### Adverse Effects and Precautions

As for Sumatriptan, p.625.

Almotriptan should not be used in patients with severe hepatic impairment since clearance is likely to be markedly impaired, and should be given with caution, and in reduced doses, to patients with mild to moderate hepatic impairment. The dose of almotriptan should also be reduced in patients with severe renal impairment.

Patients with hypersensitivity to sulfonamides may theoretically exhibit a similar reaction to almotriptan.

**Incidence of adverse effects.** Results from studies involving more than 2500 patients with migraine suggested that adverse effects of almotriptan were infrequent.[1] The commonest adverse effects reported were dizziness, nausea and vomiting, headache, paraesthesia, fatigue, and drowsiness, all of which occurred in less than 3% of patients. The incidence of chest symptoms was 0.2% in 2 large phase III studies.

1. Dodick DW. Oral almotriptan in the treatment of migraine: safety and tolerability. *Headache* 2001; **41:** 449–55.

### Interactions

As for Sumatriptan, p.626.

### Pharmacokinetics

After oral doses, peak plasma-almotriptan concentrations are obtained in about 1 to 3 hours, with a bioavailability of about 70%. Protein binding is about 35%. Almotriptan is metabolised, mainly by monoamine oxidase type A to the inactive indole acetic acid derivative and to a lesser extent by cytochrome P450 isoenzymes CYP3A4 and CYP2D6 to the inactive gamma-aminobutyric acid derivative. More than 75% of an oral dose is excreted in the urine and the remainder in faeces. About 40 to 50% of the dose in the urine and 5% in the faeces is excreted as unchanged drug. The plasma elimination half-life is about 3.5 hours in healthy subjects, increasing to about 7 hours in severe renal impairment.

Distribution into milk has been found in studies in *rats*.

◊ References.

1. Jansat JM, *et al.* Absolute bioavailability, pharmacokinetics, and urinary excretion of the novel antimigraine agent almotriptan in healthy male volunteers. *J Clin Pharmacol* 2002; **42:** 1303–10.
2. McEnroe JD, Fleishaker JC. Clinical pharmacokinetics of almotriptan, a serotonin 5-HT receptor agonist for the treatment of migraine. *Clin Pharmacokinet* 2005; **44:** 237–46.

### Uses and Administration

Almotriptan malate is a selective serotonin ($5\text{-}HT_1$) agonist with actions and uses similar to those of sumatriptan (p.627). It is used for the acute treatment of the headache phase of migraine attacks. It should not be used for prophylaxis. Almotriptan is given orally as the malate, and doses are expressed in terms of the base; almotriptan malate 8.75 mg is equivalent to about 6.25 mg of almotriptan.

The usual dose of almotriptan is 12.5 mg in the UK and 6.25 or 12.5 mg in the USA. If this is ineffective, a second dose should not be taken for the same attack. If symptoms recur within 24 hours after an initial response, a second dose may be taken after an interval of at least 2 hours. No more than 2 doses should be taken in a 24-hour period. For doses in hepatic and renal impairment see below.

◊ References.

1. Holm KJ, Spencer CM. Almotriptan. *CNS Drugs* 1999; **11:** 159–64.
2. Keam SJ, *et al.* Almotriptan: a review of its use in migraine. *Drugs* 2002; **62:** 387–414.

**Administration in hepatic or renal impairment.** In patients with hepatic or severe renal impairment, no more than 12.5 mg of almotriptan should be taken in 24 hours; a starting dose of 6.25 mg may be used. Almotriptan is contra-indicated in patients with severe hepatic disease.

**Migraine.** For comparison of the relative benefits of different triptans in migraine, see under Sumatriptan, p.627.

Further references.

1. Balbisi EA. Efficacy and safety of almotriptan malate for migraine. *Am J Health-Syst Pharm* 2002; **59:** 2184–93.
2. Dodick DW. A review of the clinical efficacy and tolerability of almotriptan in acute migraine. *Expert Opin Pharmacother* 2003; **4:** 1157–63.
3. Dowson AJ. Oral almotriptan: practical uses in the acute treatment of migraine. *Expert Rev Neurother* 2004; **4:** 339–48.
4. Mathew NT. Efficacy and tolerability of almotriptan in controlled clinical trials. *Eur Neurol* 2005; **53** (suppl 1): 29–33.
5. Pascual J. Efficacy and tolerability of almotriptan in postmarketing surveillance studies. *Eur Neurol* 2005; **53** (suppl 1): 34–40.
6. Dahlof CG, *et al.* Efficacy, speed of action and tolerability of almotriptan in the acute treatment of migraine: pooled individual patient data from four randomized, double-blind, placebo-controlled clinical trials. *Cephalalgia* 2006; **26:** 400–8.
7. Diener H-C. A review of recent clinical experience with almotriptan. *Drugs* 2006; **66** (suppl 3): 17–25.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
***Belg.:*** Almogran; ***Canad.:*** Axert; ***Denm.:*** Almogran; ***Fin.:*** Almogran; ***Fr.:*** Almogran; ***Ger.:*** Almogran; ***Irl.:*** Almogran; ***Ital.:*** Almogran; Almotrex; ***Neth.:*** Almogran; ***Norw.:*** Almogran; ***Port.:*** Almogran; Amignul; ***Spain:*** Almogran; Amignul; ***Swed.:*** Almogran; ***UK:*** Almogran; ***USA:*** Axert.

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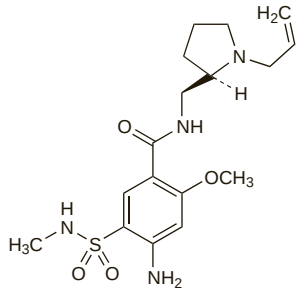
### Alpiropride *(rINN)*

Alpiroprida; Alpiropridum. (±)-*N*-[(1-Allyl-2-pyrrolidinyl)methyl]-4-amino-5-(methylsulfamoyl)-*o*-anisamide.

Альпироприд

$C_{17}H_{26}N_4O_4S$ = 382.5.
*CAS — 81982-32-3.*



### Profile

Alpiropride is a dopamine antagonist that has been given orally for the treatment and prophylaxis of migraine.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
***Port.:*** Rivistel†.

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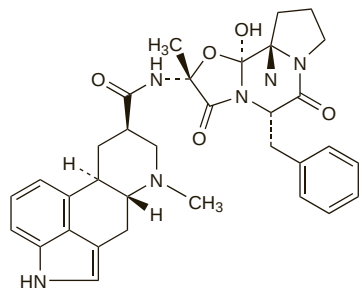
## Dihydroergotamine *(BAN, rINN)*

Dihidroergotamina; Dihydroergotamiini; Dihydroergotamin; Dihydroergotaminum. (5′*S*,8*R*)-5′-Benzyl-9,10-dihydro-12′-hydroxy-2′-methyl-3′,6′,18-trioxoergotaman.

Дигидроэрготамин

$C_{33}H_{37}N_5O_5$ = 583.7.
*CAS — 511-12-6.*
*ATC — N02CA01.*
*ATC Vet — QN02CA01.*



### Dihydroergotamine Mesilate *(BANM, rINNM)*

Dihidroergotamin-mezilát; Dihidroergotamino mesilatas; Dihydroergotamiinimesilaatti; Dihydroergotamine, mésilate de; Dihydroergotamine Mesylate *(USAN)*; Dihydroergotamine Methanesulphonate; Dihydroergotamini mesilas; Dihydroergotamin-mesilat; Dihydroergotamin-mesylát; Dihydroergotaminy mezylan; Mesilato de dihidroergotamina.

Дигидроэрготамина Мезилат

$C_{33}H_{37}N_5O_5,CH_4O_3S$ = 679.8.
*CAS — 6190-39-2.*
*ATC — N02CA01.*
*ATC Vet — QN02CA01.*

**Pharmacopoeias.** In *Eur.* (see p.vii), *Jpn*, and *US*.
**Ph. Eur. 6.2** (Dihydroergotamine Mesilate). Colourless crystals or a white or almost white crystalline powder. Slightly soluble in water and in alcohol; sparingly soluble in methyl alcohol. A 0.1% solution in water has a pH of 4.4 to 5.4. Protect from light.
**USP 31** (Dihydroergotamine Mesylate). A white to slightly yellowish powder, or off-white to faintly red powder, having a faint odour. Soluble 1 in 125 of water, 1 in 90 of alcohol, 1 in 175 of chloroform, and 1 in 2600 of ether. pH of a 0.1% solution in water is between 4.4 and 5.4. Protect from light.

### Dihydroergotamine Tartrate *(BANM, rINNM)*

Dihidroergotamino tartratas; Dihidroergotamin-tartarát; Dihydroergotamiinitartraatti; Dihydroergotamine, tartrate de; Dihydroergotamini tartras; Dihydroergotamin-tartarát; Dihydroergotamintartrat; Tartrato de dihidroergotamina.

Дигидроэрготамина Тартрат

$(C_{33}H_{37}N_5O_5)_2,C_4H_6O_6$ = 1317.4.
*CAS — 5989-77-5.*
*ATC — N02CA01.*
*ATC Vet — QN02CA01.*

**Pharmacopoeias.** In *Eur.* (see p.vii).
**Ph. Eur. 6.2** (Dihydroergotamine Tartrate). Colourless crystals or a white or almost white crystalline powder. Very slightly soluble in water; sparingly soluble in alcohol. A 0.1% suspension in water has a pH of 4.0 to 5.5. Protect from light.

### Adverse Effects and Treatment

As for Ergotamine Tartrate, p.620, although vasoconstriction may be less pronounced and the frequency of nausea and vomiting lower with dihydroergotamine mesilate than with ergotamine tartrate. Dihydroergotamine does not appear to produce physical dependence.

**Effects on the cardiovascular system.** There are conflicting reports on the risk of vasospasm in patients given dihydroergotamine with heparin for thromboembolism prophylaxis. Vasospastic or necrotic reactions have been reported on several occasions during such therapy.[1-4] In an Austrian study of 147 290 patients given drug prophylaxis for thromboembolism, complications attributable to ergotism were seen in 142 of 61 092 (0.23%) who received dihydroergotamine and heparin.[5] Others,[6] however, observed only 1 case of vasospasm in 5100 trauma patients (0.02%) given the combination. In 1989 the Swedish Adverse Drug Reactions Advisory Committee reported[7] that up to the end of September 1987 the manufacturer had received 201 reports of vasospastic reactions associated with the use of *Orstanorm* (dihydroergotamine + lidocaine) with heparin. Permanent damage occurred in 59% of these patients. Vasospastic reactions had occurred more frequently in patients who had undergone surgery for trauma and the prognosis for such patients was generally poorer than for others. Since the risk of permanent damage appeared to be related to treatment length the Committee recommended that this preparation should not be given for more than 7 days. The possibility of such reactions and the contra-indications of dihydroergotamine should be borne in mind when using this form of prophylaxis (see Venous Thromboembolism, under Uses, below).

1. van den Berg E, *et al.* Ergotism leading to threatened limb amputation or to death in two patients given heparin-dihydroergotamine prophylaxis. *Lancet* 1982; **i:** 955–6.
2. van den Berg E, *et al.* Vascular spasm during thromboembolism prophylaxis with heparin-dihydroergotamine. *Lancet* 1982; **ii:** 268–9.
3. Monreal M, *et al.* Skin and muscle necrosis during heparin-dihydroergotamine prophylaxis. *Lancet* 1984; **ii:** 820.
4. Kilroy RA, *et al.* Vascular spasm during heparin-dihydroergotamine prophylaxis. *Clin Pharm* 1987; **6:** 575–7.
5. Gatterer R. Ergotism as complication of thromboembolic prophylaxis with heparin and dihydroergotamine. *Lancet* 1986; **ii:** 638–9.
6. Schlag G, *et al.* Risk/benefit of heparin-dihydroergotamine thromboembolic prophylaxis. *Lancet* 1986; **ii:** 1465.
7. Swedish Adverse Drug Reaction Advisory Committee. Dihydroergotamine + lidocaine – vasospasm. *Bull Swed Adverse Drug React Advisory Committee* 1989; (54): 1.

**Fibrosis.** For reference to fibrosis associated with the administration of dihydroergotamine, see Methysergide Maleate, p.623.

### Precautions

As for Ergotamine Tartrate, p.620.

**Cardiovascular disorders.** For specific contra-indications and precautions in cardiovascular disorders, see under Ergotamine, p.621.

**Porphyria.** Dihydroergotamine has been associated with acute attacks of porphyria and is considered unsafe in porphyric patients.

### Interactions

As for Ergotamine (p.621).

Use with other vasoconstrictive drugs, including supplementary antimigraine treatment with ergotamine or sumatriptan, should be avoided.

### Pharmacokinetics

Peak plasma-dihydroergotamine concentrations have been attained within about 1 to 2 hours after oral doses, about 30 minutes after intramuscular injection, about 15 to 45 minutes after subcutaneous injection, and about 45 to 55 minutes after intranasal doses. However, the bioavailability of dihydroergotamine after oral doses is very low; values ranging from less than 0.1 to 1.5% have been reported. Although dihydroergotamine is incompletely absorbed from the gastrointestinal tract, the low bioavailability is considered to be determined primarily by extensive first-pass hepatic metabolism. Bioavailability after intranasal doses is 43%. Dihydroergotamine is 90 to 95% bound to plasma proteins.

Dihydroergotamine undergoes extensive metabolism, the major metabolite, 8′-β-hydroxydihydroergotamine, being active. Plasma concentrations of this metabolite are greater than those of dihydroergotamine. A further oxidation step produces 8′,10′-dihydroxydihydroergotamine, which is also active. Other metabolites are also formed. Most of a dose is excreted as metabolites, mainly in the bile; 5 to 10% is excreted in the urine of which only trace amounts are of unchanged drug. The elimination of dihydroergotamine is biphasic; half-lives of about 1 to 2 hours and 22 to 32 hours have been reported for the 2 phases, respectively.

◊ References.


1. Little PJ, *et al.* Bioavailability of dihydroergotamine in man. *Br J Clin Pharmacol* 1982; **13:** 785–90.
2. Müller-Schweinitzer E. Pharmacological actions of the main metabolites of dihydroergotamine. *Eur J Clin Pharmacol* 1984; **26:** 699–705.
3. de Marées H, *et al.* Relationship between the venoconstrictor activity of dihydroergotamine and its pharmacokinetics during acute and chronic oral dosing. *Eur J Clin Pharmacol* 1986; **30:** 685–9.
4. Humbert H, *et al.* Human pharmacokinetics of dihydroergotamine administered by nasal spray. *Clin Pharmacol Ther* 1996; **60:** 265–75.


### Uses and Administration

Dihydroergotamine is a semisynthetic ergot alkaloid that has weaker oxytocic and vasoconstrictor effects than ergotamine (p.621). Its activity as a 5-$HT_1$ agonist is believed to contribute to its antimigraine action. It is used in the treatment of migraine and cluster headache, and in the treatment of orthostatic hypotension. It has also been used for the prophylaxis of venous thromboembolism (see below).

Dihydroergotamine is commonly used as the mesilate by subcutaneous, intramuscular, or intravenous injection, although it may also be given as a nasal spray or orally.

For the treatment of migraine and to terminate an acute attack of cluster headache, dihydroergotamine mesilate is usually given by subcutaneous or intramuscular injection in doses of 1 mg repeated, if necessary, after 30 to 60 minutes up to a maximum daily dose of 3 mg. If a more rapid effect is desired it may be given intravenously in doses of 0.5 or 1 mg up to a maximum daily dose of 2 mg. The total weekly dose given by any route of injection should not exceed 6 mg. The usual nasal dose of dihydroergotamine mesilate for an acute attack of migraine is 500 micrograms sprayed into each nostril as a 0.4% solution followed after 15 minutes by an additional 500 micrograms in each nostril. A total intranasal dose of 2 mg per attack should not be exceeded. In the USA, the maximum dose in 24 hours is 3 mg and in a 7-day period is 4 mg, while maximum daily doses of up to 4 mg with a maximum dose of 12 mg in a 7-day period have been given in other countries. In some countries it is given orally; up to 10 mg daily has been given orally for the treatment of acute attacks of migraine. Lower oral doses have been given in some countries for migraine prophylaxis.

Dihydroergotamine mesilate has also been used alone or with etilefrine hydrochloride (p.1284) in the treatment of orthostatic hypotension, in usual oral doses of up to 10 mg daily in divided doses. Doses of up to 40 to 60 mg have been used in some patients.

Dihydroergotamine tartrate has been used for indications similar to those of the mesilate.

**Medication-overuse headache.** Dihydroergotamine may be used in the treatment of medication-overuse headache (p.616), including symptoms of ergotamine withdrawal.

**Migraine and cluster headache.** Although sumatriptan is often the treatment of choice to abort acute attacks of migraine (p.616) that do not respond to simple analgesic preparations, parenteral dihydroergotamine, especially with an antiemetic, is an alternative for patients who develop severe or refractory migraine.[1-3] Preparations for intranasal[4,5] use are also available; in some countries, it is given orally. In a comparative study, relief of migraine was slower after subcutaneous dihydroergotamine than after subcutaneous sumatriptan, but headache recurred less often.[6] In other studies, intranasal dihydroergotamine was not as effective as subcutaneous[5] or intranasal[7] sumatriptan.

Dihydroergotamine is also used in the treatment of cluster headache (p.616), usually in emergency settings, where it is given to abort individual headache attacks.


1. Scott AK. Dihydroergotamine: a review of its use in the treatment of migraine and other headaches. *Clin Neuropharmacol* 1992; **15:** 289–96.
2. Silberstein SD, Young WB. Safety and efficacy of ergotamine tartrate and dihydroergotamine in the treatment of migraine and status migrainosus. *Neurology* 1995; **45:** 577–84.
3. Colman I, *et al.* Parenteral dihydroergotamine for acute migraine headache: a systematic review of the literature. *Ann Emerg Med* 2005; **45:** 393–401.
4. Ziegler D, *et al.* Dihydroergotamine nasal spray for the acute treatment of migraine. *Neurology* 1994; **44:** 447–53.
5. Touchon J, *et al.* A comparison of subcutaneous sumatriptan and dihydroergotamine nasal spray in the acute treatment of migraine. *Neurology* 1996; **47:** 361–5.
6. Winner P, *et al.* A double-blind study of subcutaneous dihydroergotamine vs subcutaneous sumatriptan in the treatment of acute migraine. *Arch Neurol* 1996; **53:** 180–4.
7. Boureau F, *et al.* A clinical comparison of sumatriptan nasal spray and dihydroergotamine nasal spray in the acute treatment of migraine. *Int J Clin Pract* 2000; **54:** 281–6.


**Orthostatic hypotension.** Dihydroergotamine may be of use in patients with refractory orthostatic hypotension (p.1530). It is sometimes used in preparations with sympathomimetics such as etilefrine. After parenteral dihydroergotamine, standing blood pressure is increased, but total peripheral resistance and supine blood pressure are also increased.[1] It does not prevent postprandial hypotension, presumably because it does not constrict the splanchnic veins, although use with caffeine may overcome this problem. The main disadvantage of dihydroergotamine, however, is that it is ineffective, or at best weakly effective, when given by mouth, although there has been some evidence that oral ergotamine tartrate may be of value.

Dihydroergotamine has been suggested for use in the prevention of hypotension associated with epidural[2] or spinal anaesthesia,[3] the usual management of which is discussed in Treatment of Adverse Effects of Local Anaesthetics, p.1851. It has also been tried in the management of hypotension associated with haemodialysis.[4]


1. Anonymous. Management of orthostatic hypotension. *Lancet* 1987; **i:** 197–8.
2. Mattila M, *et al.* Dihydroergotamine in the prevention of hypotension associated with extradural anaesthesia. *Br J Anaesth* 1985; **57:** 976–82.
3. Critchley LAH, Woodward DK. Haemodynamic effects of three doses of dihydroergotamine during spinal anaesthesia. *Br J Anaesth* 2001; **87:** 499–501.
4. Milutinovic S. Dihydroergotamin in der Behandlung von Patienten mit symptomatischer Hypotonie während Dauerhämodialyse. *Arzneimittelforschung* 1987; **37:** 554–6.


**Venous thromboembolism.** Standard prophylaxis for surgical patients at high risk of venous thromboembolism is usually with heparin or low-molecular-weight heparin (p.1189). Dihydroergotamine can reduce venous stasis by vasoconstriction of capacitance vessels and has enhanced postoperative prophylaxis when used with heparin.[1] Doses of dihydroergotamine mesilate 500 micrograms with heparin 5000 units, both given subcutaneously 2 hours before surgery, have been used. This regimen has then been given every 8 to 12 hours for 5 to 14 days depending on the risk of thrombosis. The use of dihydroergotamine with low-molecular-weight heparin has been shown to be of similar efficacy to dihydroergotamine with heparin[2,3] but might offer a more convenient dosing schedule. However, although dihydroergotamine might enhance the effect of heparin, a US National Institutes of Health consensus conference warned of the potential risk associated with its vasoconstrictive effects, and the contraindications to its use.[4] In 1989 the Swedish Adverse Drug Reactions Advisory Committee recommended that dihydroergotamine with heparin should not be given for more than 7 days (see Effects on the Cardiovascular System, under Adverse Effects, above).


1. Lindblad B. Prophylaxis of postoperative thromboembolism with low dose heparin alone or in combination with dihydroergotamine: a review. *Acta Chir Scand* 1988; (suppl 543): 31–42.
2. Sasahara AA, *et al.* Low molecular weight heparin plus dihydroergotamine for prophylaxis of postoperative deep vein thrombosis. *Br J Surg* 1986; **73:** 697–700.
3. Haas S, *et al.* Prophylaxis of deep vein thrombosis in high risk patients undergoing total hip replacement with low molecular weight heparin plus dihydroergotamine. *Arzneimittelforschung* 1987; **37:** 839–43.
4. NIH Consensus Development. Prevention of venous thrombosis and pulmonary embolism. *JAMA* 1986; **256:** 744–9.


### Preparations

***USP 31:*** Dihydroergotamine Mesylate Injection.

**Proprietary Preparations** (details are given in Part 3)
***Austral.:*** Dihydergot; ***Austria:*** Detemes; DHE; Dihydergot; Divegal; Ergont; Ergovasan; Migranal; ***Belg.:*** Diergo; Dihydergot; Dystonal; ***Braz.:*** Dihydergot†; ***Canad.:*** Migranal; ***Cz.:*** Clavigrenin†; Dihydergot; ***Fin.:*** Orstanorm; ***Fr.:*** Ikaran; Seglor; Tamik; ***Ger.:*** Agit; Angionorm; Clavigrenin†; DET MS; DET MS spezial†; DHE; Dihydergot†; Dihytamin; Erganton†; Ergomimet†; Ergont†; ergotam; Verladyn; ***Gr.:*** Dihydergot; Pervone†; Verteblan†; ***Hong Kong:*** Tamik†; ***India:*** Dihydergot†; Migranil; ***Indon.:*** Dihydergot; ***Ital.:*** Diidergot; Ikaran; Migranal†; Seglor; ***Mex.:*** Dihydergot; ***Port.:*** Dihydergot†; Seglor; ***Spain:*** Dihydergot; ***Swed.:*** Migranal†; Orstanorm; ***Switz.:*** Dihydergot; Ergotonine; Ikaran†; ***Thai.:*** Poligot†; ***USA:*** DHE; Migranal; ***Venez.:*** Dihydergot.

**Multi-ingredient:** ***Arg.:*** Parsel†; Polper Vascular; ***Austria:*** Agilan; Defluina; Dihydergot; Effortil comp; Hypodyn; Tonopan; Troparin compositum; Venotop; ***Braz.:*** Cefalium; Cefaliv; Enxak; Migraliv; Parcel; Tonopan; ***Chile:*** Emagrip; Migratapsin; Migrax; Parsel†; ***Fr.:*** Diergospray; ***Ger.:*** Agit plus†; Dihydergot plus; Effortil plus; Embolex NM†; Ergo-Lonarid PD†; Ergolefrin; Ergomimet plus†; Optalidon special NOC†; ***Mex.:*** Parsel; Tonopan; ***Spain:*** Tonopan; ***Switz.:*** Dihydergot; Dihydergot plus; Effortil plus; Tonopan†; ***Venez.:*** Brudol; Difen; Dol; Ivagan; Letydol; Parsel; Tainol†.

## Eletriptan Hydrobromide

*(BANM, USAN, rINNM)*

Eletriptaanihydrobromidi; Élétriptan, Bromhydrate d'; Eletriptan-hydrobromid; Eletriptani Hydrobromidum; Hidrobromuro de eletriptán; UK-116044-04. 3-{[(*R*)-1-Methyl-2-pyrrolidinyl]methyl}-5-[2-(phenylsulfonyl)ethyl]indole hydrobromide.

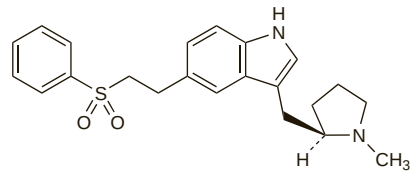
Элетриптана Гидробромид

$C_{22}H_{26}N_2O_2S,HBr$ = 463.4.

*CAS — 143322-58-1 (eletriptan); 177834-92-3 (eletriptan hydrobromide).*

*ATC — N02CC06.*

*ATC Vet — QN02CC06.*



*(eletriptan)*

### Adverse Effects and Precautions

As for Sumatriptan, p.625.

Eletriptan should not be used in patients with severe hepatic or severe renal impairment. Blood pressure effects of eletriptan are increased in renal impairment and therefore the dose should be reduced in patients with mild to moderate renal impairment. No dosage adjustment is needed in mild or moderate hepatic impairment.

**Breast feeding.** Eletriptan is distributed into breast milk and the manufacturer has suggested that infant exposure can be minimised by avoiding breast feeding for 24 hours after treatment.

### Interactions

As for Sumatriptan, p.626.

Eletriptan should not be given with potent inhibitors of the cytochrome P450 isoenzyme CYP3A4 such as erythromycin and ketoconazole; increased plasma levels of eletriptan have been noted after such combinations. It is recommended that eletriptan should not be taken within at least 72 hours of treatment with such drugs.

### Pharmacokinetics

After oral doses eletriptan is rapidly and well absorbed (at least 81%) and has a bioavailability of about 50%. Peak plasma concentrations are attained within 1.5 hours. Eletriptan is about 85% protein bound. It is primarily metabolised by the hepatic cytochrome P450