
DBL™ DOBUTAMINE HYDROCHLORIDE INJECTION

Name of medicine

Dobutamine Hydrochloride

Presentation

DBL™ Dobutamine Hydrochloride Injection is a sterile solution containing in each 20 mL vial, Dobutamine Hydrochloride USP 280.2 mg (250 mg Dobutamine equivalent) and Sodium Metabisulfite BP 4.4 mg.

Uses

Actions

Dobutamine hydrochloride is a direct acting inotropic agent whose primary activity results from stimulation of cardiac adrenergic receptors; it produces comparatively mild chronotropic, hypertensive, arrhythmogenic, and vasodilator effects. In contrast with dopamine, it does not release norepinephrine and its actions are not dependent on norepinephrine stores in the heart.

In animal studies, dobutamine hydrochloride produces less increase in heart rate and less decrease in peripheral vascular resistance for a given inotropic effect than does isoproterenol.

In humans, dobutamine hydrochloride increases stroke volume and cardiac output and decreases ventricular filling pressure and total systemic and pulmonary vascular resistances. The ventricular function curve is shifted upwards and to the left as a reflection of increased myocardial contractility.

Heart rate is not increased significantly by the usual dosage of dobutamine hydrochloride; however, significant tachycardia may occur with high doses (usually greater than 10 mcg/kg/min).

Arterial blood pressure usually is not changed significantly by dobutamine hydrochloride because the effect of the increase in cardiac output is balanced by the concomitant decrease in peripheral vascular resistance. Both increments and decrements in arterial blood pressure have been reported. Patients with pre-existing arterial hypertension, even those who are normotensive at the time, seem more susceptible to sustaining a pressor response.

In animals, dobutamine hydrochloride has been shown to decrease pulmonary hypoxic vasoconstriction. This may result in increased perfusion of poorly ventilated areas. This effect may decrease arterial oxygen saturation in some patients, but to a lesser extent than with dopamine or isoproterenol. Due to the increased cardiac output in such patients, oxygen transport is generally increased by dobutamine hydrochloride. Dobutamine hydrochloride has been shown to prevent or to revert partially the decrease in cardiac output that occurs in patients during mechanical ventilation with positive end-expiratory pressure (PEEP).

Dobutamine hydrochloride does not act at dopamine receptors; thus it does not selectively dilate renal or splanchnic vessels. Dobutamine hydrochloride may improve renal blood flow, glomerular filtration rate, urine flow, and sodium excretion by increasing cardiac output and by nonselective vasodilatation.

Dobutamine hydrochloride also exhibits inotropic effects in children, but the haemodynamic response is somewhat different than that in adults. Although cardiac output increases in children, there is a tendency for systemic vascular resistance and ventricular filling pressure to decrease less and for heart rate and arterial blood pressure to increase more in children than in adults. Pulmonary wedge pressure may increase during infusion of dobutamine hydrochloride in children 12 months of age or younger.

Facilitation of atrioventricular conduction has been observed during administration of dobutamine hydrochloride in human electrophysiologic studies and in patients with atrial fibrillation.

Like all inotropic agents, dobutamine hydrochloride increases myocardial oxygen consumption. Dobutamine hydrochloride also increases coronary blood flow and myocardial oxygen supply. The changes in oxygen demand are dependent on several factors, including the following: (a) changes in ventricular diameter, which, in turn, determines the level of wall tension required to generate

intraventricular pressure during systole; (b) changes in afterload, generally proportional to changes in systolic blood pressure; and (c) changes in heart rate. When the use of an inotropic agent in a patient with a failing, dilated heart results in a decrease in ventricular diameter, oxygen demand may increase only slightly or not at all, provided afterload and heart rate do not increase markedly. In general, dobutamine hydrochloride does not cause an imbalance between oxygen consumption and supply in either animals or humans with heart disease. Increments in oxygen delivery have often exceeded the augmentation in oxygen uptake during infusion of dobutamine hydrochloride, so that oxygen saturation in coronary sinus blood increases. The arteriovenous extraction ratio of lactic acid, an indirect evidence of unimpeded aerobic metabolism, is generally maintained during administration of dobutamine hydrochloride. In some instances, myocardial lactate extraction has decreased. Net lactate production has been reported in a few patients; this has occurred especially when heart rate and/or arterial blood pressure have increased excessively during infusion of dobutamine hydrochloride, or when ventricular dysfunction was not present prior to the administration of dobutamine hydrochloride.

In patients with angina pectoris who do not have heart failure, infusions of dobutamine hydrochloride have mimicked the effects of physical exercise, increasing myocardial oxygen demand in excess of coronary oxygen supply, and thereby producing reversible clinical signs of myocardial ischaemia. These signs have included anginal pain, ST segment depression, thallium scintigraphic perfusion defects, and new wall motion abnormalities.

Myocardial infarct size and the incidence and severity of ventricular arrhythmias were not increased in patients with acute myocardial infarction who were treated with dobutamine hydrochloride for 24 hours, as compared to similar patients who did not receive dobutamine hydrochloride. In animals, administration of dobutamine hydrochloride shortly after the ligation of coronary arteries reduces infarct size, when compared to controls receiving saline solution or dopamine. In other animals with experimental infarction who were given dobutamine hydrochloride at doses that increased both heart rate and myocardial contractility, there were electrocardiographic signs of increased ischaemia. Recent studies in animals suggest that functional deterioration and possible enlargement of experimental myocardial lesions during the administration of inotropic medicines including dobutamine hydrochloride is related to their chronotropic effect rather than to the positive inotropism. When dobutamine hydrochloride was infused at doses that produced significant inotropic effect with a minimal increase in heart rate, there was no evidence of enhanced myocardial damage.

Infusions of dobutamine hydrochloride for less than one hour in patients with congestive heart failure increase cardiac output and decrease pulmonary wedge pressure; however, haemodynamic improvements are not accompanied by increases in exercise tolerance. By contrast, longer infusions (up to 72 hours) or infusions that are repeated at regular intervals over several weeks or months do increase exercise tolerance and improve clinical status. This is true even though resting ventricular function is not always augmented.

The mechanism of the sustained improvement in ventricular function following prolonged or intermittent infusions of dobutamine hydrochloride is not understood. However, in studies involving prolonged infusions of dobutamine hydrochloride in humans, mitochondrial ultrastructural and biochemical changes have been reported, suggesting a basis for the protracted amelioration.

Dobutamine hydrochloride has been used in combination with dopamine. In general, the combination does not increase cardiac output more than does an equivalent dose of dobutamine hydrochloride alone. However, the combination of dobutamine hydrochloride and dopamine (a) increases systemic arterial pressure (which would be beneficial to hypotensive patients), (b) increases renal blood flow, urine flow, and sodium excretion, and (c) prevents the increase in ventricular filling pressure that tends to occur with dopamine alone, thus decreasing the risk of pulmonary congestion and oedema, especially in patients with compromised left ventricular function.

Dobutamine hydrochloride has also been used in combination with other vasodilators such as nitroglycerin or nitroprusside, especially in patients with ischaemic heart disease. This combination potentiates the increment in cardiac output and the decrement in systemic vascular resistance and ventricular filling pressure observed with either medicine alone. The heart rate-blood pressure product is either minimally increased or not changed by the concomitant administration of dobutamine hydrochloride and a vasodilator.

Dobutamine hydrochloride is a β -adrenergic agonist. Accordingly, its effects may be counteracted by β -adrenergic receptor antagonists. During treatment with β -antagonists, low doses of dobutamine hydrochloride will manifest varying degrees of β -adrenergic activity, such as vasoconstriction. Because the interaction between both dobutamine hydrochloride and the antagonists on the β receptors is reversible, these 2 drug classes will compete among themselves. Thus, higher doses of dobutamine hydrochloride will progressively counteract the effect of β -adrenergic receptor antagonists.

Pharmacokinetics

Although the onset of action of dobutamine hydrochloride is within 1 to 2 minutes, as much as 10 minutes may be required to reach steady state plasma concentrations and peak effects with any given infusion rate. Steady state plasma concentrations are linearly related to infusion rates. At an infusion rate of 5 mcg/kg/min, the mean plasma concentration is approximately 100 ng/mL in patients with congestive heart failure.

Plasma clearance of dobutamine hydrochloride in humans is 2.4 L/min/m², the volume of distribution is about 20% of body weight, and plasma elimination half-time is less than 3 minutes. The principal routes of disposition include methylation followed by conjugation. Metabolites are eliminated by renal and biliary mechanisms. In human urine, the major excretion products include conjugates of dobutamine and 3-O-methyl dobutamine. The 3-O-methyl derivative is inactive.

Partial tolerance to dobutamine hydrochloride develops during prolonged continuous infusions and becomes statistically significant at 72 hours. The cardiac output response to a constant infusion of dobutamine hydrochloride at 72 hours is over 70% of that obtained at the end of 2 hours in patients with congestive heart failure. This phenomenon may be caused by a decrease (down-regulation) in the number of β -adrenergic receptors.

Alteration of synaptic concentrations of catecholamines with either reserpine or tricyclic antidepressants does not alter the actions of dobutamine hydrochloride in animals; dobutamine hydrochloride acts directly, and its effects are not dependent on presynaptic mechanisms.

Indications

Dobutamine hydrochloride is indicated when inotropic support is necessary for the treatment of patients with hypoperfusion states in whom cardiac output is insufficient to meet circulatory demands. Dobutamine hydrochloride is also indicated when inotropic support is required for the treatment of patients in whom abnormally increased ventricular filling pressures introduce the risk of pulmonary congestion and oedema. Conditions which may precipitate such situations include the following hypoperfusion states:

Initially cardiac in origin

A. Acute heart failure

1. Acute myocardial infarction
2. Cardiogenic shock
3. Following cardiac surgery
4. Medicine-induced depression of cardiac contractility such as that which occurs in excessive β -adrenergic receptor blockade.

B. Chronic heart failure

1. Acute decompensation of chronic congestive heart failure
2. Temporary inotropic support in advanced chronic congestive heart failure, as an adjunct to therapy with conventional oral inotropic agents, systemic vasodilators, and diuretics.

Initially noncardiac in origin

1. Acute hypoperfusion states secondary to trauma, surgery, sepsis, or hypovolaemia when mean arterial pressure is above 70-mm Hg and pulmonary capillary wedge pressure is 18-mm Hg or greater, with inadequate response to volume repletion and increased ventricular filling pressure
2. Low cardiac output secondary to mechanical ventilation with positive end-expiratory pressure (PEEP).

Dobutamine hydrochloride may be used as a substitute for physical exercise in stress testing in the diagnosis of coronary artery disease. When dobutamine hydrochloride is used for this purpose, as is the case when exercise is used for stress testing, patients should be informed of the potential risks involved

in the test. In addition, patients should be subjected to the same close monitoring that is mandatory in standard exercise stress tests, including continuous electrocardiographic monitoring.

Dosage and administration

Administration

Because of its short half-life, dobutamine hydrochloride must be administered as a continuous intravenous infusion. Following the initiation of a constant rate infusion, or upon changing the rate, a steady-state dobutamine plasma concentration is achieved within approximately 10 minutes. Thus, loading doses or bolus injections are not necessary and are not recommended.

Recommended Dosage

The rate of infusion needed to increase cardiac output has ranged from 2.5 to 10 mcg/kg/min in the majority of patients. Frequently, doses up to 20 mcg/kg/min are required for adequate haemodynamic improvement. On rare occasions, infusion rates up to 40 mcg/kg/min have been reported.

The rate of administration and the duration of therapy should be adjusted according to the patient's response, as determined by the following clinical indicators: haemodynamic parameters such as heart rate and rhythm, arterial pressure, and, whenever possible, cardiac output and measurements of ventricular filling pressures (central venous, pulmonary capillary wedge, and left atrial), and signs of pulmonary congestion and organ perfusion (urine flow, skin temperature, and mental status).

Concentrations up to 5,000 mg/L have been administered to humans (250 mg/50 mL). The final volume administered should be determined by the fluid requirements of the patient.

Rather than abruptly discontinuing therapy with dobutamine hydrochloride, it is often advisable to decrease the dosage gradually.

Dosage Units

Most reports on dobutamine hydrochloride have expressed the dose in relation to body mass, for example, mcg/kg/min. This practice is useful to relate doses in infants and children to those in adults. Among adults, body mass has little influence on the effect of dobutamine hydrochloride; since the dosage of dobutamine hydrochloride should be titrated in each patient, adults may be as easily dosed with mcg/min units. The dosage of dobutamine hydrochloride may be initiated at 100 to 200 mcg/min and increased gradually to 1000 to 2000 mcg/min or greater, depending on the clinical and haemodynamic response of the individual patient.

Rates of Infusion Based on Concentration of Dobutamine Hydrochloride

The rates of fluid infusion that are required to deliver specific dosages are a function of the concentration of dobutamine hydrochloride in the infusate. The following table provides a guideline of infusion rates (mL/kg/min) required for 3 frequently used concentrations of dobutamine hydrochloride (250, 500, and 1000 mg/L) in order to deliver the medicine dosages (mcg/kg/min) which are indicated in the left hand column of the table.

Medicine Delivery Rate (mcg/kg/min)	Infusion Delivery Rate		
	250 mg/L * (mL/kg/min)	500 mg/L † (mL/kg/min)	1000 mg/L ‡ (mL/kg/min)
2.5	0.01	0.005	0.0025
5	0.02	0.01	0.005
7.5	0.03	0.015	0.0075
10	0.04	0.02	0.01
12.5	0.05	0.025	0.0125
15	0.06	0.03	0.015
* 250 mg/L of diluent † 500 mg/L or 250 mg/500 mL of diluent ‡ 1000 mg/L or 250 mg/250 mL of diluent			

DBL™ Dobutamine Hydrochloride Injection when diluted to 250 micrograms/mL and 500 micrograms/mL with 0.9% Sodium Chloride Injection and 5% Glucose Injection, was found to be stable for 24 hours at room temperature and in the presence of fluorescent light.

Contraindications

Dobutamine hydrochloride is contraindicated in patients who have shown previous manifestations of hypersensitivity to dobutamine hydrochloride.

Warnings and precautions

Increase in Heart Rate or Arterial Blood Pressure

Dobutamine hydrochloride may cause an increase in heart rate or blood pressure, especially in systolic pressure. Approximately 10% of the patients in clinical studies have had rate increments of 30 beats/min or more, and about 7.5% have had a 50-mm Hg or greater increase in systolic pressure. Reduction of dosage usually reverses these effects promptly. Patients with pre-existing hypertension are more likely to develop an exaggerated pressor response.

Increased Atrioventricular Conduction

Because dobutamine hydrochloride facilitates atrioventricular conduction, patients with atrial flutter or fibrillation may develop rapid ventricular responses.

Ventricular Tachyarrhythmias

Dobutamine hydrochloride may precipitate or exacerbate ventricular ectopic activity; rarely has it caused ventricular tachycardia or fibrillation.

Impaired Ventricular Filling and Ventricular Outflow Obstruction

Inotropic agents, including dobutamine hydrochloride, do not improve haemodynamics in most patients with mechanical obstruction that hinders either ventricular filling or outflow, or both. Inotropic response may be inadequate in patients with markedly reduced ventricular compliance. Such conditions are present in cardiac tamponade, valvular aortic stenosis, and idiopathic hypertrophic subaortic stenosis. Beneficial inotropic effects may be seen in some patients if the heart is dilated or under excessive effect of β -adrenergic receptor antagonists.

Hypersensitivity

Reactions suggestive of hypersensitivity associated with the administration of dobutamine hydrochloride, including skin rash, fever, eosinophilia, and bronchospasm, have been reported occasionally.

Allergic Reaction

DBL™ Dobutamine Hydrochloride Injection contains sodium bisulfite, a sulfite that may cause allergic-type reactions, including anaphylactic symptoms and life-threatening or less severe asthmatic episodes, in certain 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 in nonasthmatic people.

General

During the administration of dobutamine hydrochloride, as with any parenteral catecholamine, heart rate and rhythm, arterial blood pressure, and infusion rate should be monitored closely. When initiating therapy, electrocardiographic monitoring is advisable until a stable response is achieved.

Hypovolaemia should be corrected before treatment with dobutamine hydrochloride is instituted.

No improvement may be observed in the presence of marked mechanical obstruction, such as severe valvular aortic stenosis.

Usage for Heart Failure Complicating an Acute Myocardial Infarction

Although the treatment of heart failure and the reduction in cardiac diameter will decrease myocardial oxygen consumption, there is still concern that the use of any inotropic agent may increase myocardial oxygen demand and the size of an infarction by intensifying ischaemia. Clinical and experimental evidence with dobutamine hydrochloride following acute myocardial infarction suggests that dobutamine hydrochloride does not have an adverse effect on the myocardium when used in doses that do not cause excessive increments in heart rate or arterial pressure. The dose of dobutamine hydrochloride should be titrated to prevent an excessive increase in heart rate and systolic blood pressure.

Usage in Hypotension

When hypotension is largely attributable to decreased cardiac output and coincides with an elevated ventricular filling pressure, the infusion of dobutamine hydrochloride may help restore the pressure.

As volume is replenished in the treatment of acute hypoperfusion states and there is an increase in pulmonary wedge or central venous pressures but with no increase in cardiac output and arterial pressure, dobutamine hydrochloride may improve output and help restore the arterial pressure.

In general, when mean arterial blood pressure is less than 70-mm Hg in the absence of an increased ventricular filling pressure, hypovolaemia may be present and would require treatment with appropriate volume repleting solutions before dobutamine hydrochloride is given.

If arterial blood pressure remains low or decreases progressively during administration of dobutamine hydrochloride despite adequate ventricular filling pressure and cardiac output, consideration may be given to the concomitant use of a peripheral vasoconstrictor agent such as dopamine or norepinephrine.

Pregnancy and Lactation

Reproduction studies performed in rats at doses up to 3.5 times the normal human dose (10 mcg/kg/min for 24 h, total daily dose of 14.4 mg/kg) and in rabbits at doses up to 2 times the normal human dose have revealed no evidence of harm to the foetus or teratogenic effects due to dobutamine hydrochloride. Since there are no adequate and well-controlled studies in pregnant women, and since animal reproduction studies are not always predictive of human response, dobutamine hydrochloride should not be used during pregnancy unless the potential benefits outweigh the potential risks to the foetus.

It is not known whether this medicine is excreted in human milk. Because many medicines are excreted in human milk, caution should be exercised when dobutamine hydrochloride is administered to a nursing woman. If a mother requires dobutamine treatment, breastfeeding should be discontinued for the duration of the treatment.

Paediatric Use

Dobutamine hydrochloride has been administered to children with low-output hypoperfusion states resulting from decompensated heart failure, cardiac surgery, and cardiogenic and septic shock. Some of the haemodynamic effects of dobutamine hydrochloride may be quantitatively or qualitatively different in

children as compared to adults (see **Clinical Pharmacology**). Increments in heart rate and blood pressure appear to be more frequent and intense in children. Pulmonary wedge pressure may not decrease in children, as it does in adults, or it may actually increase, especially in infants less than one year old. Accordingly, the use of dobutamine hydrochloride in children should be monitored closely, bearing in mind these pharmacodynamic characteristics.

Effects on ability to drive and use machines

DBL™ Dobutamine Hydrochloride Injection may be likely to produce minor or moderate adverse effects that may impair the patient's ability to concentrate and react and therefore constitute a risk in the ability to drive and use machines.

Other

Carcinogenesis, Mutagenesis, Impairment of Fertility

Lifetime studies in animals have not been performed to evaluate carcinogenic potential. No mutagenic potential was observed in a bacterial mutation assay. Animal studies to evaluate effects on fertility have not been conducted.

Adverse effects

Heart Rate, Blood Pressure, and Ventricular Ectopic Activity

A 10- to 20-mm Hg increase in systolic blood pressure and an increase in heart rate of 5 to 15 beats/minute have been noted in many patients (see Warnings regarding exaggerated chronotropic and pressor effects). Approximately 5% of patients have had increased premature ventricular beats during infusions of dobutamine hydrochloride. These effects are usually dose related.

Hypotension

Precipitous decreases in blood pressure have occasionally been described in association with dobutamine therapy. Decreasing the dose or discontinuing the infusion typically results in rapid return of blood pressure to baseline values. In rare cases, however, intervention may be required and reversibility may not be immediate.

Reactions at Sites of Intravenous Infusion

Phlebitis has occasionally been reported. Local inflammatory changes have been described following inadvertent infiltration. Isolated cases of cutaneous necrosis (destruction of skin tissue) have been reported.

Miscellaneous Uncommon Effects

The following adverse effects have been reported in 1% to 3% of patients: nausea, headache, anginal pain, nonspecific chest pain, palpitations, and shortness of breath. Isolated cases of thrombocytopenia have been reported.

Administration of Dobutamine, like other catecholamines, can produce a mild reduction in serum potassium concentration, rarely to hypokalaemic levels (see **Precautions**).

Long-Term Safety

Infusions for up to 72 hours have revealed no adverse effects other than those seen with shorter infusions. There is evidence that partial tolerance develops with continuous infusions of dobutamine hydrochloride for 72 hours or more; therefore, higher doses may be required to maintain the same effects.

Interactions

The potency of dobutamine hydrochloride may be decreased if the patient is given β -adrenergic receptor antagonists. In such a case, the unopposed α -agonist effects of dobutamine hydrochloride may become apparent, including peripheral vasoconstriction and hypertension. Conversely, α -adrenergic blockade may make the β -1 and β -2 effects apparent, resulting in tachycardia and vasodilatation.

There has been no overt indication of medicine interactions in clinical studies in which dobutamine hydrochloride was administered concurrently with other medicines, including digitalis preparations, furosemide, spironolactone, lidocaine, nitroglycerin, nitroprusside, isosorbide dinitrate, morphine, atropine, heparin, protamine, potassium chloride, folic acid, and acetaminophen. Pharmacodynamic interactions with dopamine and vasodilators are described in the **Clinical Pharmacology** section.

Laboratory Test

Dobutamine, like other β_2 -agonists, can produce a mild reduction in serum potassium concentration, rarely to hypokalaemic levels. Accordingly, consideration should be given to monitoring serum potassium.

Overdosage

Overdoses of dobutamine have been reported rarely. The following is provided to serve as a guide if such an overdose is encountered.

Signs and Symptoms

Toxicity from dobutamine hydrochloride is usually due to excessive cardiac β -receptor stimulation. The duration of action of dobutamine hydrochloride is generally short ($T_{1/2} = 2$ minutes) because it is rapidly metabolised by catechol-O-methyltransferase. The symptoms of toxicity may include anorexia, nausea, vomiting, tremor, anxiety, palpitations, headache, shortness of breath, and anginal and nonspecific chest pain. The positive inotropic and chronotropic effects of dobutamine on the myocardium may cause hypertension, tachyarrhythmias, myocardial ischaemia, and ventricular fibrillation. Hypotension may result from vasodilation. If the product is ingested, unpredictable absorption may occur from the mouth and the gastrointestinal tract.

Treatment

In managing overdosage, consider the possibility of multiple medicine overdoses, interaction among medicines, and unusual medicine kinetics in your patient.

The initial actions to be taken in a dobutamine hydrochloride overdose are discontinuing administration, establishing an airway, and ensuring oxygenation and ventilation. Resuscitative measures should be initiated promptly. Severe ventricular tachyarrhythmias may be successfully treated with propranolol or lidocaine. Hypertension usually responds to a reduction in dose or discontinuation of therapy.

Protect the patient's airway and support ventilation and perfusion. If needed, meticulously monitor and maintain, within acceptable limits, the patient's vital signs, blood gases, serum electrolytes, etc. Absorption of medicines from the gastrointestinal tract may be decreased by giving activated charcoal, which, in many cases, is more effective than emesis or lavage; consider charcoal instead of or in addition to gastric emptying. Repeated doses of charcoal over time may hasten elimination of some medicines that have been absorbed. Safeguard the patient's airway when employing gastric emptying or charcoal.

Forced diuresis, peritoneal dialysis, haemodialysis, or charcoal haemoperfusion have not been established as beneficial for an overdose of dobutamine hydrochloride.

Pharmaceutical precautions

Incompatibilities

Dobutamine is incompatible with alkaline solutions such as sodium bicarbonate 5%.

Special Precautions for Storage

Store below 25°C. Protect from light.

Medicine classification

Prescription Medicine.

Package quantities

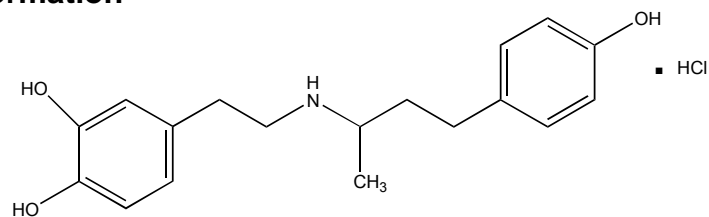
DBL™ Dobutamine Hydrochloride Injection is available as follows:

Strength

250 mg/20 mL (dobutamine equivalent)

Pack Size

Single

Further information $C_{18}H_{25}NO_3 \cdot HCl$

MW = 337.9, CAS REGISTRY NO:- 49745-95-1

(RS)-4-[2-[[3-(4-hydroxyphenyl)-1-methylpropyl]amino]-ethyl] benzene-1,2-diol

hydrochloride

Dobutamine hydrochloride is a white to practically white, crystalline powder. It is sparingly soluble in water and methyl alcohol; soluble in alcohol.

Name and address

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Date of preparation

15 December 2011