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.

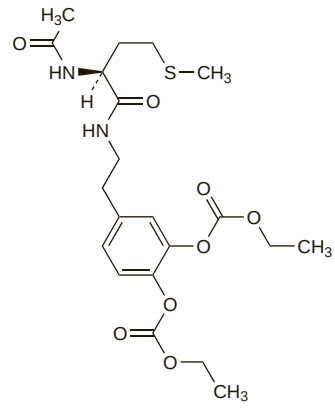
Docarpamine *(rINN)*

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.

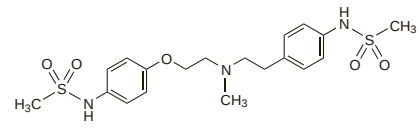
Дофетилид

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

CAS — 115256-11-6.

ATC — C01BD04.

ATC Vet — QC01BD04.



Adverse Effects and Precautions

The most frequent adverse effects with dofetilide are headache, chest pain, and dizziness. Dofetilide prolongs the QT interval and may cause severe ventricular arrhythmias, including torsade de pointes; it should not be given to patients with congenital or acquired long QT syndromes. Hypokalaemia increases the risk of arrhythmias and potassium concentrations should be corrected before starting dofetilide. The dosage of dofetilide must be individualised according to QT interval and creatinine clearance, which must be measured before starting therapy; dofetilide is contra-indicated if the corrected QT interval is above 440 milliseconds (or above 500 milliseconds in patients with ventricular conduction abnormalities) or if the creatinine clearance is less than 20 mL/minute. Treatment should begin under ECG monitoring, which must be continued for at least 3 days, and both ECG and renal function should be monitored at least every 3 months during treatment.

Interactions

Dofetilide should not be given with other drugs that prolong the QT interval. Class I or class III antiarrhythmics should be stopped at least 3 half-lives before dofetilide is given. Potassium-depleting diuretics may cause hypokalaemia or hypomagnesaemia, increasing the potential for torsade de pointes; US licensed product information for dofetilide states that hydrochlorothiazide is contra-indicated since it also causes significant increases in plasma-dofetilide concentrations. Dofetilide is metabolised to a small extent by the cytochrome P450 isoenzyme CYP3A4, and drugs or foods that inhibit this isoenzyme, such as macrolide antibacterials, HIV-protease inhibitors, diltiazem, and grapefruit juice, should be used with caution. Cimetidine, trimethoprim, ketoconazole, prochlorperazine, and megestrol, should not be given as they inhibit the renal excretion of dofetilide; verapamil is also contra-indicated as it too may substantially increase dofetilide concentrations.

◊ References.

1. Yamreudeewong W, *et al.* Potentially significant drug interactions of class III antiarrhythmic drugs. *Drug Safety* 2003; **26:** 421–38.

Pharmacokinetics

The oral bioavailability of dofetilide is more than 90%. Peak plasma concentrations occur after 2 to 3 hours and steady state concentrations after 2 to 3 days. The terminal half-life is about 10 hours. Protein binding is 60 to 70%. Dofetilide undergoes limited metabolism. About 80% of a dose is excreted in the urine, with about 80% of this as unchanged drug and 20% as 5 minimally active or inactive metabolites; metabolism may be mediated to some extent by the cytochrome P450 isoenzyme CYP3A4. Renal elimination involves both glomerular filtration and active tubular secretion via the cation transport system. The clearance of dofetilide decreases with decreasing creatinine clearance.

◊ References.

1. Allen MJ, *et al.* The pharmacokinetics and pharmacodynamics of oral dofetilide after twice daily and three times daily dosing. *Br J Clin Pharmacol* 2000; **50:** 247–53.

Uses and Administration

Dofetilide is a class III antiarrhythmic (p.1153); it selectively blocks one of the potassium channels involved in repolarisation and therefore prolongs the action potential. It is used in the treatment of atrial fibrillation and flutter (p.1160) in patients who are highly symptomatic. The initial oral dose in patients with a corrected QT interval of 440 milliseconds or less is 500 micrograms twice daily; the maintenance dose must be reduced if the QT interval becomes prolonged after the first dose, and treatment should be stopped if the QT interval exceeds 500 milliseconds. Doses should be reduced in renal impairment (see below).

◊ References.

1. McClellan KJ, Markham A. Dofetilide: a review of its use in atrial fibrillation and atrial flutter. *Drugs* 1999; **58:** 1043–59.
2. Kalus JS, Mauro VF. Dofetilide: a class III-specific antiarrhythmic agent. *Ann Pharmacother* 2000; **34:** 44–56.
3. Mounsey JP, DiMarco JP. Dofetilide. *Circulation* 2000; **102:** 2665–70.
4. Roukoz H, Saliba W. Dofetilide: a new class III antiarrhythmic agent. *Expert Rev Cardiovasc Ther* 2007; **5:** 9–19.

Administration in renal impairment. Doses of dofetilide should be reduced in patients with renal impairment based on creatinine clearance (CC). Initial doses are:

- CC 40 to 60 mL/minute: 250 micrograms twice daily
- CC 20 to 39 mL/minute: 125 micrograms twice daily
- CC below 20 mL/minute: contra-indicated

Preparations

Proprietary Preparations (details are given in Part 3)
USA: Tikosyn.

Dopamine Hydrochloride

(BANM, USAN, pINNM)

ASL-279; Dopamiinihydrokloridi; Dopamin Hidroklorür; Dopamine, chlorhydrate de; Dopamin-hidroklorid; Dopamin-hydrochlorid; Dopaminhydroklorid; Dopamini hydrochloridum; Dopamino hidrochloridas; Dopaminy chlorowodorek; Hidrocloruro de dopamina; 3-Hydroxytyramine Hydrochloride. 4-(2-Aminoethyl)pyrocatechol hydrochloride.

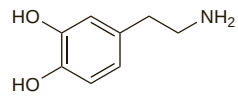
Допамина Гидрохлорид

$C_8H_{11}NO_2$,HCl = 189.6.

CAS — 51-61-6 (dopamine); 62-31-7 (dopamine hydrochloride).

ATC — C01CA04.

ATC Vet — QC01CA04.



(dopamine)

Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), *Int.*, *Jpn*, and *US.*
Ph. Eur. 6.2 (Dopamine Hydrochloride). A white or almost white crystalline powder. Freely soluble in water; soluble in alcohol; sparingly soluble in acetone and in dichloromethane. Store in airtight containers. Protect from light.
USP 31 (Dopamine Hydrochloride). A white to off-white crystalline powder that may have a slight odour of hydrochloric acid. Freely soluble in water and in aqueous solutions of alkali hydroxides; insoluble in chloroform and in ether; soluble in methyl alcohol. pH of a 4% solution in water is between 3.0 and 5.5. Store in airtight containers.

Incompatibility. Dopamine is inactivated in alkaline solutions such as sodium bicarbonate 5% and is incompatible with alkaline drugs such as furosemide[1] and thiopental sodium;[1] incompatibility with insulin[2] and with alteplase[3] has also been reported, and licensed product information states that it is incompatible with ampicillin and with amphotericin B, and that mixtures with gentamicin sulfate, cefalotin sodium, or oxacillin sodium should be avoided.

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. Yamashita SK, *et al.* Compatibility of selected critical care drugs during simulated Y-site administration. *Am J Health-Syst Pharm* 1996; **53:** 1048–51.
3. 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.

Adverse Effects and Treatment

As for Sympathomimetics, p.1407; dopamine may have adverse effects relating to both its alpha- and beta-agonist properties.

Dopamine has a short duration of action and most adverse effects respond to stopping the infusion or reducing its rate; infiltration with phentolamine may relieve pain and prevent necrosis following extravasation.

Effects on the CNS. Movement disorders are well known adverse effects of the dopamine precursor, levodopa (p.804) but do not usually occur with dopamine since it does not enter the CNS. However, there has been a report[1] of choreoathetosis in a patient receiving dopamine infusion; it was suggested that there must have been disruption to her blood-brain barrier to allow this to occur.

1. Walker VA, Massoumi M. Choreoathetosis with dopamine. *Ann Intern Med* 2005; **142:** 478–9.

Effects on the endocrine system. Dopamine has complex actions on the anterior pituitary[1] and dopamine infusion is associated with a number of endocrine effects, including suppression of prolactin, growth hormone, and thyroid hormone release. In postoperative or critically ill patients, dopamine infusion may affect the endocrine response to stress, even when given in low doses. Depression of serum-prolactin concentrations has been reported[2] in critically ill patients given dopamine in a dose of 2.5 micrograms/kg per minute to maintain renal blood flow, while a study[3] in postoperative patients given dopamine 5 micrograms/kg per minute to maintain splanchnic blood flow found that serum concentrations of both prolactin and thyroid stimulating hormone were decreased. It was suggested that these changes could adversely affect immunological function and add to morbidity in such patients.

1. Van den Berghe G, de Zegher F. Anterior pituitary function during critical illness and dopamine treatment. *Crit Care Med* 1996; **24:** 1580–90.
2. Bailey AR, Burchett KR. Effect of low-dose dopamine on serum concentrations of prolactin in critically ill patients. *Br J Anaesth* 1997; **78:** 97–9.
3. Schilling T, *et al.* Endocrine effects of dopexamine vs. dopamine in high-risk surgical patients. *Intensive Care Med* 2001; **27:** 1908–15.

Effects on the heart. For mention of the arrhythmogenic effects of dopamine on the heart, see p.1407.

Ischaemia and gangrene. Dopamine is converted to noradrenaline, a powerful vasoconstrictor, and there have been reports[1-3] of ischaemia and gangrene of the extremities in patients receiving dopamine infusion, as well as local necrosis after extravasation.[4] Extravasation of catecholamines is usually treated with an alpha blocker such as phentolamine, but there have also been reports of the use of topical glyceryl trinitrate ointment to improve capillary blood flow in patients with dopamine-induced ischaemia of the digits. The ointment was applied either to the affected area,[5] or to the warmest area of skin,[6] such as the chest or abdominal wall.

1. Alexander CS, *et al.* Pedal gangrene associated with the use of dopamine. *N Engl J Med* 1975; **293:** 591.
2. Julka NK, Nora JR. Gangrene aggravation after use of dopamine. *JAMA* 1976; **235:** 2812–13.
3. Maggi JC, *et al.* Gangrene in a neonate following dopamine therapy. *J Pediatr* 1982; **100:** 323–5.
4. Boltax RS, *et al.* Gangrene resulting from infiltrated dopamine solution. *N Engl J Med* 1977; **296:** 823.
5. Gibbs NM, Oh TE. Nitroglycerine ointment for dopamine induced peripheral digital ischaemia. *Lancet* 1983; **ii:** 290.
6. Coakley J. Nitroglycerin ointment for dopamine-induced peripheral ischaemia. *Lancet* 1983; **ii:** 633.

Precautions

As for Sympathomimetics, p.1407.

Children. There have been reports of increased pulmonary artery pressure with the use of dopamine in children after cardiac surgery,[1] and in premature infants with hypotension.[2] It has therefore been suggested that dopamine should be used with caution in children at risk of developing pulmonary hypertension.

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. Liet J-M, *et al.* Dopamine effects on pulmonary artery pressure in hypotensive preterm infants with patent ductus arteriosus. *J Pediatr* 2002; **140:** 373–5.

Interactions

As for Sympathomimetics, p.1407. Dopamine has both direct and indirect actions and may therefore interact with MAOIs; the dose of dopamine should be substantially reduced in patients taking MAOIs, and an initial dose of one-tenth the usual dose has been suggested.

Antiepileptics. Following a report in 1976 to the FDA of hypotension in patients given *phenytoin* in addition to dopamine infusion, a study[1] of this potential interaction found that dopamine given by intravenous infusion with phenytoin infusion to *dogs*, did not alter the CNS effects of phenytoin nor result in hypotension and cardiovascular collapse. Large doses of phenytoin alone had a reproducible hypotensive effect that was reduced by dopamine, suggesting a possible supportive role in phenytoin-induced hypotension.

1. Smith RD, Lomas TE. Modification of cardiovascular responses to intravenous phenytoin by dopamine in dogs: evidence against an adverse interaction. *Toxicol Appl Pharmacol* 1978; **45:** 665–73.

Dopaminergics. Severe hypertension occurred[1] in a patient who had been receiving *selegiline* for Parkinson's disease when a dopamine infusion was started. Although selegiline is considered to be a selective monoamine oxidase type B inhibitor, at higher doses it also affects monoamine oxidase type A and could