Deserpidine *(BAN, rINN)*

Canescine; Deserpidiini; Deserpidin; Deserpidina; Déserpidine; Deserpidinum; 11-Desmethoxyreserpine; Raunormine; Recanescine. Methyl 11-demethoxy-*O*-(3,4,5-trimethoxybenzoyl)reserpate.

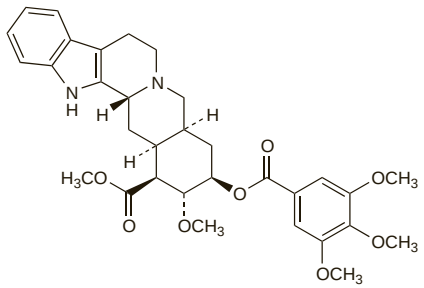
Дезерпидин

$C_{32}H_{38}N_2O_8$ = 578.7.

CAS — 131-01-1.

ATC — C02AA05.

ATC Vet — QC02AA05.



Profile

Deserpidine is an ester alkaloid isolated from the root of *Rauwolfia canescens*. It has properties similar to those described under reserpine (p.1387) and has been used in the treatment of hypertension and psychoses.

Preparations

Proprietary Preparations (details are given in Part 3)
Multi-ingredient: ***Hong Kong:*** Enduronyl†.

Desirudin *(BAN, USAN, rINN)*

CGP-39393; Desirudiini; Desirudina; Désirudine; Desirudinum; Desulphatohirudin. 63-Desulfohirudin (*Hirudo medicinalis* isoform HV1).

Дезирудин

$C_{287}H_{440}N_{80}O_{110}S_6$ = 6963.4.

CAS — 120993-53-5.

ATC — B01AE01.

ATC Vet — QB01AE01.

Adverse Effects and Precautions

As for Lepirudin, p.1323.

Teratogenicity has been observed in *animals*.

Interactions

As for Lepirudin, p.1323.

Pharmacokinetics

Maximum plasma concentrations of desirudin are reached 1 to 3 hours after subcutaneous injection. Desirudin is metabolised and excreted by the kidney, and 40 to 50% of a dose is excreted unchanged in the urine. After subcutaneous or intravenous injection the terminal elimination half-life of desirudin is 2 to 3 hours.

◊ References.

1. Lefèvre G, *et al.* Effect of renal impairment on the pharmacokinetics and pharmacodynamics of desirudin. *Clin Pharmacol Ther* 1997; **62:** 50–9.

Uses and Administration

Desirudin is a recombinant hirudin (p.1305) that is a direct inhibitor of thrombin with actions similar to Lepirudin, p.1323. It is used as an anticoagulant for the prevention of postoperative venous thromboembolism (p.1189) in patients undergoing orthopaedic surgery. It has been investigated in arterial thromboembolic disorders such as myocardial infarction and unstable angina, and as an adjunct in angioplasty procedures (see Ischaemic Heart Disease, under Uses and Administration of Lepirudin, p.1323).

In the prevention of venous thromboembolism, desirudin is given subcutaneously in a dose of 15 mg twice daily, the first dose 5 to 15 minutes before surgery, but after induction of regional block anaesthesia, if used. Treatment is continued until the patient is fully ambulant, usually for 9 to a maximum of 12 days.

Response to desirudin should be monitored using activated partial thromboplastin time (APTT) in patients with hepatic or renal impairment, or increased risk of bleeding. Doses may need to be reduced in patients with renal impairment (see below).

◊ References.

1. Matheson AJ, Goa KL. Desirudin: a review of its use in the management of thrombotic disorders. *Drugs* 2000; **60:** 679–700.

Administration in renal impairment. The dose of desirudin should be reduced in patients with renal impairment, depending on creatinine clearance (CC) and activated partial thromboplastin time (APPT), which should be measured daily. US licensed product information recommends the following doses:

- CC 31 to 60 mL/minute per 1.73 m^2, initial dose 5 mg every 12 hours, subsequently adjusted according to APPT
- CC below 31 mL/minute per 1.73 m^2, initial dose 1.7 mg every 12 hours, subsequently adjusted according to APPT

Preparations

Proprietary Preparations (details are given in Part 3)
Austral.: Revasc; ***Cz.:*** Revasc; ***Fr.:*** Revasc†; ***Ger.:*** Revasc; ***Gr.:*** Revasc†; ***Hung.:*** Revasc†; ***Neth.:*** Revasc; ***Norw.:*** Revasc†; ***NZ:*** Revasc; ***Port.:*** Revasc; ***Spain:*** Revasc; ***Switz.:*** Revasc†; ***USA:*** Iprivask.

Deslanoside *(BAN, rINN)*

Deacetyl-lanatoside C; Desacetyl-lanatoside C; Deslanosid; Deslanosídeo; Deslanosidi; Deslanósido; Deslanosidum; Deslanozidas; Dezlanozid. 3-[(*O*-β-D-Glucopyranosyl-(1→4)-*O*-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.

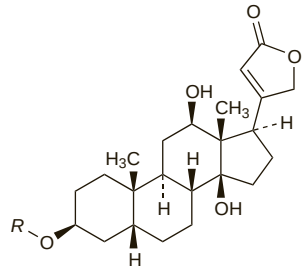
Дезланозид

$C_{47}H_{74}O_{19}$ = 943.1.

CAS — 17598-65-1.

ATC — C01AA07.

ATC Vet — QC01AA07.



R = β-D-glucose–(β-D-digitoxose)$_3$

Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), *Jpn*, and *US*.
Ph. Eur. 6.2 (Deslanoside). A white or almost white, crystalline or finely crystalline hygroscopic powder. Practically insoluble in water; very slightly soluble in alcohol. In an atmosphere of low relative humidity, it loses water. Store in airtight, glass containers at a temperature below 10°. Protect from light.
USP 31 (Deslanoside). Store in airtight containers at a temperature of 25°, excursions permitted between 15° and 30°. Protect from light.

Profile

Deslanoside, a cardiac glycoside with positive inotropic activity, is a derivative of lanatoside C. It has the general properties of digoxin (p.1259) and has been used similarly in the management of some cardiac arrhythmias and in heart failure.

Preparations

USP 31: Deslanoside Injection.

Proprietary Preparations (details are given in Part 3)
Braz.: Cedilanide.

Detajmium Bitartrate *(rINN)*

Bitartrato de detajmio; Detajmii Bitartras; Détajmium, Bitartrate de. 4-[3-(Diethylamino)-2-hydroxypropyl]ajmalinium hydrogen tartrate monohydrate.

Детаймия Битартрат

$C_{31}H_{47}N_3O_9,H_2O$ = 623.7.

CAS — 53862-81-0.

Profile

Detajmium is a class I antiarrhythmic (p.1153). It is given orally as the bitartrate, in the treatment of supraventricular and ventricular arrhythmias (p.1160). The dose range is from 75 to 300 mg daily depending upon the arrhythmia.

Preparations

Proprietary Preparations (details are given in Part 3)
Cz.: Tachmalcor; ***Ger.:*** Tachmalcor.

Diazoxide *(BAN, USAN, rINN)*

Diatsoksidi; Diazoksidas; Diazoksit; Diazoxid; Diazóxido; Diazoxidum; NSC-64198; Sch-6783; SRG-95213. 7-Chloro-3-methyl-2*H*-1,2,4-benzothiadiazine 1,1-dioxide.

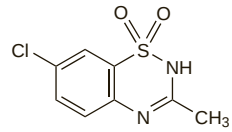
Диазоксид

$C_8H_7ClN_2O_2S$ = 230.7.

CAS — 364-98-7.

ATC — C02DA01; V03AH01.

ATC Vet — QC02DA01; QV03AH01.



Pharmacopoeias. In *Eur.* (see p.vii), *Int.*, and *US*.
Ph. Eur. 6.2 (Diazoxide). A white or almost white, fine or crystalline powder. Practically insoluble in water; slightly soluble in alcohol; freely soluble in dimethylformamide; very soluble in dilute solutions of alkali hydroxides.
USP 31 (Diazoxide). White or cream-white crystals or crystalline powder. Practically insoluble to sparingly soluble in water and in most organic solvents; freely soluble in dimethylformamide; very soluble in strong alkaline solutions. Store at a temperature of 25°, excursions permitted between 15° and 30°.

Adverse Effects

In addition to inappropriate hypotension and hyperglycaemia (which includes ketoacidosis and hyperosmolar nonketotic coma), adverse effects often include oedema due to salt and water retention, which may precipitate heart failure. Other adverse effects include: dysgeusia, nausea, anorexia, and other gastrointestinal disturbances; mild hyperuricaemia; extrapyramidal symptoms; eosinophilia and thrombocytopenia; dyspnoea; hypertrichosis; and headache, dizziness, tinnitus, and blurred vision. Hypersensitivity has occurred, manifesting as rashes, leucopenia, and fever.

During intravenous therapy, particularly after large bolus injections, adverse effects may be associated with too rapid a reduction in blood pressure and include: coronary ischaemia leading to angina, cardiac arrhythmias, marked ECG changes, tachycardia, palpitations, and bradycardia; cerebral ischaemia leading to confusion, convulsions, loss of consciousness, and neurological deficit; renal impairment; and symptoms of vasodilatation.

Diazoxide may cause a burning sensation in the injected vein; extravasation of the alkaline solution is painful.

Effects on the blood. A 26-year-old man with hypertension developed reversible haemolytic anaemia when treated with diazoxide orally on 3 separate occasions.[1]

1. Best RA, Clink HM. Haemolysis associated with diazoxide, used for the control of hypertension. *Postgrad Med J* 1975; **51:** 402–4.

Effects on the hair. *Hirsutism* and *hypertrichosis* are different types of excessive hair growth, but the terms have often been used interchangeably. Hirsutism is androgen-related whereas hypertrichosis is thought to be independent of hormone stimulation. Hypertrichosis is acknowledged to be a frequent adverse effect of diazoxide in children receiving long-term treatment for idiopathic hypoglycaemia.[1] Two such children had unusually deep (low-pitched) voices as well as marked hypertrichosis.[2] A woman on continuous diazoxide therapy who developed so-called hirsutism without signs of virilisation had raised serum concentrations of androgens.[3]

Alopecia has been reported[4] in 4 infants born to mothers who had been on long-term treatment with diazoxide during pregnancy; the condition was still present to some extent when the infants were last observed at the ages of 5 months to 1 year.

1. Burton JL, *et al.* Hypertrichosis due to diazoxide. *Br J Dermatol* 1975; **93:** 707–11.
2. West RJ. Side effects of diazoxide. *BMJ* 1978; **2:** 506.
3. Hallengren B, Hökfelt B. Increase of serum androgens during diazoxide treatment. *Lancet* 1984; **ii:** 1044–5.
4. Milner RDG, Chouksey SK. Effects of fetal exposure to diazoxide in man. *Arch Dis Child* 1972; **47:** 537–43.