol sodium succinate (1.0 g/250 mL 5% glucose admixture)
cephalothin sodium neutral (1.0 gram/250 mL 5% glucose admixture) (see note below)
oxacillin (500 milligrams/250 mL 5% glucose) (see note below)

Because of loss of potency of the antibiotic at 24 hours the following admixtures of antibiotics and dopamine in 5% glucose solution should be administered within six hours of mixing:

gentamicin sulfate (80 milligrams/250 mL 5% glucose)
cephalothin sodium (1.0 g/250 mL 5% glucose)
penicillin G potassium (5,000,000 units/250 mL 5% glucose)

NOTE: It is recommended in the literature that cephalothin sodium, oxacillin sodium and gentamicin sulfate not be mixed with any other drug. It is considered that the recommendation should also include cephalothin sodium neutral. Although studies indicate that dopamine hydrochloride may be compatible with these drugs, their admixture produces a fixed combination of potent drugs. It is suggested that admixtures containing gentamicin sulfate, cephalothin sodium, cephalothin sodium neutral or oxacillin sodium should be avoided unless all other viable alternatives have been exhausted.

Other medicines

Heparin sodium (50,000 units/250 mL 5% glucose) has been shown to be compatible with dopamine hydrochloride for 24 hours.

Lignocaine hydrochloride (1.0 g/250 mL 5% glucose) has been shown to be compatible with dopamine hydrochloride for 24 hours.

Mixing other drugs in dopamine infusion is not recommended, as sufficient evidence of compatibility is not available.

Incompatibilities - Refer to **Section 6.4 Incompatibilities**.

4.3 Contraindications

Administration of dopamine is contraindicated in the following cases:

- Pheochromocytoma: dopamine may release catecholamines into the circulation, producing an additive effect to an already abnormally high catecholamine level, and causing acute hypertension.
- Atrial or ventricular tachyarrhythmias or ventricular fibrillation.
- Concurrent use with cyclopropane and halogenated hydrocarbon anesthetics (see **Section 4.5 Interaction with Other Medicines and Other Forms of Interaction)**.

- Hyperthyroidism.
- Concurrent use with ergotamine (see **Section 4.5 Interaction with Other Medicines and Other Forms of Interaction**).

4.4 Special Warnings and Precautions for Use

Patients who are taking Monoamine oxidase (MAO) inhibitors or who have taken them within the last two to three weeks require a substantially reduced starting dose, i.e. about $1/10^{th}$ the usual dose (see **Section 4.5 Interaction with other medicines and other forms of Interaction**).

Dopamine should not be added to alkaline diluents (see **Section 4.2 Dosage and Method of Administration** and **Section 6.4 Incompatibilities**).

Hypovolemia

Hypovolemia should be fully corrected prior to treatment with dopamine with a suitable plasma expander or whole blood or plasma until the central venous pressure is 10 to 15 cm H_2O or the pulmonary wedge pressure is 14 to 18 mm Hg.

Decreased pulse pressure

Excessive dosage may be indicated by a disproportionate rise in diastolic pressure (ie. a marked decrease in pulse pressure). The infusion rate should be decreased or ceased and the patients observed carefully for further evidence of predominant vasoconstriction activity, unless such an effect is desired.

Occlusive vascular disease

Those patients with pre-existing peripheral vascular disease, such as that due to atherosclerosis, arterial embolism, Buerger's disease, Raynaud's disease, diabetic endarteritis or cold injury (e.g., frostbite), may be more susceptible to peripheral ischemia and subsequent gangrene and should be observed carefully for any changes in color or temperature of the skin in the extremities. If ischemia occurs and is thought to be due to vasoconstriction, the benefits of the dopamine infusion should be weighed against the risks of possible necrosis. Ischemia may be reversed by either decreasing the rate or discontinuing the infusion. Intravenous administration of phentolamine 5 to 10 milligrams may also reverse the ischemia.

As with any cardiac stimulant, care should be exercised when administering dopamine to patients with cardiac ischemia.

Acidosis, hypercapnia or hypoxia may reduce the effectiveness and/or increase the incidence of adverse effects of dopamine. These conditions should be corrected prior to or concurrently with administration of dopamine.

Pulmonary hypertension may be exacerbated due to dopamine-induced pulmonary vasoconstriction. Where dopamine-induced pulmonary hypertension has occurred, isoprenaline may be considered as an alternative inotropic agent.

Routine monitoring of blood pressure, ECG, cardiac status and renal output, is necessary in all patients. Where possible, the cardiac output and pulmonary wedge pressure should also be measured.

Sulfite sensitivity

Sterile Dopamine Concentrate contains sodium metabisulfite, which may cause allergic-type reactions, including anaphylactic symptoms and life-threatening or less severe asthmatic episodes in susceptible people. The overall prevalence of sulfite sensitivity in the general population is unknown and probably low. Sulfite sensitivity is seen more frequently in asthmatic than non-asthmatic people.

Hypotension

Hypotension may occur when attempting to wean patients from dopamine and it may be necessary to substitute dopamine with another pressor agent or to expand blood volume whilst gradually reducing the infusion rate.

Extravasation

Dopamine should be infused into a large vein whenever possible to prevent the possibility of extravasation into tissue adjacent to the infusion site. The infusion site should be continuously monitored for free flow. Extravasation may cause necrosis and sloughing of the surrounding tissue. A syringe with a fine hypodermic needle should be used to liberally infiltrate the ischemic area as soon as extravasation is noted.

Diabetes

Glucose solutions should be used with caution in patients with known subclinical or overt diabetes mellitus.

Use in hepatic impairment

Dopamine is metabolized in the tissues and blood by MAO inhibitors and COMT. Since the effect of impaired liver function is not known, close monitoring is advisable.

Use in renal impairment

Dopamine and its metabolites are almost completely excreted in the urine. Since the effect of impaired renal function is not known, close monitoring of such patients is advisable.

Use in the elderly

No variation in dosage is suggested for geriatric patients. Close monitoring is required for blood pressure, urine flow, and peripheral tissue perfusion.

Pediatric use

It is not recommended for use in children as safety and efficacy in this age group has not been established.

Effects on laboratory tests

Dopamine or its metabolites may interfere with urine tests for amino acids or catecholamines and also with tests for detecting uric acid or urobilinogen.

Infusion of dopamine suppresses pituitary secretion of thyroid stimulating hormone, and prolactin.

4.5 Interaction with Other Medicinal Products and Other Forms of Interaction

Alcohol: No information available.

Food: Not applicable.

Cyclopropane and halogenated anesthetics sensitize the myocardium to the effects of dopamine. Dopamine should therefore be used with extreme caution with these drugs due to the potential for developing ventricular arrhythmias or hypertension.

MAO inhibitors potentiate the effect of dopamine and prolong its duration of action. Patients being treated, or who have been treated within the previous two to three weeks, with MAO inhibitors will, therefore, require a substantially reduced dosage of dopamine (the starting dose should be reduced to $1/10^{th}$ of the usual dose or less).

Alpha and beta adrenergic receptor blocking drugs will interfere with the alpha and beta adrenergic responses induced by dopamine. The use of other pressor amines may be associated with complex interactions. The cardiac effects of dopamine are antagonized by β-adrenergic blocking agents such as propranolol and metoprolol, and the peripheral vasoconstriction caused by high doses of dopamine is antagonized by α-adrenergic blocking agents.

Hypotension may be observed with concurrent use of vasodilators such as glyceryl trinitrate, nitroprusside and calcium channel blockers.

In animal studies, large doses of butyrophenones blocked the dopaminergic mediated renal vasodilation. Whether this occurs in man is not known.

Tricyclic antidepressants may potentiate the cardiovascular effects of dopamine, possibly resulting in arrhythmias, tachycardia or severe hypertension or hyperpyrexia (see **Section 4.4 Special warnings and precautions for use**).

Concurrent use of digitalis glycosides with dopamine may increase the risk of cardiac arrhythmias. Caution and close ECG monitoring are very important if concurrent use is necessary.

Concurrent use of methysergide or other ergot alkaloids with dopamine may result in excessive vasoconstriction and should be avoided. Ergot alkaloids or oxytocin may potentiate the pressor effect of dopamine and cause severe hypertension and rupture of cerebral blood vessels. Concurrent use of ergotamine with dopamine is not recommended as it may produce vascular ischemia and gangrene.

Guanethidine may potentiate the pressor response to dopamine.

Concurrent use of intravenous phenytoin with dopamine may result in dose dependent, sudden hypotension and bradycardia and possibly cardiac arrest. If anticonvulsant therapy is necessary during administration of dopamine, an alternative to phenytoin should be considered. Caution is also advised with concurrent use of other hydantoins.

Also refer to **Section 6.4 Incompatibilities**.

4.6 Fertility, Pregnancy and Lactation

Effects on fertility

Studies in animals have not been performed to assess the effects of dopamine on fertility.

Use in pregnancy (Category B3)

Category B3: Drugs which have been taken by only a limited number of pregnant women and women of childbearing age, without an increase in the frequency of malformation or other direct or indirect harmful effects on the human fetus having been observed. Studies in animals have shown evidence of increased fetal damage, the significance of which is considered uncertain in humans.

It is not known whether dopamine crosses the placental barrier. In one animal study, the administration of dopamine to pregnant rats resulted in a decreased survival rate of the newborn and cataract formation in the survivors. The benefits of using this product should be weighed against the possible risks to the fetus.

Use in lactation

It is not known if dopamine is excreted in breast milk, nor is the effect on the infant known. Dopamine is inactive when ingested orally, nonetheless it is not recommended for breast-feeding mothers unless the expected benefits outweigh any potential risks.

4.7 Effects on Ability to Drive and Use Machines

The effects of this medicine on a person's ability to drive and use machines were not assessed as part of its registration. Patients should refrain from driving or using machines until they know that the medicinal product does not negatively affect these abilities.

4.8 Undesirable Effects

Common reactions

Adverse reactions have been observed in 19% of patients during clinical evaluation; however, only half of these were attributed to dopamine. Treatment was terminated in 5% of all patients due to adverse reactions.

Cardiovascular - Ectopic beats, tachycardia, anginal pain, palpitation, hypotension, vasoconstriction.

Gastrointestinal - Nausea, vomiting.

Nervous system - Headache.

Respiratory - Dyspnea.

Less common reactions

Biochemical abnormalities – Azotemia.

Cardiovascular – Atrial fibrillation, aberrant ventricular conduction, bradycardia, widened QRS complex, hypertension, ectopic heart beats. A few cases of peripheral cyanosis have been reported in patients receiving dopamine.

Nervous System – Piloerection, anxiety.

Serious or life threatening reactions

Gangrene of feet has occurred following doses of 10 to 14 micrograms/kg/min and higher and in a few patients with pre-existing vascular disease (see **Section 4.4 Special warnings and precautions for use**).

Fatal ventricular arrhythmias have been reported on rare occasions. Extravasation of dopamine may cause necrosis and sloughing of surrounding tissue (see **Section 4.2 Dosage and Method of Administration**).

4.9 Overdose and Treatment

Symptoms

Excessive elevation of blood pressure and vasoconstriction could be expected to occur due to the alpha-adrenergic actions of dopamine, especially in patients with a history of occlusive vascular disease (see **Section 4.8 Undesirable effects**).

Treatment

In case of accidental overdose the rate of administration should be reduced or the infusion discontinued temporarily until the patient's condition stabilizes. Since the duration of action of dopamine is quite short, no additional measures are usually necessary. If these measures fail to stabilize the patient's condition in a relatively short time, use of the short acting alpha adrenergic blocking agent, phentolamine, should be considered.

5.0 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic Properties

Mechanism of action

Dopamine hydrochloride can stimulate alpha, beta and dopamine receptors. At infusion rates

of 0.5 to 2 micrograms/kg/min, dopamine receptors are selectively activated and blood pressure either does not change or decreases slightly. The most important effects are renal and mesenteric vasodilatation. Renal plasma flow, glomerular filtration rate and sodium excretion usually increase. At infusion rates of 2 to 10 micrograms/kg/min, $beta_1$-receptors are activated and cardiac output and systolic blood pressure increase. The total peripheral resistance is relatively unchanged because of peripheral vasoconstriction (alpha effect) and muscle vasodilatation (beta effect). At infusion rates above 10 micrograms/kg/min, alpha-receptors are activated, causing vasoconstriction, and both systolic and diastolic pressures increase.

Dopamine does not cross the blood-brain barrier and so does not activate dopamine receptors in the brain.

Cardiovascular effects of dopamine at various infusion rates

	0.5 to 2 micrograms/ kg/min.	**2 to 10 micrograms/ kg/min.**	**Over 10 micrograms/ kg/min.**
Cardiac output	no change	increase	increase
Stroke volume	no change	increase	increase
Heart rate	no change	there is an initial increase	
		followed by a decrease toward	
		normal rate as infusion continues.	
Myocardial			
contractility	no change	increase	increase
Potential for excessive	low*	low*	data
myocardial oxygen	coronary blood	coronary blood	unavailable
demands	flow increased	flow increased	
Potential for			
tachyarrhythmias	low*	low*	moderate
Total systemic	slight decrease	no change	increase
vascular resistance	to no	to slight	
	change	increase	
Renal blood flow	increase	increase	decrease
Urine output	increase	increase	decrease

*Low but needs monitoring

Clinical trials

No data available.

5.2 Pharmacokinetic Properties

Absorption

The steady state blood levels following intravenous infusion have not been determined in any species, nor has the time for these to be achieved.

Distribution

Dopamine is widely distributed in the body.

Protein binding – No information is available for humans or animals (however, dopamine is rapidly metabolized and excreted).

Metabolism

Dopamine is metabolized in the liver, kidneys and plasma and the metabolites are excreted by the kidneys. The major routes of metabolism are deamination by monoamine oxidase and formation of methylated and reduced derivatives by catechol-o-methyl transferase.

On infusion of ^{14}C labelled dopamine into humans, it was found that approximately 75% of the infused dopamine was rapidly converted into metabolites of dopamine and 25% was synthesized into noradrenaline and its metabolic products. Only a trace of unlabelled adrenaline was detected. The principal metabolite of dopamine was 3-methoxy-4-hydroxy phenylethanol (18.6% of an infused dose) and the principal metabolites of noradrenaline were normetanephrine and 3-4-dihydroxy-mandelic acid.

Excretion

97% of the infused dose of ^{14}C labelled dopamine appeared in urine as metabolites. The metabolites of both dopamine and noradrenaline appear to be at least partially secreted (70% of an infused dose has been found to be secreted per 10 minutes infusion period). The degree of active excretion of dopamine is about the same as for adrenaline and noradrenaline and is inhibited by probenecid.

Onset of action

5 minutes, with a duration of action of less than 10 minutes (in patients receiving monoamine oxidase inhibitors the duration of action may be as long as 1 hour).

Half-life

Approximately 2 minutes after an intravenous bolus (due to rapid metabolism and excretion).

Clinical implication of pharmacokinetic data

Dopamine should be given by continuous infusion because of the rapid metabolism and excretion of the drug.

5.3 Preclinical Safety Data

Genotoxicity

The genotoxic potential of dopamine has not been evaluated.

Carcinogenicity

Long term studies in animals have not been performed to evaluate the carcinogenic potential of dopamine.

6.0 PHARMACEUTICAL PARTICULARS

6.1 Shelf-Life

Please see outer package for the expiry date.

6.2 Storage Conditions

Store at temperatures not exceeding 30°C.

6.3 Availability

USP Type I Clear Glass Ampoule x 5 mL (Box of 5's in plastic tray).

6.4 Incompatibilities

Dopamine should not be added to sodium bicarbonate and other alkaline solutions (see **Section 4.4 Special warnings and precautions for use**) as they will inactivate dopamine. If sodium bicarbonate is simultaneously indicated to treat acidosis, it should be given through a separate infusion line from a separate container.

Dopamine is incompatible with ampicillin or amphotericin B, so should not be mixed with either of these drugs. Dopamine decomposes when mixed with ampicillin in 5% glucose solution, because the solution is alkaline. A precipitate forms immediately on mixing dopamine with amphotericin B in 5% glucose solution. Also refer to **Section 4.5 Interactions with other medicines and other forms of interactions**.

7.0 FDA REGISTRATION NUMBER

40 mg/mL (200 mg/ 5 mL) Concentrate Solution for I.V. Infusion- DR-XY25101

8.0 DATE OF FIRST AUTHORIZATION/RENEWAL OF THE AUTHORIZATION

40 mg/mL (200 mg/ 5 mL) Concentrate Solution for I.V. Infusion- 20 December 2017

Keep out of reach of children.

For suspected adverse drug reaction, report to the FDA: www.fda.gov.ph

Seek medical attention immediately at the first sign of any adverse drug reaction.

CAUTION: Foods, Drugs, Devices and Cosmetics Act prohibits dispensing without prescription.

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