Administration in hepatic impairment. The plasma half-life of disopyramide may be increased in hepatic impairment and dosage reduction should be considered; US licensed product information recommends an oral dose of 400 mg daily in divided doses. In patients with liver cirrhosis there is also a significant reduction in the plasma concentration of α_1-acid glycoprotein;[1,2] in addition, its binding capacity for disopyramide is reduced.[1] This is associated with an increase in the free fraction of disopyramide such that measurement of total disopyramide in plasma may not be a safe indicator for dosing, and a therapeutic range 50% lower than in patients with normal hepatic function should be considered.[2]

1. Bonde J, *et al.* Kinetics of disopyramide in decreased hepatic function. *Eur J Clin Pharmacol* 1986; **31:** 73–7.
2. Echizen H, *et al.* Protein binding of disopyramide in liver cirrhosis and in nephrotic syndrome. *Clin Pharmacol Ther* 1986; **40:** 274–80.

Administration in renal impairment. Disopyramide is excreted mainly in the urine and a reduction in clearance with an increase in elimination half-life has been reported[1] in patients with renal impairment. Dosage reduction should therefore be considered. US licensed product information recommends the following oral doses based on creatinine clearance (CC):

- CC greater than 40 mL/minute: 400 mg daily in divided doses
- CC 30 to 40 mL/minute: 100 mg every 8 hours
- CC 15 to 30 mL/minute: 100 mg every 12 hours
- CC less than 15 mL/minute: 100 mg every 24 hours

Modified-release preparations should be avoided in patients with CC less than 40 mL/minute.

At therapeutic concentrations disopyramide is not significantly removed by haemodialysis;[2] the half-life is similar both on and off dialysis (16.8 versus 16.1 hours). An increased free fraction of disopyramide has been seen[3] during haemodialysis associated with an elevation in free fatty acids in plasma and in such cases free plasma-disopyramide concentrations should be monitored.

1. Francois B, *et al.* Pharmacokinetics of disopyramide in patients with chronic renal failure. *Eur J Drug Metab Pharmacokinet* 1983; **8:** 85–92.
2. Sevka MJ, *et al.* Disopyramide hemodialysis and kinetics in patients requiring long-term hemodialysis. *Clin Pharmacol Ther* 1981; **29:** 322–6.
3. Horiuchi T, *et al.* Inhibitory effect of free fatty acids on plasma protein binding of disopyramide in haemodialysis patients. *Eur J Clin Pharmacol* 1989; **36:** 175–80.

Hypertrophic cardiomyopathy. Patients with hypertrophic cardiomyopathy (p.1163) may have exercise intolerance due to left ventricular outflow obstruction. Beta blockers are usually used when symptoms are associated with exercise or emotional factors, but may not be effective in patients with symptoms at rest. Disopyramide has been used for its negative inotropic effect in such patients and a retrospective study[1] found that it improved symptoms without having a proarrhythmic effect.

1. Sherrid MV, *et al.* Multicenter study of the efficacy and safety of disopyramide in obstructive hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2005; **45:** 1251–8.

Hypotension. Disopyramide has been widely used in the management of neurally mediated hypotension (p.1174) but there is limited evidence to support its use. Although some reports[1,2] have suggested benefit, a controlled study[3] found that it was no more effective than placebo in preventing tilt-induced syncope. Adverse effects also limit the use of disopyramide, and it is generally no longer considered first line.

1. Milstein S, *et al.* Usefulness of disopyramide for prevention of upright tilt-induced hypotension-bradycardia. *Am J Cardiol* 1990; **65:** 1339–44.
2. Bhaumick SK, *et al.* Oral disopyramide in the treatment of recurrent neurocardiogenic syncope. *Int J Clin Pract* 1997; **51:** 342.
3. Morillo CA, *et al.* A placebo-controlled trial of intravenous and oral disopyramide for prevention of neurally mediated syncope induced by head-up tilt. *J Am Coll Cardiol* 1993; **22:** 1843–8.

Preparations

BP 2008: Disopyramide Capsules; Disopyramide Phosphate Capsules; ***USP 31:*** Disopyramide Phosphate Capsules; Disopyramide Phosphate Extended-release Capsules.

Proprietary Preparations (details are given in Part 3)
Austral.: Rythmodan; ***Austria:*** Rythmodan; ***Belg.:*** Rythmodan; ***Braz.:*** Dicorantil; ***Canad.:*** Rythmodan; ***Cz.:*** Rythmodan†; ***Denm.:*** Durbis†; ***Fin.:*** Disomet; ***Fr.:*** Isorythm; Rythmodan; ***Ger.:*** Diso-Duriles†; Norpace†; Rythmodul†; ***Gr.:*** Dicorynan; Ritmodan†; Rythmodan; Rythmodul†; ***Hung.:*** Palpitin-PP; ***India:*** Norpace; ***Irl.:*** Rythmodan; ***Israel:*** Rythmical; ***Ital.:*** Ritmodan; ***Jpn:*** Rythmodan; ***Mex.:*** Dimodan; ***Neth.:*** Ritmoforine; Rythmodan†; ***Norw.:*** Durbis; ***NZ:*** Rythmodan; ***Port.:*** Ritmodan†; ***S.Afr.:*** Norpace; Rythmodan; ***Spain:*** Dicorynan; ***Swed.:*** Dirytmin†; Durbis; ***Switz.:*** Norpace†; ***Turk.:*** Norpace; ***UK:*** Rythmodan; ***USA:*** Norpace.

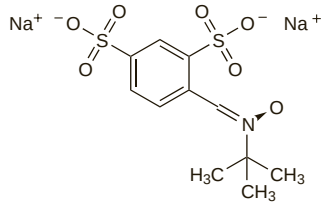
Disufenton Sodium *(USAN, rINN)*

ARL-16556; CPI-22; CXY-059; Disufentón de sodio; Disufenton Sodique; Disufentonum Natricum; NXY-059. Disodium 4-(*tert*-butyliminomethyl)benzene-1,3-disulfonate *N*-oxide.

Дисуфентон Натрия

$C_{11}H_{13}NNa_2O_7S_2$ = 381.3.

CAS — 168021-79-2.



Profile

Disufenton sodium traps free radicals. It has been investigated as a neuroprotectant for acute ischaemic and haemorrhagic stroke but results have been disappointing.

◊ References.

1. Lees KR, *et al.* The Stroke-Acute Ischemic NXY Treatment (Saint I) Trial Investigators. NXY-059 for acute ischemic stroke. *N Engl J Med* 2006; **354:** 588–600.
2. Shuaib A, *et al.* SAINT II Trial Investigators. NXY-059 for the treatment of acute ischemic stroke. *N Engl J Med* 2007; **357:** 562–71.
3. Lyden PD, *et al.* Safety and tolerability of NXY-059 for acute intracerebral hemorrhage: the CHANT trial. *Stroke* 2007; **38:** 2262–9.

Ditazole *(rINN)*

Diethamphenazole; Ditazol; Ditazolum; S-222. 2,2′-[(4,5-Diphenyloxazol-2-yl)imino]diethanol.

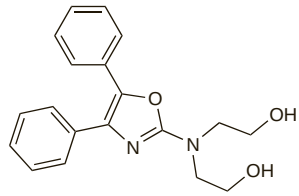
Дитазол

$C_{19}H_{20}N_2O_3$ = 324.4.

CAS — 18471-20-0.

ATC — B01AC01.

ATC Vet — QB01AC01.



Profile

Ditazole is an inhibitor of platelet aggregation used in the management of thromboembolic disorders (p.1187) in doses of 400 mg two or three times daily by mouth.

Preparations

Proprietary Preparations (details are given in Part 3)
Port.: Fendazol†; ***Spain:*** Ageroplas.

Dobutamine Hydrochloride

(BANM, USAN, rINNM)

46236; Compound 81929 (dobutamine); Dobutamiinihydrokloridi; Dobutamine, chlorhydrate de; Dobutamin-hidroklorid; Dobutamin-hydrochlorid; Dobutaminhydroklorid; Dobutamini hydrochloridum; Dobutamino hidrochloridas; Hidrocloruro de dobutamina; LY-174008 (dobutamine tartrate). (±)-4-(2-{[3-(*p*-Hydroxyphenyl)-1-methylpropyl]amino}ethyl)pyrocatechol hydrochloride.

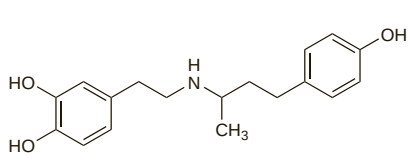
Добутамина Гидрохлорид

$C_{18}H_{23}NO_3$,HCl = 337.8.

CAS — 34368-04-2 (dobutamine); 49745-95-1 (dobutamine hydrochloride); 101626-66-8 (dobutamine tartrate).

ATC — C01CA07.

ATC Vet — QC01CA07.



(dobutamine)

Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), *Jpn*, and *US.*
Ph. Eur. 6.2 (Dobutamine Hydrochloride). A white or almost white crystalline powder. Sparingly soluble in water and in alcohol; soluble in methyl alcohol. Protect from light.

USP 31 (Dobutamine Hydrochloride). A white to practically white crystalline powder. Sparingly soluble in water and in methyl alcohol; soluble in alcohol and in pyridine. Store in airtight containers at a temperature of 15° to 30°.

Incompatibility. Dobutamine is incompatible with alkaline solutions such as sodium bicarbonate 5% and alkaline drugs such as aminophylline, furosemide,[1] and thiopental sodium;[1] physical incompatibility with bumetanide, calcium gluconate, insulin, diazepam, and phenytoin has also been suggested. There have also been reports of incompatibility with alteplase,[2] heparin,[3] and warfarin sodium.[4]

1. Chiu MF, Schwartz ML. Visual compatibility of injectable drugs used in the intensive care unit. *Am J Health-Syst Pharm* 1997; **54:** 64–5.
2. Lee CY, *et al.* Visual and spectrophotometric determination of compatibility of alteplase and streptokinase with other injectable drugs. *Am J Hosp Pharm* 1990; **47:** 606–8.
3. Yamashita SK, *et al.* Compatibility of selected critical care drugs during simulated Y-site administration. *Am J Health-Syst Pharm* 1996; **53:** 1048–51.
4. Bahal SM, *et al.* Visual compatibility of warfarin sodium injection with selected medications and solutions. *Am J Health-Syst Pharm* 1997; **54:** 2599–2600.

Adverse Effects and Treatment

As for Sympathomimetics, p.1407. Dobutamine has mainly beta$_1$-agonist properties and its principal adverse effects include dose-related increases in heart rate and blood pressure, ectopic beats, angina or chest pain, and palpitations; dosage should be reduced or temporarily stopped if they occur. Ventricular tachycardia may occur rarely; cardiac rupture has been reported rarely during dobutamine stress testing.

Effects on body temperature. A 71-year-old woman with heart failure developed a fever on 2 separate occasions 8 to 12 hours after starting an infusion of dobutamine.[1]

1. Robison-Strane SR, Bubik JS. Dobutamine-induced fever. *Ann Pharmacother* 1992; **26:** 1523–4.

Effects on the cardiovascular system. For reference to severe cardiovascular complications of dobutamine stress echocardiography, see Diagnosis and Testing under Uses and Administration, below.

For reference to fatalities occurring in patients given dobutamine, see Heart Failure under Uses and Administration, below.

Effects on the neuromuscular system. Myoclonus has been reported[1,2] in patients with renal impairment given dobutamine infusion for heart failure.

1. Wierre L, *et al.* Dobutamine-induced myoclonia in severe renal failure. *Nephrol Dial Transplant* 2004; **19:** 1336–7.
2. Boord A, Benson B. Myoclonus associated with continuous dobutamine infusion in a patient with end-stage renal disease. *Am J Health-Syst Pharm* 2007; **64:** 2241–3.

Effects on the skin. Troublesome pruritus of the scalp has been reported[1] in a patient receiving dobutamine infusions. It was suggested that this might be a direct effect of dobutamine since the reaction was so localised.

1. McCauley CS, Blumenthal MS. Dobutamine and pruritus of the scalp. *Ann Intern Med* 1986; **105:** 966.

Hypersensitivity. Hypersensitivity reactions have been reported in patients receiving dobutamine infusions, possibly due to sodium sulfite in the formulation. Redness, swelling, itching, and a sensation of warmth developed[1] around the infusion site in a patient receiving dobutamine; the reaction recurred when the infusion was repeated a week later. Eosinophilic reactions have also been reported, including hypersensitivity myocarditis[2-4] and asthma.[5]

1. Cernek PK. Dermal cellulitis—a hypersensitivity reaction from dobutamine hydrochloride. *Ann Pharmacother* 1994; **28:** 964.
2. Spear GS. Eosinophilic explant carditis with eosinophilia: ?hypersensitivity to dobutamine infusion. *J Heart Lung Transplant* 1995; **14:** 755–60.
3. Takkenberg JJM, *et al.* Eosinophilic myocarditis in patients awaiting heart transplantation. *Crit Care Med* 2004; **32:** 714–21.
4. Butany J, *et al.* Hypersensitivity myocarditis complicating hypertrophic cardiomyopathy heart. *Can J Cardiol* 2004; **20:** 911–14.
5. Aranda JM, *et al.* Dobutamine-related asthma in a patient awaiting cardiac transplantation: the eosinophilic dilemma. *J Heart Lung Transplant* 2004; **23:** 260–1.

Overdosage. A patient received an accidental overdose[1] of dobutamine when given an intravenous infusion at a rate of more than 130 micrograms/kg per minute for 30 minutes, this being three times the recommended maximum. Characteristic adverse effects such as emesis, palpitations, chest pain, dyspnoea, and paraesthesia developed, together with urinary incontinence, an effect not previously associated with dobutamine.

1. Paulman PM, *et al.* Dobutamine overdose. *JAMA* 1990; **264:** 2386–7.

Precautions

As for Sympathomimetics, p.1407. Dobutamine has primarily inotropic effects and should be avoided or used only with great caution in patients with marked

obstruction of cardiac ejection, such as idiopathic hypertrophic subaortic stenosis. It should also be used with caution in patients with acute myocardial infarction, and in cardiogenic shock complicated by severe hypotension. Hypovolaemia should be corrected before treatment.

Interference with diagnostic tests. Contamination of blood samples with dobutamine has been reported to produce falsely decreased creatinine values in an enzymatic test.[1] Colorimetric measurements of creatinine were not affected.

1. Daly TM, *et al.* "Bouncing" creatinine levels. *N Engl J Med* 1996; **334:** 1749–50.

Interactions

As for Sympathomimetics, p.1407. Most interactions with dobutamine are due to its direct $beta_1$ agonist effects on the heart, but use with beta blockers may allow its alpha- and $beta_2$-agonist effects to become apparent.

Pharmacokinetics

Like adrenaline (p.1204), dobutamine is inactive when given orally, and it is rapidly inactivated in the body by similar processes. It has a half-life of about 2 minutes. Conjugates of dobutamine and its major metabolite 3-*O*-methyldobutamine are excreted primarily in urine, with small amounts eliminated in the faeces.

◊ The primary mechanism of clearance of dobutamine appears to be distribution to other tissues, and not metabolism or elimination. It has a half-life of about 2 minutes and plasma concentrations of dobutamine reach steady state about 10 to 12 minutes after the start of an infusion. Dobutamine is used mainly for the short-term treatment of heart failure and any pharmacokinetic changes in this condition have no clinical implications in dosage titration.[1]

The pharmacokinetics of dobutamine and other cardiovascular drugs in children have been reviewed.[2]

1. Shammas FV, Dickstein K. Clinical pharmacokinetics in heart failure: an updated review. *Clin Pharmacokinet* 1988; **15:** 94–113.
2. Steinberg C, Notterman DA. Pharmacokinetics of cardiovascular drugs in children: inotropes and vasopressors. *Clin Pharmacokinet* 1994; **27:** 345–67.

Uses and Administration

Dobutamine is a sympathomimetic (p.1408) with direct effects on $beta_1$-adrenergic receptors, giving it a prominent inotropic action on the heart. It also has some alpha- and $beta_2$-agonist properties. Although it is structurally related to dopamine (p.1273), it has no specific dopaminergic properties; however, like dopamine, the inotropic action of dobutamine on the heart is associated with less cardiac-accelerating effect than that of isoprenaline.

Dobutamine is used to increase the contractility of the heart in acute heart failure, as occurs in cardiogenic shock (p.1183) and myocardial infarction (p.1175); it is also used in septic shock. Other circumstances in which its inotropic activity may be useful are during cardiac surgery and positive end-expiratory pressure ventilation.

Dobutamine is used as the hydrochloride but doses are expressed in terms of the base; 1.12 micrograms of the hydrochloride is equivalent to about 1 microgram of base. It is given by intravenous infusion as a dilute solution (0.25 to 5 mg/mL), in glucose 5% or sodium chloride 0.9%; other fluids may also be suitable and the manufacturers' guidelines should be consulted.

In the management of **acute heart failure**, dobutamine is given at a usual rate of 2.5 to 10 micrograms/kg per minute, according to the patient's heart rate, blood pressure, cardiac output, and urine output. A range of 0.5 up to 40 micrograms/kg per minute has occasionally been required. It has been recommended that treatment with dobutamine should be discontinued gradually.

Dobutamine is also used as an alternative to exercise in **cardiac stress testing**. A solution containing 1 mg/mL is given via an infusion pump in a dose of 5 micrograms/kg per minute for 8 minutes. The dose is then increased by increments of 5 micrograms/kg per minute up to a usual maximum of 20 micrograms/kg per minute, with each dose being infused for 8 minutes before the next increase; doses of up to 40 micrograms/kg per minute have sometimes been used. The ECG should be monitored continuously and the infusion stopped if arrhythmias, marked ST segment depression, or other adverse effects occur.

Action. Although dobutamine is usually considered to be a $beta_1$ agonist, *animal* studies suggest that its ability to stimulate $alpha_1$- and $beta_2$-adrenergic receptors may be as great as its $beta_1$-stimulant properties. It has been proposed that the inotropic action results from a combination of alpha-stimulant activity on myocardial $alpha_1$ receptors, a property residing mainly in the (–)-enantiomer, with $beta_1$ stimulation by the (+)-enantiomer; peripherally, alpha-mediated vasoconstriction would be opposed by the $beta_2$-agonist properties of the (+)-enantiomer, resulting in the net inotropic action with relatively little effect on blood pressure seen with the racemic mixture used clinically.[1]

Dobutamine has a thermogenic effect,[2] increasing oxygen delivery and utilisation in healthy individuals. However, using it for this purpose in critically ill patients did not improve patient outcome and in some cases might have been harmful.[3]

1. Ruffolo RR. The mechanism of action of dobutamine. *Ann Intern Med* 1984; **100:** 313–14.
2. Bhatt SB, *et al.* Effect of dobutamine on oxygen supply and uptake in healthy volunteers. *Br J Anaesth* 1992; **69:** 298–303.
3. Hayes MA, *et al.* Elevation of systemic oxygen delivery in the treatment of critically ill patients. *N Engl J Med* 1994; **330:** 1717–22.

Administration in children. Dobutamine and dopamine are both used for inotropic support in children. A study[1] in children undergoing cardiac surgery suggested that dobutamine may be preferred to dopamine since the latter could cause pulmonary vasoconstriction (see under Precautions for Dopamine, p.1273). In preterm infants, one study[2] reported that dobutamine may have a greater effect on systemic blood flow than dopamine, but a systematic review[3] found that dopamine was more effective than dobutamine in the short-term treatment of hypotension although there was insufficient evidence of long-term benefit or safety with either drug for firm recommendations to be made.

1. Booker PD, *et al.* Comparison of the haemodynamic effects of dopamine and dobutamine in young children undergoing cardiac surgery. *Br J Anaesth* 1995; **74:** 419–23.
2. Osborn D, *et al.* Randomized trial of dobutamine versus dopamine in preterm infants with low systemic blood flow. *J Pediatr* 2002; **140:** 183–91.
3. Subhedar NV, Shaw NJ. Dopamine versus dobutamine for hypotensive preterm infants. Available in The Cochrane Database of Systematic Reviews; Issue 3. Chichester: John Wiley; 2003 (accessed 07/10/05).

Diagnosis and testing. Dynamic exercise is the established mode of stress for the assessment of cardiac function. In patients who are unable to exercise, a dobutamine infusion is one of the best alternative ways of producing a pharmacological stress.[1,2] It is widely used as an adjunct in echocardiography, often combined with atropine, and may give better sensitivity than adenosine or dipyridamole;[1,3] it may also have a role with other imaging techniques such as magnetic resonance imaging.[4] However there have been instances of severe cardiovascular complications attributable to dobutamine.[5]

1. Cheitlin MD, *et al.* ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography). Summary article: *Circulation* 2003; **108:** 1146–62. Full text: http://www.americanheart.org/downloadable/heart/1060182581039Echocleanfulltext.pdf (accessed 07/10/05)
2. Marwick TH. Stress echocardiography. *Heart* 2003; **89:** 113–18.
3. Martin TW, *et al.* Comparison of adenosine, dipyridamole, and dobutamine in stress echocardiography. *Ann Intern Med* 1992; **116:** 190–6.
4. Paetsch I, *et al.* Comparison of dobutamine stress magnetic resonance, adenosine stress magnetic resonance, and adenosine stress magnetic resonance perfusion. *Circulation* 2004; **110:** 835–42.
5. Lattanzi F, *et al.* Dobutamine stress echocardiography: safety in diagnosing coronary artery disease. *Drug Safety* 2000; **22:** 251–62.

Heart failure. Dobutamine may be used in the management of acute heart failure, including decompensated chronic heart failure (see Cardiogenic Shock, under Shock, p.1183). It may also have a role in patients with severe chronic heart failure (p.1165), either as a bridge to transplantation or for palliative therapy. In less severe cases, intermittent infusions of dobutamine have been tried. A study[1] using pulsed therapy with dobutamine (30 minutes daily for 4 days each week for 3 weeks) reported symptomatic improvements similar to those achieved with exercise, but another study[2] using intermittent therapy (24 hours every 2 to 3 weeks for 6 months) failed to show any benefit. There have also been reports of sudden death in patients receiving dobutamine as infusions for 48 hours per week, and another study[3] was halted for this reason. Long-term use of intermittent dobutamine is therefore not generally recommended.[4]

1. Adamopoulos S, *et al.* Effects of pulsed β-stimulant therapy on β-adrenoceptors and chronotropic responsiveness in chronic heart failure. *Lancet* 1995; **345:** 344–9.
2. Elis A, *et al.* Intermittent dobutamine treatment in patients with chronic refractory congestive heart failure: a randomized, double-blind, placebo-controlled study. *Clin Pharmacol Ther* 1998; **63:** 682–5.
3. Dies F, *et al.* Intermittent dobutamine in ambulatory outpatients with chronic cardiac failure. *Circulation* 1986; **74:** (suppl II): 38.
4. Hunt SA, *et al.* ACC/AHA 2005 guideline update for the diagnosis and management of chronic heart failure in the adult: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure). Summary article: *J Am Coll Cardiol* 2005; **46:** 1116–43. Full version: http://content.onlinejacc.org/cgi/reprint/46/6/e1.pdf (accessed 19/08/08)

Preparations

BP 2008: Dobutamine Intravenous Infusion;
USP 31: Dobutamine for Injection; Dobutamine in Dextrose Injection; Dobutamine Injection.

Proprietary Preparations (details are given in Part 3)
Arg.: Dobucard; Dobuject†; Dobutrex†; Duvig; ***Austral.:*** Dobutrex; ***Austria:*** Inotop; ***Belg.:*** Dobutrex†; Dobutrexmerck; ***Braz.:*** Biodobutin†; Dobtan; Dobutabbott; Dobutal†; Dobutil†; Dobuton; Dobutrex†; Neobutamina; ***Canad.:*** Dobutrex†; ***Chile:*** Bagobutam†; Dobutrex†; ***Cz.:*** Dobuject; Dobutrex; ***Denm.:*** Dobutrex†; ***Fin.:*** Dobuject; Dobutrex†; ***Fr.:*** Dobutrex†; ***Gr.:*** Dobutan; Inotrex; ***Hong Kong:*** Dobutrex†; ***Hung.:*** Dobutrex†; ***India:*** Dobutrex; ***Indon.:*** Cardiject; Dobuject; Dobutel; Inotrop; ***Irl.:*** Dobutrex†; Posiject†; ***Israel:*** Butamine; Dobuject; Dobutam†; ***Ital.:*** Dobutrex†; Miozac; ***Jpn:*** Dobupum; ***Malaysia:*** Dobucard†; Dobutrex†; ***Mex.:*** Cryobutol; Dobuject; Dobutrex; Kardion†; Oxiken; ***Norw.:*** Dobutrex†; ***NZ:*** Dobutrex†; ***Philipp.:*** Dobuject; Dobutrex; ***Pol.:*** Dobuject; ***Port.:*** Dobucor; Dobutina; Inotrex; ***S.Afr.:*** Cardiject; Dobutrex; Posiject†; ***Singapore:*** Dobuject†; ***Spain:*** Dobucor; Dobutrex†; ***Swed.:*** Dobutrex; ***Switz.:*** Dobutrex; ***Thai.:*** Cardiject; Dobuject; Dobutrex†; ***UK:*** Dobutrex†; Posiject†; ***USA:*** Dobutrex†; ***Venez.:*** Doburan; Dobutrex†; Dobuxin.

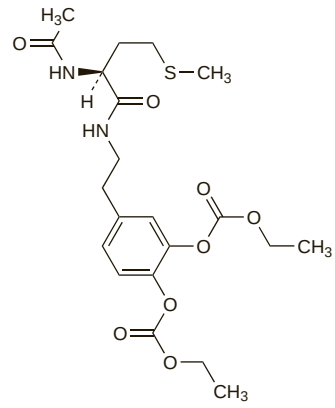
Docarpamine *(rINN)*

Docarpamina; Docarpaminum; TA-870; TA-8704. (–)-(*S*)-2-Acetamido-*N*-(3,4-dihydroxyphenethyl)-4-(methylthio)butyramide bis(ethyl carbonate) ester.

Докарпамин

$C_{21}H_{30}N_2O_8S$ = 470.5.

CAS — 74639-40-0.



Profile

Docarpamine is an orally active prodrug of dopamine (p.1273) that has been used in the treatment of acute heart failure.

Preparations

Proprietary Preparations (details are given in Part 3)
Jpn: Tanadopa†.

Dofetilide *(BAN, USAN, rINN)*

Dofetilid; Dofetilida; Dofétilide; Dofetilidi; Dofetilidum; UK-68798. β-[(*p*-Methanesulfonamidophenethyl)methylamino]methanesulfono-*p*-phenetidide.

Дофетилид

$C_{19}H_{27}N_3O_5S_2$ = 441.6.

CAS — 115256-11-6.

ATC — C01BD04.

ATC Vet — QC01BD04.