Natriuretic Peptides ⊗

Péptidos natriuréticos.

Profile

Natriuretic peptides are endogenous substances that possess diuretic, natriuretic, and vasodilator properties. Three types are known. *Atrial natriuretic peptide* (ANP), also known as atrial natriuretic factor (ANF), atriopeptin, auriculin, or cardionatrin, is produced mainly in the cardiac atria, although another form, ularitide (urodilatin), is produced in the kidney. *Brain natriuretic peptide* (BNP, B-type natriuretic peptide) was originally isolated from brain tissue but is now known to be mainly produced by the cardiac ventricles. *C-type natriuretic peptide* (CNP) is produced by the endothelium and appears to act locally as a vasodilator but has little natriuretic effect.

Natriuretic peptides have an important physiological role in fluid and electrolyte homoeostasis and in the regulation of blood pressure, and they interact closely with other complex systems such as the renin-angiotensin-aldosterone cascade. Plasma concentrations of atrial natriuretic peptide and brain natriuretic peptide are altered in some pathological states and have been used as indicators of cardiac function. Natriuretic peptides that have been investigated for therapeutic use include anaritide, a synthetic form of atrial natriuretic peptide, and ularitide; both have been studied in acute renal failure, and ularitide has also been studied in heart failure. Recombinant forms of atrial natriuretic peptide (carperitide, p.1241) and brain natriuretic peptide (nesiritide, p.1347) are used in the management of acute heart failure.

The currently available natriuretic peptides have short half-lives and have to be given parenterally. Other approaches to manipulating their effects have been investigated, including the use of atriopeptidase inhibitors (neutral endopeptidase inhibitors; neutral metalloendopeptidase inhibitors), such as candoxatrilat and ecadotril (sinorphan) to prolong the half-life of endogenous atrial natriuretic peptide. Compounds such as omapatrilat (p.1361) that inhibit both neutral endopeptidase and angiotensin-converting enzyme are also being studied.

◊ References.

1. Tan ACITL, *et al.* Atrial natriuretic peptide: an overview of clinical pharmacology and pharmacokinetics. *Clin Pharmacokinet* 1993; **24:** 28–45.
2. Richards AM. The renin-angiotensin-aldosterone system and the cardiac natriuretic peptides. *Heart* 1996; **76** (suppl 3): 36–44.
3. Wilkins MR, *et al.* The natriuretic-peptide family. *Lancet* 1997; **349:** 1307–10.
4. Levin ER, *et al.* Natriuretic peptides. *N Engl J Med* 1998; **339:** 321–8.
5. Lewis J, *et al.* Atrial natriuretic factor in oliguric acute renal failure: Anaritide Acute Renal Failure Study Group. *Am J Kidney Dis* 2000; **36:** 767–74.
6. Forssmann W, *et al.* The renal urodilatin system: clinical implications. *Cardiovasc Res* 2001; **51:** 450–62.
7. de Lemos JA, *et al.* B-type natriuretic peptide in cardiovascular disease. *Lancet* 2003; **362:** 316–22.
8. Weber M, Hamm C. Role of B-type natriuretic peptide (BNP) and NT-proBNP in clinical routine. *Heart* 2006; **92:** 843–9.
9. Mitrovic V, *et al.* Haemodynamic and clinical effects of ularitide in decompensated heart failure. *Eur Heart J* 2006; **27:** 2823–32.
10. Lüss H, *et al.* Renal effects of ularitide in patients with decompensated heart failure. *Am Heart J* 2008; **155:** 1012.e1–8.

Nebivolol *(BAN, USAN, rINN)* ⊗

Narbivolol; Nébivolol; Nebivololi; Nebivololum; R-65824. (1*RS*,1′*RS*)-1,1′-[(2*RS*,2′*SR*)-Bis(6-fluorochroman-2-yl)]-2,2′-iminodiethanol.

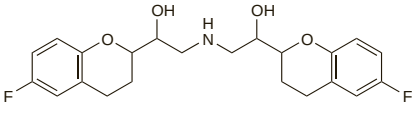
Небиволол

$C_{22}H_{25}F_2NO_4$ = 405.4.

CAS — 99200-09-6; 118457-14-0.

ATC — C07AB12.

ATC Vet — QC07AB12.



Nebivolol Hydrochloride *(BANM, USAN, rINNM)* ⊗

Hidrocloruro de nebivolol; Nébivolol, Chlorhydrate de; Nebivololi Hydrochloridum; R-67555; R-067555.

Небиволола Гидрохлорид

$C_{22}H_{25}F_2NO_4$,HCl = 441.9.

CAS — 169293-50-9; 152520-56-4.

ATC — C07AB12.

ATC Vet — QC07AB12.

Adverse Effects, Treatment, and Precautions

As for Beta Blockers, p.1226.

Interactions

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

Pharmacokinetics

Nebivolol is rapidly absorbed after oral doses. It is extensively metabolised in the liver by alicyclic and aromatic hydroxylation, *N*-dealkylation, and glucuronidation; the hydroxy metabolites are reported to be active. The rate of aromatic hydroxylation by cytochrome P450 isoenzyme CYP2D6 is subject to genetic polymorphism, and bioavailability and half-life vary widely. In fast metabolisers the elimination half-life of nebivolol is about 10 hours and that of the hydroxy metabolites is about 24 hours. Peak plasma concentrations of unchanged drug plus active metabolites are 1.3 to 1.4 times higher in slow metabolisers and the half-lives of nebivolol and its hydroxy metabolites are prolonged.

Nebivolol is about 98% bound to plasma proteins. It has high lipid solubility. It is excreted in the urine and faeces, almost entirely as metabolites. Nebivolol is distributed into breast milk in *animals*.

Uses and Administration

Nebivolol is a cardioselective beta blocker (p.1225). It has vasodilating activity, which appears to be due to a direct action on the endothelium, possibly involving nitric oxide release. It is reported to lack intrinsic sympathomimetic and membrane-stabilising activity.

Nebivolol is used in the management of hypertension (p.1171), and as an adjunct to standard therapy in patients aged 70 years and older with stable chronic heart failure (p.1165). It is given orally as the hydrochloride although doses are expressed in terms of the base; 5.45 mg of nebivolol hydrochloride is equivalent to about 5 mg of base.

In **hypertension** the usual initial dose of nebivolol is 5 mg once daily. US licensed product information allows the dose to be increased, if necessary, at intervals of 2 weeks, to a maximum dose of 40 mg once daily. Dosage reduction may be necessary in the elderly and in patients with hepatic or renal impairment (see below).

In **heart failure** the initial dose of nebivolol is 1.25 mg once daily. If tolerated, the dose should be doubled every 1 to 2 weeks up to a maximum of 10 mg once daily.

◊ Reviews.

1. Moen MD, Wagstaff AJ. Nebivolol: a review of its use in the management of hypertension and chronic heart failure. *Drugs* 2006; **66:** 1389–1409.
2. Veverka A, *et al.* Nebivolol: a third-generation β-adrenergic blocker. *Ann Pharmacother* 2006; **40:** 1353–60.
3. Agabiti Rosei E, Rizzoni D. Metabolic profile of nebivolol, a β-adrenoceptor antagonist with unique characteristics. *Drugs* 2007; **67:** 1097–1107.
4. Prisant LM. Nebivolol: pharmacologic profile of an ultraselective, vasodilatory β -blocker. *J Clin Pharmacol* 2008; **48:** 225–39.

Administration in the elderly. UK licensed product information states that, for hypertension, patients over 65 years of age should be given an initial dose of 2.5 mg of nebivolol once daily, increased to 5 mg once daily if required.

Administration in hepatic impairment. UK licensed product information contra-indicates the use of nebivolol in patients with hepatic impairment. In the USA, licensed product information also contra-indicates nebivolol in severe hepatic impairment (Child-Pugh higher than class B) but patients with moderate hepatic impairment may be given nebivolol for hypertension in an initial oral dose of 2.5 mg once daily, increased with caution if required.

Administration in renal impairment. UK licensed product information states that in hypertension the initial dose of nebivolol should be reduced to 2.5 mg once daily in patients with renal impairment, increased to 5 mg once daily for maintenance if required. US licensed product information similarly recommends an initial dose of 2.5 mg once daily in patients with severe renal impairment (creatinine clearance below 30 mL/minute); the dose may be increased cautiously if required.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Nebilet; ***Austria:*** Nomexor; ***Belg.:*** Nobiten; ***Chile:*** Nebilet; ***Cz.:*** Nebilet; Nebispes; ***Fr.:*** Nebilox; Temerit; ***Ger.:*** Nebilet; ***Gr.:*** Lobivon; ***Hung.:*** Nebilet; ***India:*** Nodon; ***Irl.:*** Nebilet; ***Ital.:*** Lobivon; Nebilox; ***Neth.:*** Hypoloc; Lobivon; Nebilet; Nebiloc; ***Pol.:*** Nebilet; ***Port.:*** Hypoloc; Nebilet; ***Rus.:*** Nebilet (Небилет); ***S.Afr.:*** Nebilet; ***Singapore:*** Nebilet; ***Spain:*** Lobivon; Nebilet; Nebilox; Silostar; ***Switz.:*** Nebilet; ***Thai.:*** Nebilet; ***Turk.:*** Vasoxen; ***UK:*** Nebilet; ***USA:*** Bystolic; ***Venez.:*** Nebilet.

Nesiritide Citrate *(USAN, rINNM)* ⊗

Citrato de nesiritida; Nésiritide, Citrate de; Nesiritidi Citras.

Незиритида Цитрат

$C_{143}H_{244}N_{50}O_{42}S_4$,x$C_6H_8O_7$.

CAS — 124584-08-3 (nesiritide); 189032-40-4 (nesiritide citrate).

ATC — C01DX19.

ATC Vet — QC01DX19.

Incompatibility. The manufacturer states that nesiritide injection is physically and/or chemically incompatible with heparin, insulin, sodium etacrynate, bumetanide, enalaprilat, hydralazine, furosemide, and the preservative sodium metabisulfite. Nesiritide binds to heparin and should not be given through heparin-coated central catheters.

Adverse Effects and Precautions

The most common adverse effects of nesiritide relate to vasodilatation and include hypotension, headache, and dizziness. Nausea and vomiting, abdominal pain, back pain, angina pectoris, insomnia, and anxiety, have also been reported. Cardiac arrhythmias have occurred but may be associated with the underlying condition. Adverse effects on renal function have been reported. If hypotension occurs the infusion of nesiritide should be stopped or the dose reduced and general supportive measures should be used; the hypotension may persist for several hours.

Nesiritide should not be used as primary therapy in patients with cardiogenic shock or with hypotension. It is not recommended in patients with low cardiac filling pressures or in those for whom vasodilators are inappropriate, such as those with significant valvular stenosis, restrictive or obstructive cardiomyopathy, constrictive pericarditis, or pericardial tamponade.

Effects on the kidneys. Nesiritide has both haemodynamic and neurohormonal effects on the kidneys and has been reported to worsen renal function. A meta-analysis[1] found that nesiritide significantly increased the risk of worsening renal function in patients with acute heart failure, and there is some evidence[2] that this may be related to the duration of treatment. However, a randomised trial[3] in patients with acute heart failure and pre-existing renal impairment found that the effect of nesiritide on renal function was neutral.

1. Sackner-Bernstein JD, *et al.* Risk of worsening renal function with nesiritide in patients with acutely decompensated heart failure. *Circulation* 2005; **111:** 1487–91. Correction. *ibid.*; 2274.
2. Chow SL, *et al.* Effect of nesiritide infusion duration on renal function in acutely decompensated heart failure patients. *Ann Pharmacother* 2007; **41:** 556–61.
3. Witteles RM, *et al.* Impact of nesiritide on renal function in patients with acute decompensated heart failure and pre-existing renal dysfunction: a randomized, double-blind, placebo-controlled clinical trial. *J Am Coll Cardiol* 2007; **50:** 1835–40.

Effects on mortality. Although nesiritide improves haemodynamics in patients with acute decompensated heart failure, its effects on mortality are controversial.[1] A retrospective study[2] comparing nesiritide with inotrope therapy or glyceryl trinitrate in patients with acute decompensated heart failure found a similar risk of in-hospital mortality with nesiritide and glyceryl trinitrate, which was significantly lower than the risk with inotrope therapy. However, a meta-analysis[3] of controlled studies comparing nesiritide with non-inotrope control therapy found that there was a trend to higher mortality at 30 days in patients given nesiritide; the results were not statistically significant, but became so after correction of the number of deaths in one of the studies.[4] A later meta-analysis[5] also found a trend towards increased mortality with nesiritide at 30 days, but the results again were not statistically significant, and there was no difference in mortality between nesiritide and control patients at 180 days.

1. Yancy CW. Benefit-risk assessment of nesiritide in the treatment of acute decompensated heart failure. *Drug Safety* 2007; **30:** 765–81.
2. Abraham WT, *et al.* In-hospital mortality in patients with acute decompensated heart failure requiring intravenous vasoactive medications: an analysis from the Acute Decompensated Heart Failure National Registry (ADHERE). *J Am Coll Cardiol* 2005; **46:** 57–64.
3. Sackner-Bernstein JD, *et al.* Short-term risk of death after treatment with nesiritide for decompensated heart failure: a pooled analysis of randomized controlled trials. *JAMA* 2005; **293:** 1900–5.
4. Aaronson KD, Sackner-Bernstein J. Risk of death associated with nesiritide in patients with acutely decompensated heart failure. *JAMA* 2006; **296:** 1465–6.
5. Arora RR, *et al.* Short and long-term mortality with nesiritide. *Am Heart J* 2006; **152:** 1084–90.

Interactions

The risk of hypotension may be increased in patients receiving nesiritide with other drugs that lower blood pressure.

Pharmacokinetics

Nesiritide is cleared from the circulation by 3 mechanisms: uptake into cells; proteolytic cleavage by endopeptidases; and excretion by the kidneys. It has a biphasic elimination, with a terminal elimination half-life of 18 minutes.

Uses and Administration

Nesiritide is a recombinant brain natriuretic peptide (see p.1347) used in the management of acutely decompensated heart failure (p.1165). It is given intravenously as the citrate, but dosage is expressed in terms of the base. The initial dose of nesiritide is 2 micrograms/kg by intravenous injection over 1 minute, followed by a maintenance infusion of 10 nanograms/kg per minute.

Heart failure. The use of nesiritide in acute decompensated heart failure (p.1165) has been reviewed.[1,2] It may be used for short-term treatment as an alternative to standard intravenous therapy with vasodilators, inotropes, or diuretics, and appears to have no proarrhythmic effects; however, its effects on mortality are controversial (see under Adverse Effects and Precautions, above) and its role in therapy remains unclear. There is some evidence from retrospective studies that it may be safely used in addition to standard therapy[3,4] and may have a role as a more prolonged treatment in patients awaiting cardiac transplantation.[5] Although nesiritide has also been given intermittently for outpatient management of chronic heart failure, this use is not currently recommended.[6]

1. Vichiendilokkul A, *et al.* Nesiritide: a novel approach for acute heart failure. *Ann Pharmacother* 2003; **37:** 247–58.
2. Keating GM, Goa KL. Nesiritide: a review of its use in acute decompensated heart failure. *Drugs* 2003; **63:** 47–70.
3. O'Dell KM, *et al.* Nesiritide for secondary pulmonary hypertension in patients with end-stage heart failure. *Am J Health-Syst Pharm* 2005; **62:** 606–9.
4. Smull DL, Jorde UP. Concomitant use of nesiritide and milrinone in decompensated congestive heart failure. *Am J Health-Syst Pharm* 2005; **62:** 291–5.
5. Witteles R, *et al.* B-type natriuretic peptide is effective therapy before care. *Ann Intern Med* 2004; **141:** 895.
6. Bauer JB, Randazzo MA. Nesiritide for outpatient treatment of heart failure. *Am J Health-Syst Pharm* 2005; **62:** 2639–42.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Natrecor; ***Indon.:*** Natrecor; ***Israel:*** Noratak; ***Switz.:*** Noratak; ***USA:*** Natrecor; ***Venez.:*** Natrecor.

Nicardipine Hydrochloride

(BANM, USAN, rINNM)

Hidrocloruro de nicardipino; Nicardipine, Chlorhydrate de; Nicardipini Hydrochloridum; Nikardipiinihydrokloridi; Nikardipin Hidroklorür; Nikardipinhydroklorid; RS-69216; RS-69216-XX-07-0; YC-93. 2-[Benzyl(methyl)amino]ethyl methyl 1,4-dihydro-2,6-dimethyl-4-(3-nitrophenyl)pyridine-3,5-dicarboxylate hydrochloride.

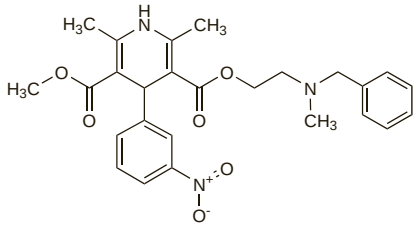
Никардипина Гидрохлорид

$C_{26}H_{29}N_3O_6$,HCl = 516.0.

CAS — 55985-32-5 (nicardipine); 54527-84-3 (nicardipine hydrochloride).

ATC — C08CA04.

ATC Vet — QC08CA04.



(nicardipine)

Pharmacopoeias. In *Chin.* and *Jpn.*

Incompatibility. The manufacturers recommend that a solution containing nicardipine hydrochloride 100 micrograms/mL is used for intravenous infusion. Suitable diluents are solutions of glucose or sodium chloride. Sodium bicarbonate and lactated Ringer's are incompatible with nicardipine infusion. Nicardipine hydrochloride (1 mg/mL in glucose 5%) has also been reported[1] to be visually incompatible with furosemide, heparin, and thiopental.

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.

Adverse Effects, Treatment, and Precautions

As for dihydropyridine calcium-channel blockers (see Nifedipine, p.1350).

Interactions

As for dihydropyridine calcium-channel blockers (see Nifedipine, p.1352).

Pharmacokinetics

Nicardipine is rapidly and completely absorbed from the gastrointestinal tract but is subject to saturable first-pass hepatic metabolism. Bioavailability of about 35% has been reported after a 30-mg dose at steady state. The pharmacokinetics of nicardipine are non-linear due to the saturable first-pass hepatic metabolism and an increase in dose may produce a disproportionate increase in plasma concentration. There is also considerable interindividual variation in plasma-nicardipine concentrations. Nicardipine is more than 95% bound to plasma proteins. Nicardipine is extensively metabolised in the liver and is excreted in the urine and faeces, mainly as inactive metabolites. The terminal plasma half-life is about 8.6 hours, thus steady-state plasma concentrations are achieved after 2 to 3 days of dosing three times daily.

◊ References.

1. Graham DJM, *et al.* Pharmacokinetics of nicardipine following oral and intravenous administration in man. *Postgrad Med J* 1984; **60** (suppl 4): 7–10.
2. Graham DJM, *et al.* The metabolism and pharmacokinetics of nicardipine hydrochloride in man. *Br J Clin Pharmacol* 1985; **20:** 23S–28S.
3. Razak TA, *et al.* The effect of hepatic cirrhosis on the pharmacokinetics and blood pressure response to nicardipine. *Clin Pharmacol Ther* 1990; **47:** 463–9.
4. Porchet HC, Dayer P. Serum concentrations and effects of (±)-nicardipine compared with nifedipine in a population of healthy subjects. *Clin Pharmacol Ther* 1990; **48:** 155–60.

Uses and Administration

Nicardipine is a dihydropyridine calcium-channel blocker with actions and uses similar to nifedipine (p.1354). It is used in the management of hypertension (p.1171) and angina pectoris (p.1157).

Nicardipine hydrochloride is generally given orally although the intravenous route has been used for the short-term treatment of hypertension.

Oral doses of nicardipine hydrochloride are similar for both **hypertension** and **angina**. The initial dose is 20 mg three times daily and may be increased at intervals of at least 3 days until the required effect is achieved. The usual maintenance dose is 30 mg three times daily, but daily doses of between 60 and 120 mg in divided doses may be given. Modified-release preparations of nicardipine hydrochloride for dosage twice daily are also available.

Nicardipine hydrochloride may be given by slow intravenous infusion as a 100 micrograms/mL solution in the short-term treatment of hypertension. An initial infusion rate of 5 mg/hour is recommended, increased, as necessary, up to a maximum of 15 mg/hour and subsequently reduced to 3 mg/hour. For use in children, see below.

Reduced doses of nicardipine hydrochloride and longer dosing intervals may be necessary in patients with hepatic or renal impairment (see below).

◊ Reviews.

1. Curran MP, *et al.* Intravenous nicardipine: its use in the short-term treatment of hypertension and various other indications. *Drugs* 2006; **66:** 1755–82.

Administration in children. Intravenous infusion of nicardipine has been used in both infants and children for the management of hypertension. In studies[1-4] in children aged between 2 days and 17 years, initial doses ranged from 0.2 to 5 micrograms/kg per minute, with maintenance infusions of 0.15 to 6 micrograms/kg per minute. Adverse effects were rare; one study[4] reported adverse effects in 5 of 31 treatment courses, including tachycardia, flushing, palpitations, and hypotension. There has also been a report[5] of the successful use of intravenous infusion of nicardipine in 8 preterm infants (gestational age 28 to 36 weeks). Infusions were given at a dose of 0.5 to 2 micrograms/kg per minute and continued for periods of 3 to 36 days. No hypotension, oedema, or tachycardia were observed.

The *BNFC* suggests that neonates and children up to age 18 years may be given nicardipine hydrochloride by continuous intravenous infusion for the management of hypertensive crises. The initial dose is 500 nanograms/kg per minute, adjusted according to response; the usual maintenance dose is 1 to 4 micrograms/kg per minute, with a maximum dose of 250 micrograms/minute.

1. Treluyer JM, *et al.* Intravenous nicardipine in hypertensive children. *Eur J Pediatr* 1993; **152:** 712–4.
2. Sartori SC, *et al.* Intravenous nicardipine for treatment of systemic hypertension in children. *Pediatrics* 1999; **104** (suppl): 676–7.
3. Tobias JD. Nicardipine to control mean arterial pressure after cardiothoracic surgery in infants and children. *Am J Ther* 2001; **8:** 3–6.
4. Flynn JT, *et al.* Intravenous nicardipine for treatment of severe hypertension in children. *J Pediatr* 2001; **139:** 38–43.
5. Gouyon JB, *et al.* Intravenous nicardipine in hypertensive preterm infants. *Arch Dis Child* 1997; **76:** F126–F127.

Administration in hepatic or renal impairment. Reduced doses of nicardipine hydrochloride and longer dosing intervals may be necessary in patients with hepatic or renal impairment. The US manufacturers recommend an initial dose of 20 mg twice daily by mouth in patients with hepatic impairment.

Cerebrovascular disorders. Nicardipine has been reported to increase cerebral blood flow[1] and use by various routes has been investigated for possible benefit in haemorrhagic[2-5] and ischaemic stroke[6,7] (p.1185), although nimodipine (p.1357) is the dihydropyridine calcium-channel blocker usually used. Nicardipine has also been tried[8] in patients with cerebrovascular insufficiency. However, studies have produced inconclusive results.

1. Savage I, James I. The effect of nicardipine hydrochloride on cerebral blood flow in normotensive volunteers. *Br J Clin Pharmacol* 1986; **21:** 591P–592P.
2. Suzuki M, *et al.* Intrathecal administration of nicardipine hydrochloride to prevent vasospasm in patients with subarachnoid hemorrhage. *Neurosurg Rev* 2001; **24:** 180–4.
3. Kasuya H, *et al.* Efficacy and safety of nicardipine prolonged-release implants for preventing vasospasm in humans. *Stroke* 2002; **33:** 1011–15.
4. Dorhout Mees SM, *et al.* Calcium antagonists for aneurysmal subarachnoid haemorrhage. Available in The Cochrane Database of Systematic Reviews; Issue 3. Chichester: John Wiley; 2007 (accessed 25/07/08).
5. Barth M, *et al.* Effect of nicardipine prolonged-release implants on cerebral vasospasm and clinical outcome after severe aneurysmal subarachnoid hemorrhage: a prospective, randomized, double-blind phase IIa study. *Stroke* 2007; **38:** 330–6.
6. Yao L, Ding D. Effect of nicardipine on somatosensory evoked potentials in patients with acute cerebral infarction. *J Neurol Neurosurg Psychiatry* 1990; **53:** 844–6.
7. Rosenbaum D, *et al.* Early treatment of ischemic stroke with a calcium antagonist. *Stroke* 1991; **22:** 437–41.
8. Silva APE, Diament CK. Nicardipine versus cinnarizine in cerebrovascular insufficiency. *Curr Ther Res* 1988; **43:** 888–99.

Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Karden; ***Belg.:*** Rydene; ***Fr.:*** Loxen; ***Ger.:*** Antagonil; ***Indon.:*** Perdipine; ***Irl.:*** Cardene†; ***Ital.:*** Bionicard; Cardioten; Cardip; Cordisol†; Lisanirc; Neucor; Nicant†; Nicapress; Nicardal; Nicarpin; Nicaven; Nimicor; Niven†; Perdipina; Ranvil†; Vasodin; ***Jpn:*** Perdipine; ***Malaysia:*** Cardepine; ***Neth.:*** Cardene; ***Philipp.:*** Cardepine; ***Port.:*** Nerdipina; ***Singapore:*** Cardibloc; ***Spain:*** Dagan; Flusemide; Lecibral; Lincil; Lucenfal; Nerdipina; Vasonase; ***Thai.:*** Cardepine; Nerdipine†; ***Turk.:*** Loxen; ***UK:*** Cardene; ***USA:*** Cardene.

Niceritrol *(BAN, rINN)*

Nicéritrol; Niceritrolum; Nikeritroli; PETN. Pentaerythritol tetranicotinate; 2,2-Bis(hydroxymethyl)propane-1,3-diol tetranicotinate.

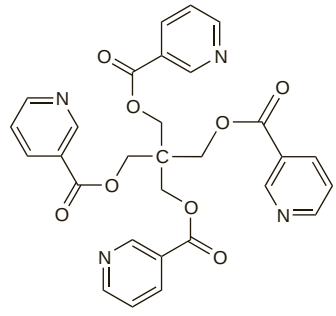
Ницеритрол

$C_{29}H_{24}N_4O_8$ = 556.5.

CAS — 5868-05-3.

ATC — C10AD01.

ATC Vet — QC10AD01.



NOTE. The synonym PETN has been applied to both niceritrol and pentaerithrityl tetranitrate.