Moxisylyte is given as the hydrochloride but the dose may be expressed in terms of the base. Moxisylyte hydrochloride 45.2 mg is equivalent to about 40 mg of moxisylyte.

In the management of **peripheral vascular disease**, the usual oral dose is the equivalent of 40 mg of moxisylyte four times daily increased if necessary to 80 mg four times daily. It should be withdrawn if there is no response in 2 weeks.

Moxisylyte has been used locally in the eye to reverse the mydriasis caused by phenylephrine and other sympathomimetics. It has also been used orally in benign prostatic hyperplasia, although such use has been associated with hepatotoxicity; the doses used in prostatic hyperplasia were generally higher than those in peripheral vascular disease.

◊ Reviews.

1. Marquer C, Bressolle F. Moxisylyte: a review of its pharmacodynamic and pharmacokinetic properties, and its therapeutic use in impotence. *Fundam Clin Pharmacol* 1998; **12:** 377–87.

Preparations

BP 2008: Moxisylyte Tablets.

Proprietary Preparations (details are given in Part 3)
Fr.: Carlytene; Icavex†; ***Irl.:*** Opilon; ***Port.:*** Arlitene†; ***UK:*** Opilon.

Moxonidine *(BAN, USAN, rINN)*

BDF-5895; BDF-5896; BE-5895; LY-326869; Moksonidi; Moksonidiini; Moksonidin; Moksonidinas; Moxonid; Moxonidin; Moxonidina; Moxonidinum; Moxonidum. 4-Chloro-5-(2-imidazolin-2-ylamino)-6-methoxy-2-methylpyrimidine.

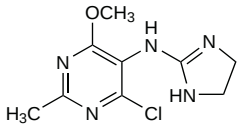
Моксонидин

$C_9H_{12}ClN_5O$ = 241.7.

CAS — 75438-57-2.

ATC — C02AC05.

ATC Vet — QC02AC05.



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

Ph. Eur. 6.2 (Moxonidine). A white or almost white powder. Very slightly soluble in water and in acetonitrile; slightly soluble in dichloromethane; sparingly soluble in methyl alcohol.

Adverse Effects and Treatment

Moxonidine has similar adverse effects to clonidine (p.1247) but causes less sedation. The incidence of dry mouth may also be lower.

Precautions

Moxonidine should not be used in patients with conduction disorders, bradycardia, severe arrhythmias, severe heart failure, severe ischaemic heart disease, severe hepatic or renal impairment, or a history of angioedema. Licensed product information suggests that it should also be avoided in patients with intermittent claudication or Raynaud's disease, Parkinson's disease, epilepsy, glaucoma, and depression. Moxonidine is distributed into breast milk and should not be used during breast feeding.

Although rebound hypertension has not been reported after moxonidine withdrawal it should not be stopped abruptly but should be withdrawn gradually over 2 weeks. As for clonidine (p.1247), if patients are also receiving a beta blocker, this should be stopped several days before moxonidine is withdrawn.

Interactions

The hypotensive effect of moxonidine may be enhanced by other antihypertensives and drugs that cause hypotension. The effect of sedatives and hypnotics, including benzodiazepines, may be enhanced by moxonidine.

Pharmacokinetics

Moxonidine is well absorbed when given orally and has a bioavailability of about 88%. Peak plasma concentrations occur 0.5 to 3 hours after an oral dose. It is excreted almost entirely in the urine as unchanged drug and metabolites; about 50 to 75% of an oral dose is excreted as unchanged drug. The mean plasma elimination half-life is 2 to 3 hours and is prolonged in renal impairment. Moxonidine is about 7% bound to plasma proteins. It is distributed into breast milk.

Uses and Administration

Moxonidine is a centrally acting antihypertensive structurally related to clonidine (p.1247). It appears to act through stimulation of central imidazoline receptors to reduce sympathetic tone, and also has $alpha_2$-adrenoceptor agonist activity. It is used in the treatment of hypertension (p.1171) and has also been investigated for heart failure (but see below).

In the treatment of hypertension, moxonidine is given orally in a usual initial dose of 200 micrograms once daily. The dose may be increased if necessary, after 3 weeks, to 400 micrograms daily as a single dose or in 2 divided doses, and after a further 3 weeks, to a maximum dose of 600 micrograms daily in 2 divided doses. The dose should be reduced in patients with renal impairment (see below).

◊ References.

1. Chrisp P, Faulds D. Moxonidine: a review of its pharmacology, and therapeutic use in essential hypertension. *Drugs* 1992; **44:** 993–1012.
2. Schachter M, *et al.* Safety and tolerability of moxonidine in the treatment of hypertension. *Drug Safety* 1998; **19:** 191–203.
3. Bousquet P, Feldman J. Drugs acting on imidazoline receptors: a review of their pharmacology, their use in blood pressure control and their potential interest in cardioprotection. *Drugs* 1999; **58:** 799–812.
4. Schachter M. Moxonidine. *Prescribers' J* 1999; **39:** 113–17.
5. Fenton C, *et al.* Moxonidine: a review of its use in essential hypertension. *Drugs* 2006; **66:** 477–96.

Administration in renal impairment. UK licensed product information states that in patients with moderate renal impairment (GFR 30 to 60 mL/minute) single doses of moxonidine should not exceed 200 micrograms and the daily dose should not exceed 400 micrograms; moxonidine should not be given in severe impairment (GFR less than 30 mL/minute).

Heart failure. Heart failure is usually treated with diuretics, ACE inhibitors, and beta blockers (see p.1165). Beta blockers are thought to act by suppressing the sympathetic nervous system, which is activated in heart failure. Centrally-acting antihypertensives such as moxonidine also suppress sympathetic activation and might therefore have a role in heart failure. A study[1] in patients with heart failure found that moxonidine reduced plasma-noradrenaline concentrations and increased left ventricular ejection fraction, but also led to an increase in adverse effects. A further study[2] was stopped early due to increased mortality in the group receiving moxonidine.

1. Swedberg K, *et al.* Effects of sustained-release moxonidine, an imidazoline agonist, on plasma norepinephrine in patients with chronic heart failure. *Circulation* 2002; **105:** 1797–1803.
2. Cohn JN, *et al.* Adverse mortality effect of central sympathetic inhibition with sustained-release moxonidine in patients with heart failure (MOXCON). *Eur J Heart Fail* 2003; **5:** 659–67.

Preparations

Proprietary Preparations (details are given in Part 3)
Austral.: Physiotens; ***Austria:*** Monox; Moxin; Normoxin; ***Belg.:*** Gilutens†; Moxon; ***Braz.:*** Cynt; ***Cz.:*** Cynt; Moxogamma; Moxostad; Physiotens; ***Denm.:*** Moxonat; Physiotens; ***Fin.:*** Physiotens; ***Fr.:*** Physiotens; ***Ger.:*** Cynt; Moxobeta; Moxocard; moxodura; Moxogamma; Physiotens; ***Gr.:*** Cynt; Fisiotens; ***Hong Kong:*** Physiotens; ***Hung.:*** Cynt; Moxogamma; Moxostad; Physiotens; ***Indon.:*** Physiotens; ***Ital.:*** Fisiotens; ***Malaysia:*** Physiotens; ***Neth.:*** Moxamar; Moxaviv; Moxoham; Moxonur; Moxotel; Moxovasc; Normatens; Ratiomox; ***Norw.:*** Physiotens; ***Philipp.:*** Physiotens; ***Pol.:*** Moxogamma; Physiotens; ***Port.:*** Moxon; ***Rus.:*** Cynt (Цинт)†; Physiotens (Физиотенз); ***S.Afr.:*** Physiotens; ***Singapore:*** Physiotens†; ***Spain:*** Moxon; ***Swed.:*** Physiotens; ***Switz.:*** Physiotens; ***Turk.:*** Cynt; ***UK:*** Physiotens.

Nadolol *(BAN, USAN, rINN)* ⊗

Nadololi; Nadololis; Nadololum; SQ-11725. (2*R*,3*S*)-5-(3-*tert*-Butylamino-2-hydroxypropoxy)-1,2,3,4-tetrahydronaphthalene-2,3-diol.

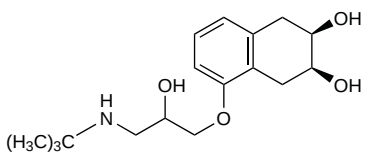
Надолол

$C_{17}H_{27}NO_4$ = 309.4.

CAS — 42200-33-9.

ATC — C07AA12.

ATC Vet — QC07AA12.



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

Ph. Eur. 6.2 (Nadolol). A white or almost white crystalline powder. Slightly soluble in water; freely soluble in alcohol; practically insoluble in acetone.

USP 31 (Nadolol). A white or off-white, practically odourless, crystalline powder. Soluble in water at pH 2; slightly soluble in water at pH 7 to 10; freely soluble in alcohol and in methyl alcohol; insoluble in acetone, in ether, in petroleum spirit, in trichloroethane, and in benzene; slightly soluble in chloroform, in dichloromethane, and in isopropyl alcohol.

Adverse Effects, Treatment, and Precautions

As for Beta Blockers, p.1226.

Breast feeding. Nadolol is distributed into breast milk and concentrations in milk are higher than those in maternal plasma. In a study[1] in 12 normotensive women given nadolol 80 mg daily by mouth for 5 days, the mean nadolol concentration in milk for the 24 hours after the last dose was 357 nanograms/mL; the equivalent mean serum-nadolol concentration was only 77 nanograms/mL. It was calculated that a 5-kg infant would therefore ingest about 2 to 7% of an equivalent adult dose. No adverse effects have been seen in breast-fed infants whose mothers were given nadolol and the American Academy of Pediatrics considers[2] that it is therefore usually compatible with breast feeding.

1. Devlin RG, *et al.* Nadolol in human serum and breast milk. *Br J Clin Pharmacol* 1981; **12:** 393–6.
2. American Academy of Pediatrics. The transfer of drugs and other chemicals into human milk. *Pediatrics* 2001; **108:** 776–89. Correction. *ibid.*; 1029. Also available at: http://aappolicy.aappublications.org/cgi/content/full/pediatrics%3b108/3/776 (accessed 10/01/08)

Hypersensitivity. Hypersensitivity pneumonitis was associated with nadolol in a patient given the drug for migraine.[1] Symptoms improved when nadolol was withdrawn.

1. Levy MB, *et al.* Nadolol and hypersensitivity pneumonitis. *Ann Intern Med* 1986; **105:** 806–7.

Interactions

The interactions associated with beta blockers are discussed on p.1228.

Pharmacokinetics

Nadolol is incompletely absorbed from the gastrointestinal tract to give peak plasma concentrations about 3 or 4 hours after a dose. It has low lipid solubility. Nadolol is widely distributed and concentrations found in breast milk have been higher than those in serum. It is only about 30% bound to plasma proteins. It does not appear to be metabolised and is excreted mainly in the urine. The plasma half-life has been reported as ranging from about 12 to 24 hours. Nadolol is reported to be dialysable.

◊ In 4 patients with mild hypertension given nadolol 2 mg orally or intravenously, the elimination half-life from plasma was an average of 10 to 12 hours (a range of 5.9 to 12.2 hours after intravenous doses, and a range of 9.6 to 14.2 hours after oral doses). Calculations based on urinary excretion and plasma concentration data suggested that about 33% was absorbed after oral dosage. There was evidence of biliary as well as urinary excretion since after intravenous dosage about 73% was excreted in urine and 23% in faeces. Nadolol did not appear to be metabolised.[1] In a similar study of therapeutic oral doses, terminal half-lives ranging from 14 to 17 hours were reported for nadolol 80 mg given as a single dose and the same dose daily in a multiple dosage regimen.[2]

1. Dreyfuss J, *et al.* Metabolic studies in patients with nadolol: oral and intravenous administration. *J Clin Pharmacol* 1977; **17:** 300–7.
2. Dreyfuss J, *et al.* Pharmacokinetics of nadolol, a beta-receptor antagonist: administration of therapeutic single- and multiple-dosage regimens to hypertensive patients. *J Clin Pharmacol* 1979; **19:** 712–20.

Children. The pharmacokinetics of nadolol given intravenously and orally were studied in six children aged 3 months to 14 years.[1] The elimination half-lives for the two oldest children aged 10 and 14 years were 7.3 and 15.7 hours, respectively. These values are similar to those reported for adults whereas in the children 22 months of age or younger, shorter half-lives of 3.2 to 4.3 hours were found. The shorter half-lives were probably a result of a reduction in the total apparent volume of distribution of nadolol in the youngest children. Elimination rates were similar after either intravenous or oral dosage.

1. Mehta AV, *et al.* Pharmacokinetics of nadolol in children with supraventricular tachycardia. *J Clin Pharmacol* 1992; **32:** 1023–7.

Uses and Administration

Nadolol is a non-cardioselective beta blocker (p.1225). It is reported to lack intrinsic sympathomimetic and

membrane-stabilising activity. Nadolol is given orally in the management of hypertension (p.1171), angina pectoris (p.1157), and cardiac arrhythmias (p.1160). It is also used in the management of hyperthyroidism (p.2165) and in the prophylactic treatment of migraine (p.616).

In the treatment of **hypertension**, nadolol is usually given in an initial dose of 40 to 80 mg once daily, increased weekly according to response to 240 mg or more daily.

In **angina pectoris**, the usual initial dose is 40 mg once daily, increased weekly according to response to usual doses of up to 160 mg daily; some patients may require up to 240 mg daily. Doses of 40 to 160 mg once daily have also been given for **cardiac arrhythmias**.

Doses of 40 to 160 mg once daily are used in **migraine** prophylaxis.

As an adjunct in the treatment of **hyperthyroidism**, doses of 80 to 160 mg once daily have been given; most patients are reported to require the higher dose.

Patients with renal impairment may require a reduction in dose (see below).

Administration in renal impairment. Nadolol is excreted mainly in the urine and doses should be reduced in patients with renal impairment, usually by increasing the dosage interval. For patients with hypertension or angina pectoris, US licensed product information recommends the following dosage intervals, based on creatinine clearance (CC):

- CC between 31 and 50 mL/minute per 1.73 m^2: give every 24 to 36 hours
- CC between 10 and 30 mL/minute per 1.73 m^2: give every 24 to 48 hours
- CC less than 10 mL/minute per 1.73 m^2: give every 40 to 60 hours.

Preparations

USP 31: Nadolol and Bendroflumethiazide Tablets; Nadolol Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Corgard; ***Belg.:*** Corgard; ***Braz.:*** Corgard; ***Canad.:*** Apo-Nadol; Corgard; ***Chile:*** Corgard; ***Fr.:*** Corgard; ***Ger.:*** Solgol; ***Hong Kong:*** Apo-Nadol; Corgard†; ***Ital.:*** Corgard; ***Malaysia:*** Corgard†; ***Mex.:*** Corgard; ***NZ:*** Corgard; ***Port.:*** Anabet; ***S.Afr.:*** Corgard; ***Spain:*** Corgard; Solgol; ***Switz.:*** Corgard; ***UK:*** Corgard; ***USA:*** Corgard; ***Venez.:*** Corgard.

Multi-ingredient: ***Ger.:*** Sotaziden N; ***Mex.:*** Corgaretic; ***S.Afr.:*** Corgaretic; ***UK:*** Corgaretic†; ***USA:*** Corzide.

Nadroparin Calcium *(BAN, rINN)*

CY-216; Nadropariinikalsium; Nadroparin Kalsiyum; Nadroparin vápenatá sůl; Nadroparina cálcica; Nadroparine calcique; Nadroparinkalcium; Nadroparin-kalcium; Nadroparino kalcio druska; Nadroparinum calcicum.

Надропарин Кальций

ATC — B01AB06.
ATC Vet — QB01AB06.

Pharmacopoeias. In *Eur.* (see p.vii).
Ph. Eur. 6.2 (Nadroparin Calcium). It is prepared by nitrous acid depolymerisation of heparin obtained from the intestinal mucosa of pigs. The majority of the components have a 2-*O*-sulfo-α-L-idopyranosuronic acid structure at the non-reducing end and a 6-*O*-sulfo-2,5-anhydro-D-mannitol structure at the reducing end of their chain. The mass-average molecular mass ranges between 3600 and 5000, with a characteristic value of 4300. The mass percentage of chains lower than 2000 is not more than 15%. The degree of sulfation is about 2 per disaccharide unit.
The potency is not less than 95 units and not more than 130 units of anti-factor Xa activity per mg with reference to the dried substance, and the ratio of anti-factor Xa activity to anti-factor IIa (antithrombin) activity is between 2.5 and 4.0.

Profile

Nadroparin calcium is a low-molecular-weight heparin (p.1329) with anticoagulant properties. It is used in the treatment and prophylaxis of venous thromboembolism (p.1189) and to prevent clotting during extracorporeal circulation. It is also used in the management of unstable angina (p.1157).

Doses are expressed in terms of anti-factor Xa activity (anti-Xa units) although different values may be encountered in the literature depending upon the reference preparation used. For *prophylaxis* of **venous thromboembolism** during surgery, patients at moderate risk of thrombosis are given 2850 units of nadroparin calcium by subcutaneous injection daily for at least 7 days or until the patient is ambulant; the first dose is given 2 to 4 hours before the procedure. For patients at high risk of thrombosis the dose is adjusted according to body-weight. Usual doses are 38 units/kg 12 hours before surgery, 12 hours postoperatively and then daily until 3 days after the procedure; the dose is then increased by 50% to 57 units/kg daily. The total duration of treatment should be at least 10 days.

For the *treatment* of thromboembolism, nadroparin calcium is given in a dose of 85 units/kg by subcutaneous injection every 12 hours for up to 10 days. Alternatively, a dose of 171 units/kg is given once daily.

For prevention of clotting in the extracorporeal circulation during **haemodialysis** sessions lasting less than 4 hours, nadroparin calcium is given into the arterial line of the circuit at the beginning of the dialysis session. The usual dose is 2850 units for patients weighing less than 50 kg, 3800 units for patients weighing 50 to 69 kg, and 5700 units for patients weighing 70 kg or more. Doses should be reduced in patients at high risk of haemorrhage.

In the management of unstable **angina**, nadroparin calcium is given subcutaneously in a dose of 86 units/kg every 12 hours, for about 6 days. An initial dose of 86 units/kg may be given intravenously. Low-dose aspirin should also be given.

Elimination of nadroparin is prolonged in renal impairment, and doses may need to be reduced in moderate or severe impairment.

◊ References.

1. Barradell LB, Buckley MM. Nadroparin calcium: a review of its pharmacology and clinical applications in the prevention and treatment of thromboembolic disorders. *Drugs* 1992; **44:** 858–88.

Preparations

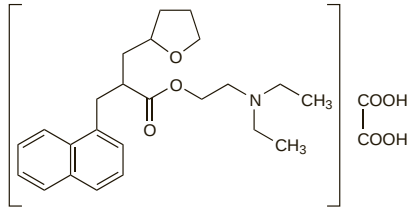
Proprietary Preparations (details are given in Part 3)
Arg.: Fraxiparine; ***Austria:*** Fraxiparin; ***Belg.:*** Fraxiparine; Fraxodi; ***Braz.:*** Fraxiparina; ***Canad.:*** Fraxiparine; ***Chile:*** Fraxiparine; ***Cz.:*** Fraxiparine; ***Fr.:*** Fraxiparine; Fraxodi; ***Ger.:*** Fraxiparin; Fraxodi; ***Gr.:*** Fraxiparine; ***Hong Kong:*** Fraxiparine; ***Hung.:*** Fraxiparine; Fraxodi; ***Indon.:*** Fraxiparine; ***Israel:*** Fraxiparine; ***Ital.:*** Fraxiparina; Fraxodi; Seledie; Seleparina; ***Malaysia:*** Fraxiparine; ***Mex.:*** Fraxiparine; Fraxodi; ***Neth.:*** Fraxiparine; Fraxodi; ***Norw.:*** Fraxiparine†; ***NZ:*** Fraxiparine†; ***Philipp.:*** Fraxiparine; ***Pol.:*** Fraxiparine; Fraxodi; ***Port.:*** Fraxiparina; Fraxodi; ***Rus.:*** Fraxiparine (Фраксипарин); ***S.Afr.:*** Fraxiparine; ***Singapore:*** Fraxiparine; ***Spain:*** Fraxiparina; ***Swed.:*** Fraxiparine†; ***Switz.:*** Fraxiforte; Fraxiparine; ***Thai.:*** Fraxiparine; ***Turk.:*** Fraxiparine; Fraxodi; ***Venez.:*** Fraxiparina.

Naftidrofuryl Oxalate *(BANM, rINNM)*

EU-1806; LS-121; Nafronyl Oxalate *(USAN)*; Naftidrofuril-hidrogén-oxalát; Naftidrofurilio-vandenilio oksalatas; Naftidrofuryl Hydrogen Oxalate; Naftidrofuryl, hydrogénooxalate de; Naftidrofuryl, Oxalate de; Naftidrofuryli hydrogenooxalas; Naftidrofuryli Oxalas; Naftidrofuryl-oxalát; Naftidrofurylväteoxalat; Naftidrofuryylivetyoksalaatti; Oxalato de naftidrofurilo. 2-Diethylaminoethyl 3-(1-naphthyl)-2-tetrahydrofurfurylpropionate hydrogen oxalate.

Нафтидрофурила Оксалат

$C_{24}H_{33}NO_3.C_2H_2O_4$ = 473.6.
CAS — 31329-57-4 (naftidrofuryl); 3200-06-4 (naftidrofuryl oxalate).
ATC — C04AX21.
ATC Vet — QC04AX21.



Pharmacopoeias. In *Eur.* (see p.vii).
Ph. Eur. 6.2 (Naftidrofuryl Hydrogen Oxalate; Naftidrofuryl Oxalate BP 2008). A white or almost white powder. Freely soluble in water; freely soluble or soluble in alcohol; slightly or sparingly soluble in acetone.

Adverse Effects

Naftidrofuryl oxalate given orally may cause nausea and epigastric pain. Rash has been reported occasionally. Hepatitis or hepatic failure has occurred rarely. Convulsions and depression of cardiac conduction may occur after overdosage. After intravenous use cardiac arrhythmias, hypotension, and convulsions have been reported and intravenous preparations have been withdrawn from the market (see below).

◊ In early 1995 the UK CSM published details of adverse reactions to naftidrofuryl.[1] After parenteral doses of naftidrofuryl 47 reports of 79 reactions had been received, the most serious consequences being 9 cases of cardiac arrhythmias, 3 of convulsions, and 2 of hypotension. It was also noted that 2 fatal cases of cardiac arrest had occurred in Germany after bolus intravenous doses and it was stressed that the drug must not be given as a bolus but as a slow intravenous infusion. Additionally, 16 reports, including one fatality, of hepatitis or hepatic failure associated with oral naftidrofuryl had been received although this appeared to be a rare reaction.

Later in 1995, after a review conducted in the UK and Europe, it was announced by the CSM that intravenous naftidrofuryl was to be withdrawn.[2] It was considered that the risks of cardiac and neurological toxicity outweighed the benefits of intravenous dosage in peripheral vascular disease. The oral form of naftidrofuryl would remain available.

1. Committee on Safety of Medicines/Medicines Control Agency. Adverse reactions with naftidrofuryl (Praxilene). *Current Problems* 1995; **21:** 2. Available at: http://www.mhra.gov.uk/home/idcplg?IdcService=GET_FILE&dDocName=CON2015618&RevisionSelectionMethod=LatestReleased (accessed 08/05/08)
2. Committee on Safety of Medicines/Medicines Control Agency. Withdrawal of naftidrofuryl infusion (Praxilene Forte). *Current Problems* 1995; **21:** 7. Available at: http://www.mhra.gov.uk/home/idcplg?IdcService=GET_FILE&dDocName=CON2015619&RevisionSelectionMethod=LatestReleased (accessed 08/05/08)

Effects on the kidneys. Calcium oxalate crystals in the renal tubules of 2 patients with acute renal failure[1] were associated with the high amounts of oxalate they had received when naftidrofuryl oxalate was given intravenously.

1. Moesch C, *et al.* Renal intratubular crystallisation of calcium oxalate and naftidrofuryl oxalate. *Lancet* 1991; **338:** 1219–20.

Uses and Administration

Naftidrofuryl oxalate is used as a vasodilator in the treatment of peripheral (p.1178) and cerebral vascular disorders (p.1165). It is also claimed to enhance cellular oxidative capacity thereby protecting cells against the results of ischaemia.

Naftidrofuryl oxalate is given orally in usual doses of 100 to 200 mg three times daily for peripheral vascular disorders and 100 mg three times daily for cerebrovascular disorders.

Naftidrofuryl oxalate has also been given parenterally. However, intravenous use has been associated with serious adverse effects (see above) and intravenous preparations have been withdrawn.

◊ References.

1. De Backer TLM, *et al.* Naftidrofuryl for intermittent claudication. Available in The Cochrane Database of Systematic Reviews; Issue 2. Chichester: John Wiley; 2008 (accessed 08/05/08).

Preparations

BP 2008: Naftidrofuryl Capsules.

Proprietary Preparations (details are given in Part 3)
Arg.: Iridus†; ***Austria:*** Dusodril; Naftodril; ***Belg.:*** Praxilene; ***Braz.:*** Iridux; ***Cz.:*** Enelbin; ***Fr.:*** Di-Actane; Gevatran; Naftilux; Praxilene; ***Ger.:*** Artocoron†; Azunaftil†; Dusodril; Nafti; Naftilong; ***Gr.:*** Praxilene; ***Hong Kong:*** Praxilene; ***Hung.:*** Naftilong; ***Indon.:*** Frilix; Nafoxal; Praxilene; Vascuprax; ***Irl.:*** Praxilene; ***Ital.:*** Praxilene; ***Mex.:*** Iridus; ***Philipp.:*** Praxilene; ***Port.:*** Praxilene; ***Singapore:*** Praxilene; ***Spain:*** Praxilene; ***Switz.:*** Praxilene; Sodipryl retard; ***Thai.:*** Praxilene; ***UK:*** Praxilene; ***Venez.:*** Fuxaten†; Iridus.

Nasaruplase *(rINN)*

Nasaruplasa; Nasaruplasum; Prourokinase, Glycosylated. Prourokinase (enzyme-activating) (human clone pA3/pD2/pF1 protein moiety), glycosylated.

Назаруплаза

CAS — 99821-44-0.

NOTE. The term prourokinase has been used for both nasaruplase and saruplase (p.1390).

Nasaruplase Beta *(USAN, rINN)*

Abbott-74187; ABT-187; Nasaruplasa beta; Nasaruplase Bêta; Nasaruplasum Beta. Prourokinase (enzyme-activating) human (clone pUK4/pUK18 protein moiety), glycosylated (murine cell line SP2/0).

Назаруплаза Бета

CAS — 136653-69-5.

Profile

Nasaruplase is a thrombolytic under investigation in acute ischaemic stroke.

◊ References.

1. Furlan A, *et al.* Intra-arterial prourokinase for acute ischemic stroke. The PROACT II study: a randomized controlled trial. *JAMA* 1999; **282:** 2003–11.

Nateplase *(rINN)*

Nateplasa; Natéplase; Nateplasum. A mixture of *N*-[N^2-(*N*-glycyl-L-alanyl)-L-arginyl]plasminogen activator (human tissue-type 1-chain form, protein moiety), glycoform β (major component) and plasminogen activator (human tissue-type 1-chain form, protein moiety), glycoform β.

Натеплаза

CAS — 159445-63-3.

Profile

Nateplase is a thrombolytic related to alteplase (p.1207) that has been used in acute myocardial infarction (p.1175).

Preparations

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