Peripheral vascular disease. Ketanserin is one of many drugs that have been tried in the management of peripheral vascular disease (p.1178) but results have been contradictory. Subgroup analysis of the multicentre Prevention of Atherosclerotic Complications with Ketanserin Trial (PACK),[1] involving 3899 patients with intermittent claudication, suggested that ketanserin might be of benefit in preventing limb amputation in some patients. Conflicting results have also been reported in patients with Raynaud's syndrome (see Vasospastic Arterial Disorders, p.1188). A systematic review[2] found that ketanserin led to a small improvement in Raynaud's syndrome in patients with systemic sclerosis but that adverse effects increased; the authors concluded that ketanserin was not clinically beneficial in such patients.

Ketanserin has also been tried in other conditions associated with impaired peripheral blood flow: see Wounds and Ulcers, below.

1. Prevention of Atherosclerotic Complications with Ketanserin Trial Group. Prevention of atherosclerotic complications: controlled trial of ketanserin. *BMJ* 1989; **298:** 424–30. Correction. *ibid.*: 644.
2. Pope JE, *et al.* Ketanserin for Raynaud's phenomenon in progressive systemic sclerosis. Available in The Cochrane Database of Systematic Reviews; Issue 2. Chichester: John Wiley; 1998 (accessed 26/09/05).

Shivering. Numerous drugs, including ketanserin, have been tried for the treatment of postoperative shivering (p.1779). Ketanserin 10 mg given intravenously has stopped shivering after general anaesthesia.[1,2]

1. Joris J, *et al.* Clonidine and ketanserin both are effective treatment for postanesthetic shivering. *Anesthesiology* 1993; **79:** 532–9.
2. Crisinel D, *et al.* Efficacité de la kétansérine sur le frisson postanesthésique. *Ann Fr Anesth Reanim* 1997; **16:** 120–5.

Wounds and ulcers. Several controlled studies[1-5] have noted improved healing of decubitus, venous, and ischaemic ulcers (see Wounds and Ulcers, p.1585) after topical use of ketanserin 2%. However, when applied topically to surgical wounds no improvement was found and it was suggested that ketanserin is only of benefit where blood supply is compromised.[6]

1. Tytgat H, van Asch H. Topical ketanserin in the treatment of decubitus ulcers: a double-blind study with 2% ketanserin ointment against placebo. *Adv Therapy* 1988; **5:** 143–52.
2. Roelens P. Double-blind placebo-controlled study with topical 2% ketanserin ointment in the treatment of venous ulcers. *Dermatologica* 1989; **178:** 98–102.
3. Janssen PAJ, *et al.* Use of topical ketanserin in the treatment of skin ulcers: a double-blind study. *J Am Acad Dermatol* 1989; **21:** 85–90.
4. Martinez-de Jesus FR, *et al.* Randomized single-blind trial of topical ketanserin for healing acceleration of diabetic foot ulcers. *Arch Med Res* 1997; **28:** 95–9.
5. Salazar JJ, *et al.* Use of topical ketanserin for the treatment of ulcers in leprosy patients. *Indian J Lepr* 2001; **73:** 103–10.
6. Lawrence CM, *et al.* The effect of ketanserin on healing of fresh surgical wounds. *Br J Dermatol* 1995; **132:** 580–6.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Serefrex†; ***Belg.:*** Sufrexal; ***Ital.:*** Serepress; ***Mex.:*** Sufrexal; ***Neth.:*** Ketensin; ***Port.:*** Sufrexal†; ***Thai.:*** Sufrexal†.

Multi-ingredient: *Mex.:* Sufrexal P.

Labetalol Hydrochloride

(BANM, USAN, rINNM) ⊗

AH-5158A; Hidrocloruro de labetalol; Ibidomide Hydrochloride; Labétalol, chlorhydrate de; Labetalol hydrochlorid; Labetalol-hidroklorid; Labetalolhydroklorid; Labetaloli hydrochloridum; Labetalolihydrokloridi; Labetalolio hidrocloridas; Sch-15719W. 5-[1-Hydroxy-2-(1-methyl-3-phenylpropylamino)ethyl]salicylamide hydrochloride.

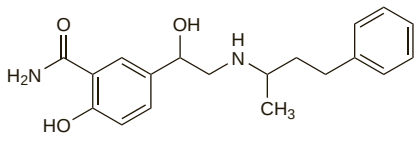
Лабеталола Гидрохлорид

$C_{19}H_{24}N_2O_3,HCl$ = 364.9.

CAS — 36894-69-6 (labetalol); 32780-64-6 (labetalol hydrochloride).

ATC — C07AG01.

ATC Vet — QC07AG01.



(labetalol)

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

Ph. Eur. 6.2 (Labetalol Hydrochloride). A white or almost white powder. Sparingly soluble in water and in alcohol; practically insoluble in dichloromethane. A 1% solution in water has a pH of 4.0 to 5.0.

USP 31 (Labetalol Hydrochloride). A white to off-white powder. Soluble in water and in alcohol; insoluble in chloroform and in ether. A 1% solution in water has a pH of 4.0 to 5.0. Store in airtight containers at a temperature of 25°, excursions permitted between 15° and 30°. Protect from light.

Incompatibility. Labetalol hydrochloride is compatible with standard intravenous solutions such as glucose 5% and sodium chloride 0.9%. However, precipitation has been reported when labetalol hydrochloride is added to sodium bicarbonate injection 5%.[1] The precipitate is probably labetalol base.[2]

Immediate formation of a precipitate has also been reported when labetalol (generally 5 mg/mL in glucose 5%) was mixed with other drugs including ceftriaxone,[3] furosemide,[4] heparin,[5] insulin,[5] proton pump inhibitors such as pantoprazole,[6] and thiopental.[4] There has also been a report of immediate haze after admixture of labetalol hydrochloride (800 micrograms/mL) with warfarin sodium.[7]

1. Yuen P-HC, *et al.* Compatibility and stability of labetalol hydrochloride in commonly used intravenous solutions. *Am J Hosp Pharm* 1983; **40:** 1007–9.
2. Alam AS. Identification of labetalol precipitate. *Am J Hosp Pharm* 1984; **41:** 74.
3. Leader WG, Jones JM. Incompatibility between ceftriaxone sodium and labetalol hydrochloride. *Am J Health-Syst Pharm* 1996; **53:** 2639.
4. Chiu MF, Schwartz ML. Visual compatibility of injectable drugs used in the intensive care unit. *Am J Health-Syst Pharm* 1997; **54:** 64–5.
5. Yamashita SK, *et al.* Compatibility of selected critical care drugs during simulated Y-site administration. *Am J Health-Syst Pharm* 1996; **53:** 1048–51.
6. Péré H, *et al.* Compatibilité du pantoprazole injectable lors d'administration en Y. *Pharmactuel* 2004; **37:** 193–6.
7. Bahal SM, *et al.* Visual compatibility of warfarin sodium injection with selected medications and solutions. *Am J Health-Syst Pharm* 1997; **54:** 2599–2600.

Adverse Effects

The adverse effects associated with beta blockers are described on p.1226. Labetalol also has alpha-blocking activity, which contributes to its adverse effects and these effects may predominate. Orthostatic hypotension may be a problem with high doses or at the start of treatment. Other effects associated with alpha blockade include dizziness, scalp tingling, and nasal congestion. Male sexual function may be impaired to a greater extent than with beta blockade alone. Muscle weakness, tremor, urinary retention, hepatitis, and jaundice have also been reported.

Effects on the liver. By 1990, the FDA had received 11 reports of hepatocellular damage associated with labetalol therapy.[1] Three patients died. Liver function should be monitored and labetalol stopped in patients who develop liver function abnormalities. The *R,R*-isomer of labetalol, dilevalol, was withdrawn from the market because of hepatotoxicity.[2]

1. Clark JA, *et al.* Labetalol hepatotoxicity. *Ann Intern Med* 1990; **113:** 210–13.
2. Harvengt C. Labetalol hepatotoxicity. *Ann Intern Med* 1991; **114:** 341.

Hypersensitivity. Hypersensitivity reactions associated with labetalol may manifest as fever.[1,2] Anaphylactoid reaction to labetalol has also been reported.[3]

1. D'Arcy PF. Drug reactions and interactions: drug fever with labetalol. *Int Pharm J* 1987; **1:** 43–4.
2. Stricker BH, *et al.* Fever induced by labetalol. *JAMA* 1986; **256:** 619–20.
3. Ferree CE. Apparent anaphylaxis from labetalol. *Ann Intern Med* 1986; **104:** 729–30.

Overdosage. Acute oliguric renal failure developed after a short period of moderate hypotension in a patient who ingested labetalol 16 g. Renal function subsequently recovered.[1] Renal failure has also been reported[2] after ingestion of labetalol 6 g. The patient recovered after treatment with glucagon, isoprenaline, and dialysis. Another patient[3] developed circulatory collapse and impaired consciousness after being given labetalol 800 mg orally for hypertensive crisis; glucagon and sympathomimetics were given to restore blood pressure, but amrinone infusion was also needed to improve cardiac output and mental state.

1. Smit AJ, *et al.* Acute renal failure after overdose of labetalol. *BMJ* 1986; **293:** 1142–3.
2. Korzets A, *et al.* Acute renal failure associated with a labetalol overdose. *Postgrad Med J* 1990; **66:** 66–7.
3. Kollef MH. Labetalol overdose successfully treated with amrinone and alpha-adrenergic receptor agonists. *Chest* 1994; **105:** 626–7.

Precautions

As for Beta Blockers, p.1227.

Because labetalol causes orthostatic hypotension it is recommended that injections are given to patients when they are lying down and that patients should remain lying down for the next 3 hours.

Labetalol should be withdrawn from patients who develop signs of hepatic impairment.

Breast feeding. Labetalol is distributed into breast milk, although it has been suggested[1] that the proportion of a maternal dose likely to be ingested by the infant is very low. In a study[2] in 25 patients, the mean concentration of labetalol in breast milk was less than in maternal plasma in patients given doses between 330 and 800 mg daily, although in 1 patient given 1200 mg daily a higher concentration was found in breast milk. In another study,[3] the concentration of drug in milk exceeded maternal plasma concentration in 2 of 3 mothers, and in 1 infant the plasma-labetalol concentration was similar to that of the mother. However, no adverse effects have been seen in breast-feeding infants whose mothers were given labetalol, and the American Academy of Pediatrics considers[4] that it is therefore usually compatible with breast feeding.

1. Atkinson H, Begg EJ. Concentrations of beta-blocking drugs in human milk. *J Pediatr* 1990; **116:** 156.
2. Michael CA. Use of labetalol in the treatment of severe hypertension during pregnancy. *Br J Clin Pharmacol* 1979; **8** (suppl 2): 211S–215S.
3. Lunell NO, *et al.* Transfer of labetalol into amniotic fluid and breast milk in lactating women. *Eur J Clin Pharmacol* 1985; **28:** 597–9.
4. 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)

Interactions

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

Pharmacokinetics

Labetalol is readily absorbed from the gastrointestinal tract, but is subject to considerable first-pass metabolism. Bioavailability varies widely between patients and may be increased in the presence of food. Peak plasma concentrations occur about 1 to 2 hours after an oral dose. Labetalol has low lipid solubility and only very small amounts appear to cross the blood-brain barrier in *animals*. It is about 50% protein bound. Labetalol crosses the placenta and is distributed into breast milk (see above). Labetalol is metabolised mainly in the liver, the metabolites being excreted in the urine with only small amounts of unchanged labetalol; its major metabolite has not been found to have significant alpha- or beta-blocking effects. Excretion also occurs in the faeces via the bile. The elimination half-life at steady state is reported to be about 6 to 8 hours. On intravenous infusion, the elimination half-life is about 5.5 hours. Labetalol is not removed by dialysis.

The elderly. Analysis[1] of data from 4 single-dose studies and 3 multidose studies indicated that age did not appear to be a significant factor in oral clearance in elderly patients receiving labetalol for long-term management of hypertension.

1. Rocci ML, *et al.* Effects of age on the elimination of labetalol. *Clin Pharmacokinet* 1989; **17:** 452–7.

Pregnancy. The concentration of labetalol has been found to be lower in amniotic fluid[1] and fetal plasma[2] than in maternal plasma. A ratio of infant to maternal drug concentration of 0.2 to 0.8 has been reported[2] based on concentration in infant cord blood at delivery [time since last maternal dose not stated]. In another study,[3] however, higher concentrations were found in cord plasma than in maternal plasma at delivery when infants were delivered 12 to 24 hours after the last maternal dose.

The half-life of labetalol was reported as 24 hours in a neonate of 37 weeks' gestation whose mother had received labetalol 600 mg daily for 11 weeks prior to delivery.[4]

1. Lunell NO, *et al.* Transfer of labetalol into amniotic fluid and breast milk in lactating women. *Eur J Clin Pharmacol* 1985; **28:** 597–9.
2. Michael CA. Use of labetalol in the treatment of severe hypertension during pregnancy. *Br J Clin Pharmacol* 1979; **8** (suppl 2): 211S–215S.
3. Boulton DW, *et al.* Transplacental distribution of labetalol stereoisomers at delivery. *Br J Clin Pharmacol* 1999; **47:** 573–4.
4. Haraldsson A, Geven W. Half-life of maternal labetalol in a premature infant. *Pharm Weekbl (Sci)* 1989; **11:** 229–31.

Uses and Administration

Labetalol is a non-cardioselective beta blocker (p.1225). It is reported to possess some intrinsic sympathomimetic and membrane-stabilising activity. In addition, it has selective $alpha_1$-blocking properties which decrease peripheral vascular resistance. The ratio of alpha- to beta-blocking activity has been estimated to be about 1:3 after oral doses and 1:7 after intravenous doses.

Labetalol is used as the hydrochloride in the management of hypertension (p.1171). It is also used to induce hypotension during surgery. Labetalol decreases blood pressure more rapidly than other beta blockers; the full antihypertensive effect may be seen within 1 to 3 hours of an oral dose.

In **hypertension** labetalol hydrochloride is usually given in an initial oral dose of 100 mg twice daily with food, gradually increased if necessary according to response and standing blood pressure, to 200 to 400 mg twice daily; total daily doses of 2.4 g, in two to four divided doses, have occasionally been required. Lower doses may be adequate in elderly patients; an initial dose of 50 to 100 mg twice daily has been recommended, and the usual maintenance dose is 100 to 200 mg twice daily.

For the emergency treatment of hypertension labetalol hydrochloride may be given by slow intravenous injection. In the UK a dose of 50 mg is recommended, given over a period of at least 1 minute; if necessary this dose may be repeated at intervals of 5 minutes until a total of 200 mg has been given. In the USA an initial dose of 20 mg is recommended, given over 2 minutes; subsequent doses of 40 to 80 mg may be given every 10 minutes, if necessary, up to a maximum of 300 mg. Blood pressure should be monitored, and the patient should remain supine during the injection and for 3 hours afterwards, to avoid excessive orthostatic hypotension. After bolus intravenous injection a maximum effect is usually obtained within 5 minutes and usually lasts up to 6 hours, although it may extend as long as 18 hours.

Labetalol hydrochloride has also been given by intravenous infusion in usual doses of 2 mg/minute. Suggested concentrations for intravenous infusions are 1 mg/mL or 2 mg/3 mL of suitable diluent. In hypertension in pregnancy, labetalol infusion may be started at the rate of 20 mg/hour, then doubled every 30 minutes until a satisfactory response is obtained or a dose of 160 mg/hour is reached. In hypertension after myocardial infarction, labetalol infusion may be started at the rate of 15 mg/hour and gradually increased until a satisfactory response is obtained or a dose of 120 mg/hour is reached.

The initial dose in **hypotensive anaesthesia** is 10 to 20 mg intravenously, with increments of 5 to 10 mg if satisfactory hypotension is not achieved after 5 minutes. A higher initial dose may be required in patients who do not receive halothane anaesthesia.

For the use of labetalol in children, see below.

Action. Labetalol has 2 optical centres; it is used as the racemic mixture of the 4 stereoisomers. The *R,R*- isomer is responsible for the beta-blocking activity and has limited alpha-blocking activity; it also has beta-adrenergic mediated peripheral vasodilating activity. The *S,R*-isomer has the most potent alpha-blocking activity. The *S,S*-isomer has some alpha-blocking activity and the *R,S*-isomer does not appear to have either alpha- or beta-adrenergic blocking effect.[1] The pure *R,R*-isomer, dilevalol, was withdrawn from the market because of hepatotoxicity.

1. Gold EH, *et al*. Synthesis and comparison of some cardiovascular properties of the stereoisomers of labetalol. *J Med Chem* 1982; **25:** 1363–70.

Administration in children. Labetalol has been used in the management of hypertension in children,[1] although experience is limited. The *BNFC* suggests the following doses:

for **hypertensive emergencies**, labetalol hydrochloride may be given by intravenous infusion as follows:

- neonates: 500 micrograms/kg per hour adjusted at intervals of at least 15 minutes according to response, to a maximum of 4 mg/kg per hour
- 1 month to 12 years: 0.5 to 1 mg/kg per hour adjusted at intervals of at least 15 minutes according to response, to a maximum of 3 mg/kg per hour
- 12 to 18 years: 30 to 120 mg/hour adjusted at intervals of at least 15 minutes according to response

for **hypertension**, labetalol hydrochloride may be given as follows:

- 1 month to 12 years: 1 to 2 mg/kg three or four times daily by mouth or a single intravenous injection in a dose of 250 to 500 micrograms/kg to a maximum of 20 mg
- 12 to 18 years: similar doses to adults (see above) although a lower initial oral dose of 50 to 100 mg twice daily is recommended

1. Bunchman TE, *et al*. Intravenously administered labetalol for treatment of hypertension in children. *J Pediatr* 1992; **120:** 140–4.

Preparations

BP 2008: Labetalol Injection; Labetalol Tablets;
USP 31: Labetalol Hydrochloride Injection; Labetalol Hydrochloride Oral Suspension; Labetalol Hydrochloride Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Biascor; ***Austral.:*** Presolol; Trandate; ***Austria:*** Trandate; ***Belg.:*** Trandate; ***Canad.:*** Trandate; ***Chile:*** Trandate; ***Cz.:*** Coreton†; Trandate; ***Denm.:*** Trandate; ***Fin.:*** Albetol; ***Fr.:*** Trandate; ***Gr.:*** Trandate; ***Hong Kong:*** Trandate; ***Irl.:*** Trandate; ***Israel:*** Trandate; ***Ital.:*** Ipolab; Trandate; ***Malaysia:*** Tolbetol†; Trandate; ***Neth.:*** Trandate; ***Norw.:*** Trandate; ***NZ:*** Hybloc; Trandate; ***Port.:*** Trandate†; ***S.Afr.:*** Trandate; ***Singapore:*** Trandate; ***Spain:*** Trandate; ***Swed.:*** Trandate; ***Switz.:*** Trandate; ***UK:*** Trandate; ***USA:*** Normodyne†; Trandate; ***Venez.:*** Trandate†.

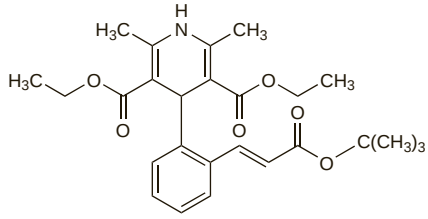
Multi-ingredient: ***Ital.:*** Trandiur.

Lacidipine *(BAN, USAN, rINN)*

GR-43659X; GX-1048; Lacidipin; Lacidipino; Lacidipinum; Lasidipiini; Lasidipin. Diethyl 4-{2-[(*tert*-butoxycarbonyl)vinyl]phenyl}-1,4-dihydro-2,6-dimethylpyridine-3,5-dicarboxylate.

Лацидипин

$C_{26}H_{33}NO_6$ = 455.5.
CAS — 103890-78-4.
ATC — C08CA09.
ATC Vet — QC08CA09.



Pharmacopoeias. In *Br.*
BP 2008 (Lacidipine). A white to pale yellow crystalline powder. Practically insoluble in water; sparingly soluble in dehydrated alcohol; freely soluble in acetone and in dichloromethane.

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

Lacidipine is rapidly but poorly absorbed from the gastrointestinal tract after oral doses and undergoes extensive first-pass metabolism; the bioavailability has been reported to be 2 to 9%, or 18.5% (range 4 to 52%) using a more sensitive assay method. It is more than 95% bound to plasma proteins. Lacidipine is eliminated by metabolism in the liver and metabolites are excreted mainly by the biliary route. About 70% of an oral dose is eliminated in the faeces, the remainder in the urine. The average steady-state terminal elimination half-life of lacidipine is 13 to 19 hours.

Uses and Administration

Lacidipine is a dihydropyridine calcium-channel blocker with actions similar to those of nifedipine (p.1354). It is used in the treatment of hypertension (p.1171).

The usual initial dose of lacidipine is 2 mg once daily by mouth increased if necessary after 3 to 4 weeks or more to 4 mg daily; a further increase in dose to 6 mg daily may be necessary in some patients.

◊ Reviews.

1. Lee CR, Bryson HM. Lacidipine: a review of its pharmacodynamic and pharmacokinetic properties and therapeutic potential in the treatment of hypertension. *Drugs* 1994; **48:** 274–96.
2. Zanchetti A, ed. Cardiovascular advantages of a third generation calcium antagonist: symposium on lacidipine. *Drugs* 1999; **57** (suppl 1): 1–29.
3. McCormack PL, Wagstaff AJ. Lacidipine: a review of its use in the management of hypertension. *Drugs* 2003; **63:** 2327–56.

Preparations

BP 2008: Lacidipine Tablets.

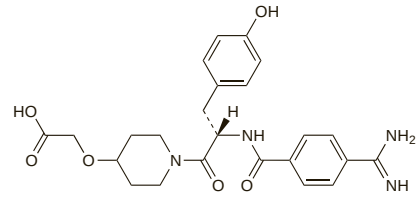
Proprietary Preparations (details are given in Part 3)
Arg.: Lacipil†; Midotens†; ***Belg.:*** Motens; ***Braz.:*** Lacipil; Midotens; ***Cz.:*** Lacipil; ***Denm.:*** Midotens; ***Fr.:*** Caldine; ***Ger.:*** Motens; ***Gr.:*** Balnox; Lacipil; Lacitens; Motens; ***Hong Kong:*** Lacipil; ***Hung.:*** Lacipil; ***India:*** Sinopil; ***Indon.:*** Lacipil; ***Ital.:*** Aponil; Lacipil; Lacirex; Ladip; Viapres; ***Malaysia:*** Lacipil; ***Mex.:*** Lacipil; Midotens; ***Neth.:*** Motens; ***Philipp.:*** Lacipil; ***Pol.:*** Lacipil; ***Port.:*** Lacipil; Tens; ***Rus.:*** Lacipil (Лаципил); ***Singapore:*** Lacipil; ***Spain:*** Lacimen; Lacipil; Motens; ***Switz.:*** Motens; ***Thai.:*** Motens; ***Turk.:*** Lacipil; ***UK:*** Motens; ***Venez.:*** Lacipil; Tens†.

Lamifiban *(USAN, rINN)*

Lamifibán; Lamifibanum; Ro-44-9883; Ro-44-9883/000. ({1-[*N*-(*p*-Amidinobenzoyl)-L-tyrosyl]-4-piperidyl}oxy)acetic acid.

Ламифибан

$C_{24}H_{28}N_4O_6$ = 468.5.
CAS — 144412-49-7 (lamifiban); 243835-65-6 (lamifiban hydrochloride).



Profile

Lamifiban is a glycoprotein IIb/IIIa-receptor antagonist. It has been investigated as an antiplatelet drug given intravenously for the management of thromboembolic disorders, such as unstable angina and myocardial infarction.

◊ References.

1. Théroux P, *et al*. Platelet membrane receptor glycoprotein IIb/IIIa antagonism in unstable angina: the Canadian Lamifiban Study. *Circulation* 1996; **94:** 899–905.
2. The PARAGON Investigators. International, randomized, controlled trial of lamifiban (a platelet glycoprotein IIb/IIIa inhibitor), heparin, or both in unstable angina. *Circulation* 1998; **97:** 2386–95.
3. The PARADIGM Investigators. Combining thrombolysis with the platelet glycoprotein IIb/IIIa inhibitor lamifiban: results of the Platelet Aggregation Receptor Antagonist Dose Investigation and Reperfusion Gain in Myocardial Infarction (PARADIGM) trial. *J Am Coll Cardiol* 1998; **32:** 2003–10.
4. Global Organization Network (PARAGON)-B Investigators. Randomized, placebo-controlled trial of titrated intravenous lamifiban for acute coronary syndromes. *Circulation* 2002; **105:** 316–21.

Lanatoside C *(BAN, rINN)*

Celanide; Celanidum; Lanatosid C; Lanatosidi C; Lanatósido C; Lanatosidum C; Lanatozyd c. 3-[(*O*-β-D-Glucopyranosyl-(1→4)-*O*-3-acetyl-2,6-dideoxy-β-D-*ribo*-hexopyranosyl-(1→4)-*O*-2,6-dideoxy-β-D-*ribo*-hexopyranosyl-(1→4)-*O*-2,6-dideoxy-β-D-*ribo*-hexopyranosyl)oxy]-12,14-dihydroxy-3β,5β,12β-card-20(22)-enolide.

Ланатозид С

$C_{49}H_{76}O_{20}$ = 985.1.
CAS — 17575-22-3.
ATC — C01AA06.
ATC Vet — QC01AA06.