Lipidil†; ***Philipp.:*** Fibrafen; Lipanthyl; Lipway; Nubrex; Trolip; ***Pol.:*** Apo-Feno; Fenardin; Fenoratio; Grofibrat; Lipanthyl; ***Port.:*** Apteor; Catalip; Lipanthyl; Lipofen; Supralip; ***Rus.:*** Lipanthyl (Липантил); ***S.Afr.:*** Lipsin†; ***Singapore:*** Fenogal Lidose; Lexemin; Lipanthyl; ***Spain:*** Liparison; Secalip; ***Swed.:*** Lipanthyl; ***Switz.:*** Lipanthyl; ***Thai.:*** Fenox; Fibrolan; Lexemin; Lipanthyl; Supralip; ***Turk.:*** Lipanthyl; Lipofen; ***UK:*** Fenogal; Lipantil; Supralip; ***USA:*** Antara; Lipofen; Lofibra; Tricor; Triglide.

Fenoldopam Mesilate *(BANM, rINNM)*

Fénoldopam, Mésilate de; Fenoldopam Mesylate *(USAN)*; Fenoldopami Mesilas; Mesilato de fenoldopam; SKF-82526-j. 6-Chloro-2,3,4,5-tetrahydro-1-(*p*-hydroxyphenyl)-1*H*-3-benzazepine-7,8-diol methanesulfonate.

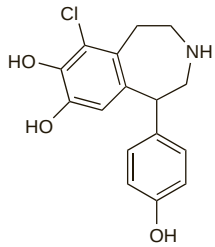
Фенолдопама Мезилат

$C_{16}H_{16}ClNO_3,CH_4O_3S$ = 401.9.

CAS — 67227-56-9 (fenoldopam); 67227-57-0 (fenoldopam mesilate).

ATC — C01CA19.

ATC Vet — QC01CA19.



(fenoldopam)

Pharmacopoeias. In *US.*

USP 31 (Fenoldopam Mesylate). A white to off-white powder. Soluble in water. Store in airtight containers at a temperature of 25°, excursions permitted between 15° and 30°. Protect from moisture.

Incompatibility. Physical incompatibility has been reported[1] with fenoldopam 80 micrograms/mL (as the mesilate) in 0.9% sodium chloride injection and the following drugs during simulated Y-site administration: aminophylline; ampicillin sodium; amphotericin B; bumetanide; cefoxitin sodium; dexamethasone sodium phosphate; diazepam; fosphenytoin sodium; furosemide; ketorolac tromethamine; methohexital sodium; methylprednisolone sodium succinate; pentobarbital sodium; phenytoin sodium; prochlorperazine edisilate; sodium bicarbonate; and thiopental sodium.

1. Trissel LA, *et al.* Compatibility of fenoldopam mesylate with other drugs during simulated Y-site administration. *Am J Health-Syst Pharm* 2003; **60:** 80–5.

Stability. Fenoldopam mesilate, at concentrations ranging from 4 to 300 micrograms/mL in glucose 5% or sodium chloride 0.9%, has been reported[1] to be stable for 72 hours when stored at temperatures of 4° or 23°.

1. Trissel LA, *et al.* Stability of fenoldopam mesylate in two infusion solutions. *Am J Health-Syst Pharm* 2002; **59:** 846–8.

Adverse Effects and Precautions

The adverse effects of fenoldopam are mainly due to vasodilatation and include hypotension, flushing, dizziness, headache, and reflex tachycardia. Nausea and vomiting, and ECG abnormalities have also been reported. Hypokalaemia has occurred and serum-electrolyte concentrations should be monitored during therapy; blood pressure and heart rate should also be monitored. Fenoldopam may increase intra-ocular pressure and it should be used with caution in patients with glaucoma. Caution is also required in patients in whom hypotension could be deleterious, such as those with acute cerebral infarction or haemorrhage.

Effects on the heart. Although fenoldopam is usually associated with reflex tachycardia, precipitous bradycardia in 2 patients given fenoldopam infusion in a clinical study[1] forced the drug to be stopped.

1. Taylor AA, *et al.* Sustained hemodynamic effects of the selective dopamine-1 agonist, fenoldopam, during 48-hour infusions in hypertensive patients: a dose-tolerability study. *J Clin Pharmacol* 1999; **39:** 471–9.

Interactions

The hypotensive effects of fenoldopam may be enhanced by other drugs with hypotensive actions. Beta blockers may block fenoldopam-induced reflex tachycardia and use of the drugs together is not recommended.

Pharmacokinetics

Steady-state plasma concentrations of fenoldopam are reached about 20 minutes after starting continuous intravenous infusion. Fenoldopam is extensively metabolised with only about 4% of a dose being excreted unchanged. It is metabolised by conjugation (mainly glucuronidation, methylation, and sulfation). Fenoldopam and its metabolites are excreted mainly in the urine, and the remainder in the faeces. The elimination half-life of fenoldopam is about 5 minutes.

Uses and Administration

Fenoldopam is a dopamine agonist that is reported to have a selective action at dopamine D_1-receptors, leading to vasodilatation. It is used in the short-term management of severe hypertension (below) and has also been tried in heart failure.

Fenoldopam is given intravenously as the mesilate, although doses are expressed in terms of the base; 1.31 micrograms of fenoldopam mesilate is equivalent to about 1 microgram of fenoldopam.

In the management of hypertensive crises, fenoldopam mesilate is given by continuous intravenous infusion for up to 48 hours, as a solution containing 40 micrograms/mL of fenoldopam. The dose should be adjusted according to response, in usual increments of 50 to 100 nanograms/kg per minute at not less than 15-minute intervals. The usual dose range is from 100 to 1600 nanograms/kg per minute.

In the management of hypertensive crises in children, fenoldopam mesilate is given by continuous intravenous infusion for up to 4 hours, as a solution containing 60 micrograms/mL of fenoldopam. US licensed product information states that the initial dose used in clinical studies was 200 nanograms/kg per minute; adjustments according to response every 20 to 30 minutes up to 500 nanograms/kg per minute were usually well-tolerated. No benefit was seen from doses above 800 nanograms/kg per minute.

Hypertension. Fenoldopam has a rapid onset of action and short elimination half-life and may be used as an alternative to sodium nitroprusside in the management of hypertensive crises (see under Hypertension, p.1171). Its use has been reviewed.[1-3] Comparative studies with sodium nitroprusside in patients with acute severe hypertension have shown fenoldopam to be equally effective in rapidly lowering blood pressure. Additionally, in contrast to nitroprusside, urine output, creatinine clearance, and sodium excretion may be increased by fenoldopam. Fenoldopam may therefore be particularly useful in patients with renal impairment, although this remains to be established.

1. Brogden RN, Markham A. Fenoldopam: a review of its pharmacodynamic and pharmacokinetic properties and intravenous clinical potential in the management of hypertensive urgencies and emergencies. *Drugs* 1997; **54:** 634–50.
2. Post JB, Frishman WH. Fenoldopam: a new dopamine agonist for the treatment of hypertensive urgencies and emergencies. *J Clin Pharmacol* 1998; **38:** 2–13.
3. Murphy MB, *et al.* Fenoldopam: a selective peripheral dopamine-receptor agonist for the treatment of severe hypertension. *N Engl J Med* 2001; **345:** 1548–57.

Nephrotoxicity. Fenoldopam increases renal blood flow and has been tried to reduce the renal toxicity that may be associated with use of contrast media (see Effects on the Kidneys under Adverse Effects of Amidotrizoic Acid, p.1476). Small studies in patients at risk of renal toxicity have shown benefit with fenoldopam,[1,2] but larger randomised trials[3,4] have found no advantage with fenoldopam plus hydration compared with hydration using sodium chloride 0.45% alone. However, a later meta-analysis[5] in patients undergoing cardiovascular surgery, who are at risk of acute renal failure, found that fenoldopam consistently reduced the need for renal replacement therapy, and reduced mortality.

A study[6] in patients undergoing liver transplantation (p.1815) suggested that fenoldopam may have a role in preserving renal function, possibly by counteracting the renal toxicity associated with ciclosporin.

1. Chu VL, Cheng JWM. Fenoldopam in the prevention of contrast media-induced acute renal failure. *Ann Pharmacother* 2001; **35:** 1278–82. Correction. *ibid.*; 1677.
2. Lepor NE. A review of contemporary prevention strategies for radiocontrast nephropathy: a focus on fenoldopam and N-acetylcysteine. *Rev Cardiovasc Med* 2003; **4** (suppl 1): S15–S20.
3. Allaqaband S, *et al.* Prospective randomized study of N-acetylcysteine, fenoldopam, and saline for prevention of radiocontrast-induced nephropathy. *Catheter Cardiovasc Interv* 2002; **57:** 279–83.
4. Stone GW, *et al.* Fenoldopam mesylate for the prevention of contrast-induced nephropathy: a randomized controlled trial. *JAMA* 2003; **290:** 2284–91.
5. Landoni G, *et al.* Fenoldopam reduces the need for renal replacement therapy and in-hospital death in cardiovascular surgery: a meta-analysis. *J Cardiothorac Vasc Anesth* 2008; **22:** 27–33.
6. Biancofiore G, *et al.* Use of fenoldopam to control renal dysfunction early after liver transplantation. *Liver Transpl* 2004; **10:** 986–92.

Preparations

USP 31: Fenoldopam Mesylate Injection.

Proprietary Preparations (details are given in Part 3)
Irl.: Corlopam†; ***Ital.:*** Corlopam; ***Neth.:*** Corlopam; ***USA:*** Corlopam.

Fenquizone *(USAN, rINN)* ⊗

Fenquizona; Fenquizonum; MG-13054. 7-Chloro-1,2,3,4-tetrahydro-4-oxo-2-phenylquinazoline-6-sulphonamide.

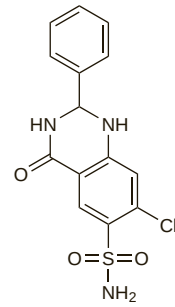
Фенхизон

$C_{14}H_{12}ClN_3O_3S$ = 337.8.

CAS — 20287-37-0.

ATC — C03BA13.

ATC Vet — QC03BA13.



Fenquizone Potassium *(rINNM)* ⊗

Fenquizona potásica; Fenquizone Potassique; Kalii Fenquizonum.

Калия Фенхизон

$C_{14}H_{12}ClN_3O_3S,K$ = 376.9.

CAS — 52246-40-9.

ATC — C03BA13.

ATC Vet — QC03BA13.

Profile

Fenquizone potassium is a diuretic that is given orally in the treatment of oedema and hypertension (p.1171) in doses equivalent to 10 to 20 mg of fenquizone daily. 11.2 mg of the potassium salt is equivalent to about 10 mg of the base.

◊ References.

1. Beermann B, Grind M. Clinical pharmacokinetics of some newer diuretics. *Clin Pharmacokinet* 1987; **13:** 254–66.
2. Costa FV, *et al.* Hemodynamic and humoral effects of chronic antihypertensive treatment with fenquizone: importance of aldosterone response. *J Clin Pharmacol* 1990; **30:** 254–61.

Preparations

Proprietary Preparations (details are given in Part 3)
Ital.: Idrolone.

Fibrinolysin

Fibrinolysin (Human) *(BAN, rINN)*; Fibrinase; Fibrinolisina (humana); Fibrinolysine (humaine); Fibrinolysinum (humanum); Plasmiini; Plasmin; Plasminum.

Фибринолизин (Человека)

CAS — 9001-90-5 (fibrinolysin); 9004-09-5 (human fibrinolysin).

ATC — B01AD05.

ATC Vet — QB01AD05.

NOTE. In *Martindale* the term fibrinolysin is used for the exogenous substance and plasmin for the endogenous substance.

Profile

Fibrinolysin is a proteolytic enzyme derived from the activation of human plasminogen. Fibrinolysin derived from cattle (bovine fibrinolysin) and other animals is also available. Fibrinolysin converts fibrin into soluble products and also hydrolyses some other proteins. The role of plasmin (endogenous fibrinolysin) in the control of haemostasis is described further on p.1045.

Fibrinolysin is used (generally as bovine fibrinolysin) with deoxyribonuclease for the debridement of wounds. It was formerly given parenterally for the treatment of thrombotic disorders. A modified form of fibrinolysin, microplasmin, is under investigation for use in ophthalmic surgery.

Preparations

Proprietary Preparations (details are given in Part 3)

Multi-ingredient: ***Arg.:*** Clorfibrase; ***Austria:*** Fibrolan; ***Braz.:*** Cauterex; Dermofibrin C†; Fibrabene; Fibrase; Fibrinase c/Cloranfenicol; Gino-Cauterex; Gino-Fibrase; Procutan†; ***Chile:*** Elase; ***Cz.:*** Fibrolan; ***Fr.:*** Elase; ***Ger.:*** Fibrolan†; ***Hung.:*** Fibrolan; ***Ital.:*** Elase†; ***Malaysia:*** Elase; ***Mex.:*** Fibrase; Fibrase SA; ***Pol.:*** Fibrolan; ***Switz.:*** Fibrolan.

Flecainide Acetate *(BANM, USAN, rINNM)*

Acetato de flecainida; Flécaïnide, acétate de; Flecainidi acetas; Flekainidacetat; Flekainid-acetát; Flekainidiasetaatti; Flekainido acetatas; R-818. *N*-(2-Piperidylmethyl)-2,5-bis(2,2,2-trifluoroethoxy)benzamide acetate.

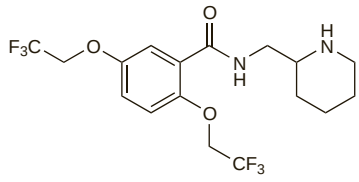
Флекаинида Ацетат

$C_{17}H_{20}F_6N_2O_3,C_2H_4O_2$ = 474.4.

CAS — 54143-55-4 (flecainide); 54143-56-5 (flecainide acetate).

ATC — C01BC04.

ATC Vet — QC01BC04.



(flecainide)

Pharmacopoeias. In *Eur.* (see p.vii) and *US*.

Ph. Eur. 6.2 (Flecainide Acetate). A white or almost white, very hygroscopic crystalline powder. Soluble in water and in dehydrated alcohol; freely soluble in dilute acetic acid; practically insoluble in dilute hydrochloric acid. A 2.5% solution in water has a pH of 6.7 to 7.1. Protect from light.

USP 31 (Flecainide Acetate). A white to slightly off-white crystalline powder; pK_a is 9.3. Soluble in water; freely soluble in alcohol.

Stability. Storage of an extemporaneously prepared flecainide syrup in a refrigerator led to crystallisation of the drug and a toxic dose being given.[1] It was suggested that oral liquid formulations of flecainide should be freshly reconstituted from a powder before each dose. However, other extemporaneous formulations have been reported[2,3] to be stable at room temperature and under refrigeration.

1. Stuart AG, *et al.* Is there a genetic factor in flecainide toxicity? *BMJ* 1989; **298:** 117–18.
2. Wiest DB, *et al.* Stability of flecainide acetate in an extemporaneously compounded oral suspension. *Am J Hosp Pharm* 1992; **49:** 1467–70.
3. Allen LV, Erickson MA. Stability of baclofen, captopril, diltiazem hydrochloride, dipyridamole, and flecainide acetate in extemporaneously compounded oral liquids. *Am J Health-Syst Pharm* 1996; **53:** 2179–84.

Adverse Effects

The most common adverse effects caused by flecainide affect the CNS and include dizziness, visual disturbances, and lightheadedness. Nausea, vomiting, headache, tremor, peripheral neuropathy, ataxia, and paraesthesia may also occur. These effects are generally transient and respond to dosage reduction. Other adverse CNS effects that have been reported rarely include hallucinations, amnesia, confusion, depression, dyskinesias, and convulsions. Skin reactions, including rare cases of urticaria, have also occurred and there have been isolated cases of photosensitivity. Disturbances of liver function have been reported rarely. Corneal deposits, pulmonary fibrosis, and pneumonitis have occurred during long-term therapy. Cardiovascular effects are less common than those on the CNS, but can be serious and sometimes fatal. Ventricular tachyarrhythmias have been reported, particularly in patients with a history of ventricular tachyarrhythmias and taking high doses of flecainide. Chest pain and myocardial infarction have also occurred. Flecainide produced an increased mortality rate when it was assessed for the control of asymptomatic ventricular arrhythmias in patients who had previously suffered a myocardial infarction (see Cardiac Arrhythmias under Uses and Administration, below).

Incidence of adverse effects. In a report of the non-cardiac adverse effects of flecainide from 1 short-term and 3 longer-term studies,[1] the most common adverse effects during both short- and long-term studies were dizziness and visual disturbances, which occurred in about 30% of patients. Headache and nausea both occurred in about 10% of patients. Other adverse effects reported include dyspnoea, chest pain, asthenia, fatigue, and tremor. Therapy was stopped because of non-cardiac adverse effects in 10% of patients in the short-term trial, and in 6% of those in the chronic studies. A review of 60 studies using flecainide[2] reported that non-cardiac adverse effects (mainly gastrointestinal and CNS adverse effects) occurred in 12% of patients. The UK CSM stated in June 1991 that it had received reports of neurological (4 patients with sensory neuropathy, 2 with ataxia), corneal (2 with corneal deposits), and pulmonary (3 with pulmonary fibrosis and pneumonitis) reactions associated with the long-term use of flecainide.[3]

1. Gentzkow GD, Sullivan JY. Extracardiac adverse effects of flecainide. *Am J Cardiol* 1984; **53:** 101B–105B.
2. Hohnloser SH, Zabel M. Short- and long-term efficacy and safety of flecainide acetate for supraventricular arrhythmias. *Am J Cardiol* 1992; **70:** 3A–10A.
3. Committee on Safety of Medicines. Multi-system adverse reactions following long-term flecainide therapy. *Current Problems 31* 1991. Also available at: http://www.mhra.gov.uk/home/idcplg?IdcService=GET_FILE&dDocName=CON2024449&RevisionSelectionMethod=LatestReleased (accessed 08/05/07)

Effects on the blood. Severe granulocytopenia believed to be related to flecainide occurred in a 66-year-old man 3 months after starting therapy.[1] Haematological findings suggested an immune-mediated reaction in which flecainide binds to normal neutrophils with subsequent recognition by specific antibodies resulting in enhanced destruction of mature granulocytes in peripheral blood and bone marrow.

1. Samlowski WE, *et al.* Flecainide-induced immune neutropenia: documentation of a hapten-mediated mechanism of cell destruction. *Arch Intern Med* 1987; **147:** 383–4.

Effects on the eyes. In addition to visual disturbance, symptomatic corneal deposits have been reported[1] in patients taking flecainide. A study[2] in 38 patients found small corneal deposits in 14.5%, but visual function tests were normal.

1. Ulrik H, *et al.* Corneal deposits associated with flecainide. *BMJ* 1991; **302:** 506–7.
2. Ikäheimo K, *et al.* Adverse ocular effects of flecainide. *Acta Ophthalmol Scand* 2001; **79:** 175–6.

Effects on the heart. Like most antiarrhythmics, flecainide can have proarrhythmic effects,[1] and severe ventricular arrhythmias have been reported,[2] including fatal ventricular fibrillation[3] in a neonate given flecainide for supraventricular tachycardia. There has also been a report[4] of torsade de pointes, although this is generally less common with class Ic than with class Ia antiarrhythmics. For reports of increased cardiac mortality in patients given flecainide for asymptomatic arrhythmias, see Cardiac Arrhythmias under Uses and Administration, below.

1. Herre JM, *et al.* Inefficacy and proarrhythmic effects of flecainide and encainide for sustained ventricular tachycardia and ventricular fibrillation. *Ann Intern Med* 1990; **113:** 671–6.
2. Falk RH. Flecainide-induced ventricular tachycardia and fibrillation in patients treated for atrial fibrillation. *Ann Intern Med* 1989; **111:** 107–11.
3. Ackland F, *et al.* Flecainide induced ventricular fibrillation in a neonate. *Heart* 2003; **89:** 1261.
4. Nogales Asensio JM, *et al.* Torsade-de-pointes in a patient under flecainide treatment: an unusual case of proarrhythmicity. *Int J Cardiol* 2007; **114:** e65–e67.

Effects on the liver. Elevated liver enzymes and jaundice, reversible on stopping treatment, have been reported rarely with flecainide.

Conjugated hyperbilirubinaemia with jaundice developed in a newborn infant after maternal treatment with flecainide for fetal supraventricular tachycardia.[1]

1. Vanderhal AL, *et al.* Conjugated hyperbilirubinemia in a newborn infant after maternal (transplacental) treatment with flecainide acetate for fetal tachycardia and fetal hydrops. *J Pediatr* 1995; **126:** 988–90.

Effects on the lungs. There have been reports[1-4] of interstitial pneumonitis associated with flecainide. See also under Incidence of Adverse Effects, above.

1. Akoun GM, *et al.* Flecainide-associated pneumonitis. *Lancet* 1991; **337:** 49.
2. Hanston P, *et al.* Flecainide-associated interstitial pneumonitis. *Lancet* 1991; **337:** 371–2.
3. Robain A, *et al.* Flecainide-associated pneumonitis with acute respiratory failure in a patient with the LEOPARD syndrome. *Acta Cardiol* 2000; **55:** 45–7.
4. Pesenti S, *et al.* Diffuse infiltrative lung disease associated with flecainide: report of two cases. *Respiration* 2002; **69:** 182–5.

Effects on mental state. Dysarthria and visual hallucinations were associated with elevated plasma concentration of flecainide (2500 nanograms/mL) in a patient.[1] A serial rise and fall in plasma-bilirubin concentration during flecainide therapy also suggested possible direct hepatotoxicity. There has also been a report[2] of paranoid psychosis in a patient receiving flecainide for neuropathic pain.

1. Ramhamadany E, *et al.* Dysarthria and visual hallucinations due to flecainide toxicity. *Postgrad Med J* 1986; **62:** 61–2.
2. Bennett MI. Paranoid psychosis due to flecainide toxicity in malignant neuropathic pain. *Pain* 1997; **70:** 93–4.

Effects on the nervous system. Peripheral neuropathy, reversible on stopping treatment, has been reported[1,2] in patients receiving flecainide long-term. The authors of one report[1] stated that, at the time (1992), the UK CSM had received 4 other reports possibly associated with flecainide and 3 reports of aggravation of pre-existing neuropathy;[1] not all cases were reversible.

1. Palace J, *et al.* Flecainide induced peripheral neuropathy. *BMJ* 1992; **305:** 810.
2. Malesker MA, *et al.* Flecainide-induced neuropathy. *Ann Pharmacother* 2005; **39:** 1580.

Lupus erythematosus. There has been a report[1] of a patient who developed painful eye movement during flecainide therapy. The pain resolved on withdrawal but recurred when flecainide was restarted, and was accompanied by lateral rectus spasm, a facial rash, and positive antinuclear factor, suggestive of lupus erythematosus.

1. Skander M, Isaacs PET. Flecainide, ocular myopathy, and antinuclear factor. *BMJ* 1985; **291:** 450.

Treatment of Adverse Effects

In oral overdosage with flecainide activated charcoal may be considered if the patient presents within 1 hour of ingestion. Treatment is largely symptomatic and supportive and may need to be continued for extended periods of time because of the long half-life and the possibility of non-linear elimination at very high doses. Haemodialysis or haemoperfusion are unlikely to enhance elimination.

Overdosage. Severe cardiovascular toxicity in flecainide overdosage may be resistant to pacing and inotropes, and hypoperfusion of the kidneys and liver may reduce the elimination of flecainide, prolonging the toxic effects. Gastric decontamination is of uncertain benefit; forced diuresis has been used,[1] but probably had a negligible effect, and haemodialysis and haemoperfusion are not effective.[2] Patients may therefore require intensive and prolonged supportive treatment,[1,3] and there have been reports of the use of extracorporeal membrane oxygenation,[4] cardiopulmonary bypass,[5,6] or intra-aortic balloon pumping,[7] to maintain organ perfusion and allow flecainide elimination to occur, with complete recovery in some cases.[3,4,6,7] There have been reports[8,9] of the successful use of intravenous hypertonic sodium bicarbonate, and it has been suggested that it may antagonise the sodium channel blockade produced by flecainide, as well as reversing the metabolic acidosis that commonly occurs.

1. Winkelmann BR, Leinberger H. Life-threatening flecainide toxicity: a pharmacodynamic approach. *Ann Intern Med* 1987; **106:** 807–14.
2. Braun J, *et al.* Failure of haemoperfusion to reduce flecainide intoxication: a case study. *Med Toxicol* 1987; **2:** 463–7.
3. Hanley NA, *et al.* Survival in a case of life-threatening flecainide overdose. *Intensive Care Med* 1998; **24:** 740–2.
4. Auzinger GM, Scheinkestel CD. Successful extracorporeal life support in a case of severe flecainide intoxication. *Crit Care Med* 2001; **29:** 887–90.
5. Yasui RK, *et al.* Flecainide overdose: is cardiopulmonary support the treatment? *Ann Emerg Med* 1997; **29:** 680–2.
6. Corkeron MA, *et al.* Extracorporeal circulatory support in near-fatal flecainide overdose. *Anaesth Intensive Care* 1999; **27:** 405–8.
7. Timperley J, *et al.* Flecainide overdose—support using an intra-aortic balloon pump. *BMC Emerg Med* 2005; **5:** 10.
8. Goldman MJ, *et al.* Sodium bicarbonate to correct widened QRS in a case of flecainide overdose. *J Emerg Med* 1997; **15:** 183–6.
9. Lovecchio F, *et al.* Hypertonic sodium bicarbonate in an acute flecainide overdose. *Am J Emerg Med* 1998; **16:** 534–7.

Precautions

Flecainide treatment should be started in hospital or under specialist supervision and pacing rescue should be available when it is used in patients with conduction defects. Its use is limited to serious or life-threatening arrhythmias and it should not be given to control asymptomatic arrhythmias especially in patients with a history of myocardial infarction (see Cardiac Arrhythmias under Uses and Administration, below). Flecainide has some negative inotropic activity and may precipitate or aggravate heart failure in patients with compromised left ventricular function; it should therefore be used with extreme caution, if at all, in patients with heart failure. Flecainide has been shown to increase the endocardial pacing threshold and should be used with caution in patients with pacemakers. Electrolyte imbalances should be corrected before starting flecainide therapy. Reduction of dosage may be necessary in patients with renal impairment; extreme caution is needed in patients with pronounced hepatic impairment.

Breast feeding. Flecainide is distributed into breast milk but there have been no reports of infant exposure. Flecainide 100 mg was given orally every 12 hours to 11 healthy women, beginning 1 day after delivery and continuing for 5 days.[1] The mean elim-