Felodipine is given orally, generally in a modified-release formulation for use once daily in the morning. In **hypertension** the usual initial dose is 5 mg daily, adjusted as required; the usual maintenance dose is 2.5 to 10 mg daily and doses above 20 mg daily are not usually needed. In **angina** the usual initial dose is 5 mg daily increased if necessary to 10 mg daily.

Lower doses may be required in patients with hepatic impairment (see below) and in the elderly.

◊ Reviews.

1. Todd PA, Faulds D. Felodipine: a review of the pharmacology and therapeutic use of the extended release formulation in cardiovascular disorders. *Drugs* 1992; **44:** 251–77.
2. Walton T, Symes LR. Felodipine and isradipine: new calcium-channel-blocking agents for the treatment of hypertension. *Clin Pharm* 1993; **12:** 261–75.

Administration in hepatic impairment. In 9 patients with liver cirrhosis given felodipine 750 micrograms by intravenous infusion over 20 minutes and 10 mg orally as single doses on separate occasions the mean oral bioavailability was 17.1% which was not significantly different from published values in healthy subjects, but the maximum plasma concentrations were almost twice as high as normal, apparently due to reduced systemic clearance and volume of distribution.[1] The fact that bioavailability was not increased suggests that much pre-systemic metabolism takes place in the gut rather than the liver. Although increased adverse effects were not associated with the raised felodipine concentrations in this study it is recommended that therapy in cirrhotic patients begin at lower doses than in patients with normal liver function. US licensed product information recommends that an initial dose of 2.5 mg once daily should be used in patients with hepatic impairment.

1. Regårdh CG, *et al.* Pharmacokinetics of felodipine in patients with liver disease. *Eur J Clin Pharmacol* 1989; **36:** 473–9.

Preparations

USP 31: Felodipine Extended-Release Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Munobal; Plendil; ***Austral.:*** Agon†; Felodur; Plendil; ***Austria:*** Felodistad; Munobal; Plendil; ***Belg.:*** Plendil; Renedil; ***Braz.:*** Splendil; ***Canad.:*** Plendil; Renedil; ***Chile:*** Splendil; ***Cz.:*** Auronal; Felocor; Plendil; Presid; ***Denm.:*** Felodin; Hydac†; Plendil; Plendur; ***Fin.:*** Hydac; Plendil; ***Fr.:*** Flodil; ***Ger.:*** Felo-Puren; Felobeta; Felocor; Felogamma; Modip; Munobal; ***Gr.:*** Plendil; ***Hong Kong:*** Plendil; ***Hung.:*** Plendil; Presid; ***India:*** Felogard; ***Indon.:*** Nirmadil; Plendil; ***Irl.:*** Plendil; ***Israel:*** Penedil; ***Ital.:*** Feloday; Plendil; Prevex; ***Jpn:*** Splendil†; ***Malaysia:*** Plendil; ***Mex.:*** Fedin; Munobal; Plendil; ***Neth.:*** Plendil; Renedil; ***Norw.:*** Plendil; ***NZ:*** Felo; Plendil; ***Philipp.:*** Dilahex; Felim; Felop; Lodistad; Plendil; Versant; ***Pol.:*** Felohexal; Plendil; ***Port.:*** Mencor; Preslow; ***Rus.:*** Felodip (Фелодип); Plendil (Пленди́л); ***S.Afr.:*** Plendil; ***Singapore:*** Plendil; ***Spain:*** Fensel; Perfudal; Plendil; ***Swed.:*** Plendil; ***Switz.:*** Felodil; Munobal†; Plendil; ***Thai.:*** Felim; Felohexal; Feloten; Plendil; ***Turk.:*** Plendil; ***UK:*** Cardioplen; Felotens; Keloc; Neofel; Plendil; Vascalpha; ***USA:*** Plendil; ***Venez.:*** Munobal†; Plendil.

Multi-ingredient: ***Arg.:*** Nikion†; Triacor; ***Austral.:*** Triasyn; ***Austria:*** Triapin; Unimax; ***Belg.:*** Logimat; ***Cz.:*** Logimax†; Triasyn; Unimax†; ***Denm.:*** Logimax; ***Fin.:*** Logimax; Unimax; ***Fr.:*** Logimax; ***Ger.:*** Delmuno; Mobloc; Unimax; ***Gr.:*** Logimax; Triacor; Unitens; ***Hong Kong:*** Logimax; ***Hung.:*** Logimax; Triasyn; ***Irl.:*** Triapin; ***Israel:*** Logimax; ***Mex.:*** Logimax; Triacor; ***Neth.:*** Logimax; Triapin; Unimax; ***Philipp.:*** Logimax; Triapin; ***Port.:*** Unimax; ***Rus.:*** Logimax (Логимакс); ***S.Afr.:*** Tri-Plen; ***Spain:*** Logimax; ***Swed.:*** Logimax; ***Switz.:*** Logimax; Unimax; ***UK:*** Triapin; ***USA:*** Lexxel.

Fendiline Hydrochloride *(pINNM)*

Fendiline, Chlorhydrate de; Fendilini Hydrochloridum; Hidrocloruro de fendilina. *N*-(2-Benzhydrylethyl)-α-methylbenzylamine hydrochloride.

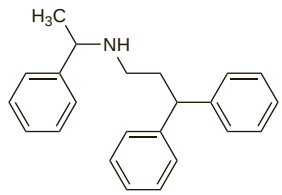
Фендилина Гидрохлорид

$C_{23}H_{25}N,HCl$ = 351.9.

CAS — 13042-18-7 (fendiline); 13636-18-5 (fendiline hydrochloride).

ATC — C08EA01.

ATC Vet — QC08EA01.



(fendiline)

Profile

Fendiline hydrochloride is a calcium-channel blocker used as a vasodilator in ischaemic heart disease.

Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Sensit†; ***Ger.:*** Sensit; ***Gr.:*** Sensit.

Fenofibrate *(BAN, rINN)*

Fenofibraatti; Fenofibrát; Fenofibrat; Fenofibratas; Fénofibrate; Fenofibrato; Fenofibratum; LF-178; Procetofen; Procetofene. Isopropyl 2-[4-(4-chlorobenzoyl)phenoxy]-2-methylpropionate.

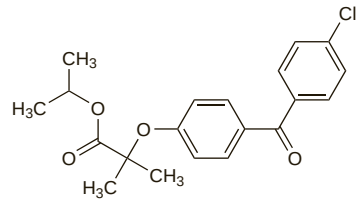
Фенофибрат

$C_{20}H_{21}ClO_4$ = 360.8.

CAS — 49562-28-9.

ATC — C10AB05.

ATC Vet — QC10AB05.



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

Ph. Eur. 6.2 (Fenofibrate). A white or almost white, crystalline powder. M.p. 79° to 82°. Practically insoluble in water; slightly soluble in alcohol; very soluble in dichloromethane. Protect from light.

USP 31 (Fenofibrate). A white or almost white, crystalline powder. M.p. 79° to 82°. Practically insoluble in water; slightly soluble in alcohol; very soluble in dichloromethane. Protect from light.

Adverse Effects and Precautions

As for Bezafibrate, p.1232.

Interactions

As for Bezafibrate, p.1232.

UK licensed product information for fenofibrate suggests that in patients taking oral anticoagulants, the dose of anticoagulant should be reduced by about one-third when treatment with fenofibrate is started, and then adjusted gradually if necessary.

Pharmacokinetics

Fenofibrate is readily absorbed from the gastrointestinal tract when taken with food; absorption may be reduced if fenofibrate is given on an empty stomach, although this depends on the formulation (see Bioavailability, below). It is rapidly hydrolysed to its active metabolite fenofibric acid which is about 99% bound to plasma albumin. The plasma elimination half-life is about 20 hours. Fenofibric acid is excreted mainly in the urine, mainly as the glucuronide conjugate, but also as a reduced form of fenofibric acid and its glucuronide. It is not removed by haemodialysis.

◊ References.

1. Chapman MJ. Pharmacology of fenofibrate. *Am J Med* 1987; **83** (suppl 5B): 21–5.

Bioavailability. Fenofibrate is poorly soluble in water and has a low bioavailability when given orally.[1] Bioavailability is increased by food, particularly if there is a high fat content, and fenofibrate is therefore usually given with meals. Changes to the formulation, particularly with regard to the particle size, have been made to improve solubility,[1] with the aim of increasing bioavailability and reducing the influence of food. Micronisation improves bioavailability to a certain extent, and allows a lower dose to be given; 300 mg of non-micronised fenofibrate is usually considered equivalent to about 200 mg of the standard micronised form. Microcoating may further improve bioavailability,[2] but absorption is still affected by the presence of food.[3] Nanoparticle,[4] stabilised microparticle,[3] or semi-solid formulations,[5] however, appear to have a more consistent bioavailability and may be given with or without food.

1. Vogt M, *et al.* Dissolution enhancement of fenofibrate by micronization, cogrinding and spray-drying: comparison with commercial preparations. *Eur J Pharm Biopharm* 2008; **68:** 283–8.
2. Guichard JP, *et al.* A new formulation of fenofibrate: suprabioavailable tablets. *Curr Med Res Opin* 2000; **16:** 134–8.
3. Guivarc'h PH, *et al.* A new fenofibrate formulation: results of six single-dose, clinical studies of bioavailability under fed and fasting conditions. *Clin Ther* 2004; **26:** 1456–69.
4. Sauron R, *et al.* Absence of a food effect with a 145 mg nanoparticle fenofibrate tablet formulation. *Int J Clin Pharmacol Ther* 2006; **44:** 64–70.
5. Sonet B, *et al.* Randomised crossover studies of the bioequivalence of two fenofibrate formulations after administration of a single oral dose in healthy volunteers. *Arzneimittelforschung* 2002; **52:** 200–4.

Uses and Administration

Fenofibrate, a fibric acid derivative, is a lipid regulating drug with actions on plasma lipids similar to those of bezafibrate (p.1233).

It is used to reduce low-density lipoprotein (LDL)-cholesterol, total cholesterol, triglycerides, and apolipoprotein B, and to increase high-density lipoprotein (HDL)-cholesterol, in the management of hyperlipidaemias (p.1169), including type IIa, type IIb, type III, type IV, and type V hyperlipoproteinaemias.

Fenofibrate is given orally. It is usually given with food to improve bioavailability although this may not be necessary with all preparations (see Bioavailability, above). It is available in a range of formulations with differing bioavailabilities and the dose is therefore specific to the preparation.

Standard micronised formulations of fenofibrate are available as 67-mg capsules to be taken several times daily, or as 200- or 267-mg capsules for once daily dosage. The usual initial dose is 67 mg three times daily or 200 mg once daily; the dose may be reduced to 67 mg twice daily or increased to 67 mg four times daily or 267 mg once daily according to response.

Preparations with improved bioavailability may be given in doses of around 40 to 160 mg once daily.

Non-micronised formulations may also be available and are given in an initial dose of 200 to 300 mg daily in divided doses, adjusted according to response to between 200 and 400 mg daily; 100 mg of non-micronised fenofibrate is therapeutically equivalent to 67 mg of the standard micronised form.

The dose of fenofibrate should be reduced in renal impairment (see below). For the dose of fenofibrate in children, see also below.

◊ Reviews.

1. Keating GM, Croom KF. Fenofibrate: a review of its use in primary dyslipidaemia, the metabolic syndrome and type 2 diabetes mellitus. *Drugs* 2007; **67:** 121–53.

Administration in children. Experience with fenofibrate in children is limited and it should only be given under specialist advice. The dose depends on the formulation:

- For *standard micronised* fenofibrate the *BNFC* recommends that children are given the 67-mg capsule formulation. The oral dose is one 67-mg capsule per 20 kg body-weight daily for children aged 4 to 15 years; those aged 15 to 18 years may be given the adult dose (see above).
- *Non-micronised* fenofibrate is licensed for use in some countries in children from the age of 10 years, in an oral dose of 5 mg/kg daily.

Administration in renal impairment. A single-dose study[1] in patients with mild (creatinine clearance (CC) 30 to 50 mL/minute) or severe renal impairment (CC below 10 mL/minute or undergoing haemodialysis) found that the plasma elimination half-life of fenofibric acid was prolonged, with a range of 54 to 362 hours; no correlation was found between half-life and serum creatinine or CC. Fenofibrate metabolites were not removed by haemodialysis, and repeated dosing in patients undergoing regular haemodialysis led to significant accumulation of fenofibric acid.[1]

Fenofibrate is therefore not generally recommended in patients with severe renal impairment although UK licensed product information allows a dose of 134 mg of standard micronised fenofibrate daily for patients with CC between 20 and 60 mL/minute and 67 mg daily for patients with CC below 20 mL/minute. US licensed product information for improved bioavailability formulations suggests initial daily doses of about 40 to 50 mg (equivalent to about 67 mg of standard micronised fenofibrate) in patients with renal impairment, but contra-indicates use in those with severe impairment.

1. Desager JP, *et al.* Effect of hemodialysis on plasma kinetics of fenofibrate in chronic renal failure. *Nephron* 1982; **31:** 51–4.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Craveril; Fenobrate; Fenolip†; Lipidil†; Lipoplasmin†; Minuslip; Procetoken; Qualecon†; Sclerofin; ***Austral.:*** Lipidil; ***Austria:*** Fenolip; Lipcor; Lipsin; ***Belg.:*** Docfenofi; Fenofitop; Fenogal; Lipanthyl; ***Braz.:*** Lipanon; Lipidil; ***Canad.:*** Apo-Feno; Lipidil; ***Chile:*** Lipidil; ***Cz.:*** Apo-Feno; Febira; Fenofix; Grofibrat†; Hypolip†; Lipanthyl; Lipirex; Lipohexal; Suprelip; ***Fin.:*** Lipanthyl; ***Fr.:*** Fegenor; Lipanthyl; Lipirex; Secalip; ***Ger.:*** ClL; durafenat; Fenobeta; Fenofanton; Lipanthyl; Lipidil; Normalip pro; ***Gr.:*** Lipanthyl; Lipidil; Neo-Disterin; Planitrix†; Zerlubron†; ***Hong Kong:*** Apo-Feno-Micro; Fegenor; Lexemin; Lipanthyl; Qualipantyl; Trolip†; ***Hung.:*** Feno-Micro; Fenobrat; Lipanthyl†; Lipidil; ***India:*** Fenolip; Lipicard; ***Indon.:*** Evothyl; Felosma; Hyperchol; Lipanthyl; Trichol; Trolip; Yosenob; Zumafib; ***Irl.:*** Lipantil; ***Ital.:*** Fulcro; Lipanthyl†; Lipofene; Lipsin; Nolipax; Scleril; Tilene; Volutine†; ***Jpn:*** Tricor; ***Malaysia:*** Apo-Feno-Micro; Lexemin; Lipanthyl; ***Mex.:*** Controlip;

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.

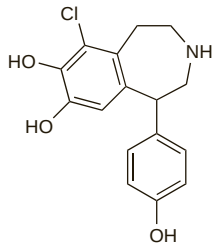
Фенолдопама Мезилат

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

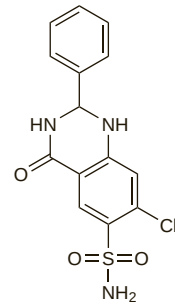
Фенхизон

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