Product Summary

1. Trade Name of the Medicinal Product

Sterile Dopamine Concentrate BP 200mg/5ml

2. Qualitative and Quantitative Composition

Dopamine Hydrochloride BP 40mg/ml

3. Pharmaceutical Form

Solution for injection

Clinical Particulars

4.1. Therapeutic Indications

Dopamine hydrochloride is used for the correction of poor perfusion, low cardiac output, impending renal failure and shock associated with myocardial infarction, trauma, endotoxic septicaemia, open heart surgery and heart failure.

4.2. Posology and Method of Administration

Preparation

The normal usage is a concentration of 1,600mcg/ml of dopamine hydrochloride in an infusion fluid. This may be prepared by aseptically transferring solution equivalent to 800mg of dopamine hydrochloride into 500ml of one of the following sterile intravenous solutions.

- Sodium chloride intravenous infusion BP
- Sodium chloride and dextrose intravenous infusion BP
- 5% dextrose intravenous infusion BP
- 5% dextrose in compound sodium lactate intravenous infusion BP
- Compound sodium lactate intravenous infusion BP (Hartmann’s Lactate Ringer)

Dilution should be made just before administration, if possible. However, the infusion dilutions are stable for at least 24 hours if necessary.
NB. Do not add dopamine hydrochloride to sodium bicarbonate or other alkaline solution as drug decomposition will occur.

Dosage and administration

After dilution, dopamine should be administered through an intravenous catheter or needle in as large a vein as possible. It is essential to use a IV drop chamber or infusion pump so that the rate of infusion may be controlled in drops (ml) per minute. The infusion rate should be adjusted according to the patient's condition. Where appropriate, restoration of the blood volume with a suitable plasma expander should take place before dopamine therapy.

In patients likely to respond to modest increments in heart force and renal perfusion, begin dopamine infusion at the rate of 2mcg/kg/min. In more seriously ill patients begin dopamine infusion at 5mcg/kg/min.

In all cases the dosage should be increased if required in steps of 5–10 mcg/kg/min until the optimum dosage for the particular patient is reached, as judged by increases in blood pressure, urine flow and perfusion generally.

Doses as high as 50mcg/kg/min and above have been used successfully although at these levels urine output should be checked frequently, and should it fail, dose reduction should be considered.

Route of administration

Intravenous infusion

4.3. Contra-indications

Dopamine should not be used in patients with phaeochromocytoma and tachyarrhythmias.

4.4. Special Warnings and Precautions for Use

None known.

4.5. Interactions with other Medicaments and other forms of Interaction
Concurrent use of monoamine oxidase inhibitors (MAOI) and dopamine may intensify and prolong its effects. Patients receiving MAOI within 2-3 weeks prior to dopamine administration, the initial dose should be reduced to at least one-tenth of the usual dose.

The cardiac effects of dopamine are antagonised by β-adrenergic blocking agents, and peripheral vasoconstriction caused by high doses of dopamine is antagonised by α-adrenergic blocking agents.

When dopamine is administered during halothane or cyclopropane anaesthesia ventricular arrhythmias may occur.

Administration of IV phenytoin to patients receiving dopamine has resulted in hypertension and hypotension and bradycardia.

Dopamine should not be used in patients receiving ergot alkaloids because of the possibility of excessive peripheral vasoconstriction. Tricyclic antidepressants and guanethidine may potentiate the pressor responses to dopamine.

Dopexamine may attenuate the positive inotropic effect of higher doses of dopamine.

Doaminergic drugs (entacapone) may enhance the effects of dopamine when both drugs are given simultaneously.

Doxapram may cause hypertension in patients receiving dopamine.

4.6. Pregnancy and Lactation

No evidence of teratogenicity has been found in animal tests, but as with all drugs in pregnancy, the expected benefits to the patients should be weighed against the possible risks to the foetus.

4.7. Effects on Ability to Drive and Use Machines

Not applicable

4.8. Undesirable Effects
Adverse reactions to dopamine are related to its pharmacological action. As dopamine is a normal body product, immune mediated reactions are not seen.

The most frequently reported reactions have been ectopic beats, tachycardia, anginal pain, palpitations, dyspnoea, nausea, vomiting, hypotension and vasoconstriction.

Other rarely reported reactions include aberrant conduction bradycardia, piloerection, widened QRS complex, azotaemia and hypertension. Vomiting rarely occurs. However, should it occur, electrolyte replacement should be given.

Hypovolaemia should be corrected where necessary prior to dopamine infusion. Vasconstriction can occur due to the alpha adrenergic actions of dopamine, particularly in patients with a history of occlusive vascular disease. If required, this condition can be rapidly reversed by reducing or discontinuing the infusion, as dopamine has a half-life of less than 2 minutes.

Extravasation at the site of infusion can cause local vasoconstriction, hence it is desirable to infuse into a large vein. Any resulting ischaemia can be reversed by infiltration of the affected area with 10 – 15ml of saline containing 5-10mg of phentolamine mesylate. A syringe with a fine hypodermic needle should be used to infiltrate the ischaemic area liberally as soon as extravasation is noted.

4.9. Overdose

Accidental overdose, as evidenced by excessive blood pressure elevation, can be controlled by reduction or discontinuation of the infusion for as short period due to the short duration of action of the drug. If this is insufficient, an infusion of phentolamine mesylate should be considered.

Pharmacological Properties

5.1. Pharmacodynamic Properties

Dopamine hydrochloride exerts differing effects according to the rate of infusion.

At 1-5mcg/kg/min, dopamine dilutes the renal mesenteric vascular beds by action on so called “dopaminergic” receptors, causing increases in blood flow glomular filtration rate, sodium excretion and urine output.
At 5-20 mcg/kg/min, dopamine exerts a direct intropic effect on the myocardium causing dose related increases in cardiac output with minimal effect on heart rate. There are usually increases in blood pressure and further increases in urine output.

At 20 mcg/kg/min and above, dopamine increases cardiac output still further with a rise in blood pressure by alpha adrenergic action on peripheral blood vessels. However, even at these higher doses, renal blood flow is higher than before commencement of therapy.

5.2. Pharmacokinetic Properties

The vasoconstrictor properties of dopamine preclude its administration by subcutaneous or intramuscular administration. Like adrenaline it is inactive when given by mouth, and it is rapidly inactivated in the body by similar processes, with a half-life of about 2 minutes. Dopamine is a metabolic precursor of noradrenaline and a proportion is excreted as the metabolic products of noradrenaline. Nevertheless, the majority appears to be directly metabolised into dopamine-related metabolic products.

5.3. Preclinical Safety Data

Not applicable.

Pharmaceutical Particulars

6.1. List of Excipients

Sodium Metabisulphite BP
Water for Injections BP
May contain Sodium Hydroxide or Hydrochloric Acid.

6.2. Incompatibilities

Dopamine hydrochloride should not be added to sodium bicarbonate or other alkaline solution as drug decomposition will occur.

6.3. Shelf Life
3 years unopened.

6.4. Special Precautions for Storage

Do not store above 25ºC. Protect from light. Do not use if discoloured.

6.5. Nature and Contents of Container

Clear glass 5ml ampoules in cartons of 10.

6.6. Instruction for Use/Handling

Not applicable

Administrative Data

7. Marketing Authorisation Holder

Martindale Pharmaceuticals Ltd
Bampton Road
Romford RM3 8UG
England

8. Marketing Authorisation Number

PL 0156/0087

9. Date of First Authorisation/Renewal of Authorisation

2nd May 1998
10. Date of (Partial) Revision of the Text

May 2001