minoxidil might occur in these patients during multiple-dose therapy. It was advised that minoxidil be started with smaller doses or at longer dosage intervals in patients with renal impairment.

1. Halstenson CE, *et al.* Disposition of minoxidil in patients with various degrees of renal function. *J Clin Pharmacol* 1989; **29:** 798–802.

Alopecia. Minoxidil is used topically to stimulate hair growth in alopecia (p.1577), although its mechanism of action is poorly understood.[1] Increases in pigmented non-vellus hair may be due to thickening and pigmentation of existing vellus rather than new growth.[2] Measurement over 96 weeks showed[3] that minoxidil in solutions of 2 or 5% had a greater effect on hair weight than number of hairs in men with androgenetic alopecia (male-pattern baldness) with the 5% solution being more effective; 24 weeks after treatment was stopped both values had returned to baseline. Another study[4] also showed that 5% minoxidil had a greater effect than 2%, and produced an earlier response. However, 5% minoxidil has been found[5] less effective than oral finasteride. Even with continued use there is a waning of effect with minoxidil.[6,7] It may be more effective in retarding the progression of male-pattern baldness than in reversing it,[2] and users are advised to abandon treatment if there is insufficient benefit after a year.[8]

Minoxidil has also been used in women with female pattern hair loss, and as with men the 5% solution has been found[9] more effective than the 2%. In women with no evidence of biochemical hyperandrogenism minoxidil 2% was more effective[10] than oral cyproterone; where there was such evidence, cyproterone was superior.

Topical minoxidil has been shown to be safe in a large prospective study[11] of men and women with androgenetic alopecia.

Minoxidil appeared to have no beneficial effect on alopecia areata,[12] although one study indicated that topical minoxidil with 0.5% dithranol cream was more effective than either treatment alone.[13]

1. Messenger AG, Rundegren J. Minoxidil: mechanisms of action on hair growth. *Br J Dermatol* 2004; **150:** 186–94.
2. Katz HI. Topical minoxidil: review of efficacy and safety. *Cutis* 1989; **43:** 94–8.
3. Price VH, *et al.* Changes in hair weight and hair count in men with androgenetic alopecia, after application of 5% and 2% topical minoxidil, placebo, or no treatment. *J Am Acad Dermatol* 1999; **41:** 717–21.
4. Olsen EA, *et al.* A randomized clinical trial of 5% topical minoxidil versus 2% topical minoxidil and placebo in the treatment of androgenetic alopecia in men. *J Am Acad Dermatol* 2002; **47:** 377–85.
5. Arca E, *et al.* An open, randomized, comparative study of oral finasteride and 5% topical minoxidil in male androgenetic alopecia. *Dermatology* 2004; **209:** 117–25.
6. de Groot AC, *et al.* Minoxidil: hope for the bald? *Lancet* 1987; **i:** 1019–22.
7. Anonymous. Topical minoxidil does little for baldness. *Drug Ther Bull* 1989; **27:** 74–5.
8. Shrank AB. Treating young men with hair loss. *BMJ* 1989; **298:** 847–8.
9. Lucky AW, *et al.* A randomized, placebo-controlled trial of 5% and 2% topical minoxidil solutions in the treatment of female pattern hair loss. *J Am Acad Dermatol* 2004; **50:** 541–53.
10. Vexiau P, *et al.* Effects of minoxidil 2% vs. cyproterone acetate treatment on female androgenetic alopecia: a controlled, 12-month randomized trial. *Br J Dermatol* 2002; **146:** 992–9.
11. Shapiro J. Safety of topical minoxidil solution: a one-year, prospective, observational study. *J Cutan Med Surg* 2003; **7:** 322–9.
12. Anonymous. Topical minoxidil for baldness: a reappraisal. *Med Lett Drugs Ther* 1994; **36:** 9–10.
13. Fiedler VC, *et al.* Treatment-resistant alopecia areata. *Arch Dermatol* 1990; **126:** 756–9.

CHEMOTHERAPY-INDUCED ALOPECIA. Minoxidil 2% solution was applied daily to the scalp of a boy with acute lymphoblastic leukaemia whose hair had failed to regrow satisfactorily after intensive chemotherapy.[1] Almost normal hair growth, achieved over a 9-month period, was attributed to the use of minoxidil.

A small study[2] in women undergoing combination chemotherapy including doxorubicin found that topical minoxidil applied throughout therapy and for up to 4 months afterwards reduced the duration of alopecia by an average of 50 days.

Other methods for reducing chemotherapy-induced alopecia are described under the Treatment of Adverse Effects of Antineoplastics, p.639.

1. Vickers MA, Barton CJ. Minoxidil induced hair growth after leukaemia treatment? *Arch Dis Child* 1995; **73:** 184.
2. Duvic M, *et al.* A randomized trial of minoxidil in chemotherapy-induced alopecia. *J Am Acad Dermatol* 1996; **35:** 74–8.

Preparations

BP 2008: Minoxidil Scalp Application;
USP 31: Minoxidil Tablets; Minoxidil Topical Solution.

Proprietary Preparations (details are given in Part 3)
Arg.: Anagen; Ivix; Locemix; Macbirs Minoxidil; Minoxile; Toneon†; Tricolocion; Tricoplus; Tricoxane†; Ylox; ***Austral.:*** Loniten; Regaine; ***Austria:*** Loniten; Regaine; Rogaine; ***Belg.:*** Neoxidil; Regaine; ***Braz.:*** Loniten; Neoxidil†; Regaine; ***Canad.:*** Apo-Gain; Loniten; Minox†; Rogaine; ***Chile:*** Alopek; Regaine; Tricoxane; ***Cz.:*** Minoxitrim†; Neocapil; Regaine; ***Denm.:*** Regaine; ***Fin.:*** Recrea; Regaine†; Rogaine; ***Fr.:*** Alopexy; Alostil; Lonoten; Regaine; ***Ger.:*** Lonolox; Regaine; ***Gr.:*** Axelan; Botafex; Dermolantyl; Ebersedin; Hairway; Loniten; Lotorin; Minodril; Monoxidil†; Neo-Pruristam; Nherea; Oxofenil; Regaine; Stemeral; ***Hong Kong:*** Apo-Gain†; Hairgrow; Headway; Loniten; Minoxi; Neoxidil†; Regaine; Regro; ***Hung.:*** Neocapil; Regaine; ***India:*** Mintop; ***Indon.:*** Aloxid; Eminox; Regaine; Regrou; ***Irl.:*** Loniten; Rogaine; ***Israel:*** Alopexy; Hair-Treat; Hairgaine; Minoxi; Neoxidil; Regaine; ***Ital.:*** Aloxidil; Loniten; Minovital; Minoximen; Normoxidil†; Regaine; Tricoxidil; ***Malaysia:*** Apo-Gain; Epokelan†; Headway; Regaine; Regro; ***Mex.:*** Folcres†; Regaine; ***Neth.:*** Alopexy; Lonnoten; Regaine; ***Norw.:*** Regaine†; Rogaine; ***NZ:*** Headway; Rogaine; ***Philipp.:*** Regroe; ***Pol.:*** Loxon; Piloxidil; Regaine; ***Port.:*** Biocrinal; Crinalsofex; Hairtene; Loniten†; Mantai; Minocalve; Minox; Neoxidil; Regaine; Tricovivax; Zeldilon; ***Rus.:*** Regaine (Регейн); ***S.Afr.:*** Loniten; Regaine; ***Singapore:*** Growell; Minoxitrim; Neoxidil†; Regaine; Regro; ***Spain:*** Alopexy; Carexidil; Dinaxil Capilar; Lacovin; Loniten; Regaine; Regaxidil; Riteban†; ***Swed.:*** Recrea; Regaine†; Revexan; Rogaine; ***Switz.:*** Alopexy; Loniten; Neocapil; Piloxil†; Regaine; ***Thai.:*** Loniten; Manoxidil; Minoxitrim†; Modil; Noxidil; Nuhair; Regaine; Regrowth; Reten; ***UK:*** Loniten; Regaine; ***USA:*** Loniten†; Rogaine; ***Venez.:*** Guayaten; Regaine†; Topixidil; Zitoxil.

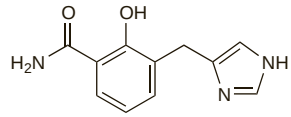
Multi-ingredient: ***Arg.:*** Tricoplus Coneff†.

Mivazerol *(rINN)*

Mivazérol; Mivazerolum; UCB-22073. α-Imidazol-4-yl-2,3-cresotamide.

Мивазерол

$C_{11}H_{11}N_3O_2$ = 217.2.
CAS — 125472-02-8.



Profile

Mivazerol is an alpha$_2$-adrenoceptor agonist that has been investigated for the prevention of perioperative complications resulting from myocardial ischaemia in patients with ischaemic heart disease undergoing non-cardiac surgery.

◊ References.

1. Oliver MF, *et al.* Effect of mivazerol on perioperative cardiac complications during non-cardiac surgery in patients with coronary heart disease: the European Mivazerol Trial (EMIT). *Anesthesiology* 1999: **91:** 951–61.

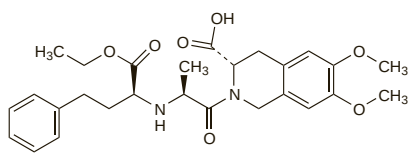
Moxipril Hydrochloride

(BANM, USAN, rINNM)

CI-925; Hidrocloruro de moexipril; Moeksipril Hidroklorür; Moexipril, Chlorhydrate de; Moexiprili Hydrochloridum; RS-10085-197; SPM-925. (3*S*-{2[*R**(*R**)],3*R**})-2-(2-{[1-(Ethoxycarbonyl)-3-phenylpropyl]amino}-1-oxopropyl)-1,2,3,4-tetrahydro-6,7-dimethoxy-3-isoquinoline-carboxylic acid hydrochloride.

Моэксиприла Гидрохлорид

$C_{27}H_{34}N_2O_7$,HCl = 535.0.
CAS — 103775-10-6 (moexipril); 82586-52-5 (moexipril hydrochloride).
ATC — C09AA13.
ATC Vet — QC09AA13.



(moexipril)

Adverse Effects, Treatment, and Precautions

As for ACE inhibitors, p.1193.

Interactions

As for ACE inhibitors, p.1196.

Pharmacokinetics

Moexipril acts as a prodrug of the diacid moexiprilat, its active metabolite. After oral doses moexipril is rapidly but incompletely absorbed and is metabolised to moexiprilat in the gastrointestinal mucosa and liver. Absorption is reduced in the presence of food. The bioavailability of moexiprilat is about 13% after oral doses of moexipril, and peak plasma concentrations of moexiprilat are reached in about 1.5 hours. Both moexipril and moexiprilat are moderately bound to plasma proteins. Moexipril is excreted mainly in the urine as moexiprilat, unchanged drug, and other metabolites; some moexiprilat may also be excreted in the faeces. The functional elimination half-life of moexiprilat is about 12 hours.

Uses and Administration

Moexipril is an ACE inhibitor (p.1193). It is used in the treatment of hypertension (p.1171).

Moexipril owes its activity to moexiprilat, to which it is converted after oral doses. The haemodynamic effects are seen about 1 hour after an oral dose and the maximum effect occurs after about 3 to 6 hours, although the full effect may not develop for 2 to 4 weeks during chronic dosing. Moexipril is given orally as the hydrochloride.

In the treatment of hypertension, the usual initial dose of moexipril hydrochloride is 7.5 mg once daily. Since there may be a precipitous fall in blood pressure in some patients when starting therapy with an ACE inhibitor, the first dose should preferably be given at bedtime. An initial dose of 3.75 mg once daily, given under close medical supervision, is suggested for patients who are taking a *diuretic*; if possible the diuretic should be withdrawn 2 or 3 days before moexipril is started and resumed later if necessary. An initial dose of 3.75 mg once daily is also recommended for patients with renal or hepatic impairment and for the elderly.

The usual maintenance dose is 7.5 to 30 mg daily, which may be given in 2 divided doses if control is inadequate with a single dose.

◊ Reviews.

1. Brogden RN, Wiseman LR. Moexipril: a review of its use in the management of essential hypertension. *Drugs* 1998; **55:** 845–60.
2. Chrysant SG, Chrysant GS. Pharmacological and clinical profile of moexipril: a concise review. *J Clin Pharmacol* 2004; **44:** 827–36.

Administration in renal impairment. In patients with renal impairment (creatinine clearance 40 mL/minute or less) an initial dose of moexipril hydrochloride 3.75 mg is given; in the USA it is required that the maximum dose in such patients should not exceed 15 mg daily.

Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Fempress; ***Cz.:*** Moex; ***Fr.:*** Moex; ***Ger.:*** Fempress; ***Gr.:*** Tensotec†; ***Hong Kong:*** Moex; ***Irl.:*** Perdix†; ***Israel:*** Perdix; ***Ital.:*** Fempires; ***Malaysia:*** Tensotec†; ***Philipp.:*** Univasc; ***Pol.:*** Cardiotensin; ***Port.:*** Tensotec; ***Rus.:*** Moex (Моэкс); ***S.Afr.:*** Perdix; ***Turk.:*** Univasc; ***UK:*** Perdix; ***USA:*** Univasc.

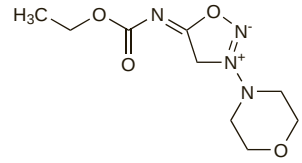
Multi-ingredient: ***Austria:*** Fempress Plus; ***Ger.:*** Fempress Plus; ***Ital.:*** Enulid; Femipres Plus; ***Philipp.:*** Uniretic; ***Rus.:*** Moex Plus (Моэкс Плюс); ***USA:*** Uniretic.

Molsidomine *(BAN, USAN, rINN)*

CAS-276; Molsidomiini; Molsidomin; Molsidomina; Molsidominum; Morsydomine; SIN-10. *N*-Ethoxycarbonyl-3-morpholinosydnonimine.

Молсидомин

$C_9H_{14}N_4O_4$ = 242.2.
CAS — 25717-80-0.
ATC — C01DX12.
ATC Vet — QC01DX12.



Pharmacopoeias. In *Eur.* (see p.vii).
Ph. Eur. 6.2 (Molsidomine). A white or almost white, crystalline powder. Sparingly soluble in water; soluble in anhydrous alcohol and in dichloromethane. A 1% solution in water has a pH of 5.5 to 7.5. Protect from light.

Profile

Molsidomine is a nitrovasodilator used in angina pectoris (p.1157). It may also be used in heart failure (p.1165) and after myocardial infarction (below).

Molsidomine is given in usual oral doses of 1 to 4 mg two to four times daily. Modified-release preparations are also available. It is also given intravenously in single doses of 2 to 4 mg and doses of 2 mg may be repeated at intervals of at least 2 hours if necessary; total doses of up to 40 mg daily have been given. Infusions may be given at a rate of up to 3 mg/hour.

Molsidomine is metabolised to linsidomine (p.1325), an active metabolite.

Carcinogenicity. Molsidomine tends to degrade into morpholine (even when protected from the light), a compound considered potentially carcinogenic. This finding led to the suspension of marketing of one molsidomine formulation;[1] an earlier temporary suspension was related to evidence of carcinogenicity in some *animals*, although this has not been confirmed in humans.

1. Anonymous. Corvaton Tropfen. *Dtsch Apotheker Ztg* 1989; **129** (49): VI.

Myocardial infarction. Although intravenous nitrates (glyceryl trinitrate or sodium nitroprusside) may be used in the management of acute myocardial infarction (p.1175), molsidomine and its active metabolite linsidomine (a nitric oxide donor) had no effect on mortality.[1]

1. European Study of Prevention of Infarct with Molsidomine (ESPRIM) Group. The ESPRIM trial: short-term treatment of acute myocardial infarction with molsidomine. *Lancet* 1994; **344:** 91–7.

Pharmacokinetics. The pharmacokinetics of molsidomine have been reviewed.[1] Molsidomine is metabolised in the liver to linsidomine and other morpholine derivatives. Prolonged elimination half-lives of molsidomine and linsidomine due to reduced plasma clearance have been reported in patients with liver cirrhosis.[2]

1. Rosenkranz B, *et al.* Clinical pharmacokinetics of molsidomine. *Clin Pharmacokinet* 1996; **30:** 372–84.
2. Spreux-Varoquaux O, *et al.* Pharmacokinetics of molsidomine and its active metabolite, linsidomine, in patients with liver cirrhosis. *Br J Clin Pharmacol* 1991; **32:** 399–401.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Molsicor; Molsidaine; ***Austria:*** Molsidolat; Molsihexal; ***Belg.:*** Coruno; Corvatard†; Corvaton; ***Cz.:*** Corvaton; Molsihexal; Molsiket; ***Fr.:*** Corvasal; ***Ger.:*** Corvaton; duracoron†; Molsi-Azu†; Molsi-Puren; Molsi†; Molsibeta; Molsihexal; molsiket; ***Hung.:*** Corvaton; ***Port.:*** Corvaton†; ***Rus.:*** Dilasidom (Диласидом); Sydnopharm (Сиднофарм); ***Spain:*** Corpea; Molsidain; ***Switz.:*** Corsifar†; Corvaton.

Monteplase *(rINN)*

E-6010; Monteplasa; Montéplase; Monteplasum.

Монтеплаза

$C_{2569}H_{3896}N_{746}O_{783}S_{39}$ = 59009.5.
CAS — 156616-23-8.

Profile

Monteplase is a thrombolytic related to alteplase (p.1207) that is used in acute myocardial infarction (p.1175) and venous thromboembolism (p.1189). For acute myocardial infarction, the usual dose is 27 500 units/kg given by intravenous injection as soon as possible after the onset of symptoms. For pulmonary embolism, the usual dose is 13 750 units/kg to 27 500 units/kg.

◊ References.

1. Kawai C, *et al.* A prospective, randomized, double-blind multicenter trial of a single bolus injection of the novel modified t-PA E6010 in the treatment of acute myocardial infarction: comparison with native t-PA. *J Am Coll Cardiol* 1997; **29:** 1447–53.
2. Inoue T, *et al.* A new thrombolytic agent, monteplase, is independent of the plasminogen activator inhibitor in patients with acute myocardial infarction: initial results of the COmbining Monteplase with Angioplasty (COMA) trial. *Am Heart J* 2002; **144:** E5.
3. Inoue T, *et al.* Long-term benefits of monteplase before coronary angioplasty in acute myocardial infarction. *Am J Cardiol* 2005; **95:** 506–8.
4. Inoue T, *et al.* Therapeutic potential of monteplase in acute myocardial infarction. *Am J Cardiovasc Drugs* 2005; **5:** 225–31.

Preparations

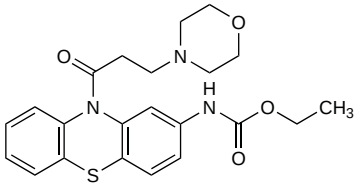
Proprietary Preparations (details are given in Part 3)
Jpn: Cleactor.

Moracizine *(BAN, rINN)*

EN-313; Moracizina; Moracizinum; Moricizine *(USAN)*. Ethyl [10-(3-morpholinopropionyl)phenothiazin-2-yl]carbamate.

Морацизин

$C_{22}H_{25}N_3O_4S$ = 427.5.
CAS — 31883-05-3.
ATC — C01BG01.
ATC Vet — QC01BG01.



Moracizine Hydrochloride *(BANM, rINNM)*

Hidrocloruro de moracizina; Moracizine, Chlorhydrate de; Moracizinhydroklorid; Moracizini Hydrochloridum; Morasitsiinihydrokloridi.

Морацизина Гидрохлорид

$C_{22}H_{25}N_3O_4S$,HCl = 464.0.
CAS — 29560-58-5.
ATC — C01BG01.
ATC Vet — QC01BG01.

Pharmacopoeias. In *Chin.* and *US*.
USP 31 (Moricizine Hydrochloride). A white to off-white crystalline powder. Soluble in water and in alcohol. Store in airtight containers.

Adverse Effects

The most common adverse effects associated with moracizine affect the CNS and the gastrointestinal tract and include dizziness, headache, fatigue, nausea, and abdominal pain. Other adverse effects include dyspnoea, dry mouth, blurred vision, impotence, and urinary-tract disorders. There have been occasional reports of fever, thrombocytopenia, hepatic dysfunction, hypothermia, and skin rash.

Like other antiarrhythmics moracizine can provoke or worsen arrhythmias. This may range from an increase in the frequency of premature ventricular contractions to induction or worsening of ventricular tachycardia.

An increased mortality rate occurred when moracizine was tested in the control of asymptomatic ventricular arrhythmias in post-infarction patients (see Cardiac Arrhythmias under Uses and Administration, below).

Effects on body temperature. Fever with elevated creatine phosphokinase and hepatic transaminase concentrations was associated with moracizine in 2 patients.[1] The fever abated within 48 hours of stopping moracizine and recurred within 24 hours of rechallenge in both patients. Results suggested a similarity to the neuroleptic malignant syndrome that is associated with other phenothiazine derivatives.

1. Miura DS, *et al.* Ethmozine toxicity: fever of unknown origin. *J Clin Pharmacol* 1986; **26:** 153–5.

Precautions

As for Flecainide Acetate, p.1288.

Interactions

Use of moracizine with other antiarrhythmics or arrhythmogenic drugs may increase the incidence of cardiac arrhythmias. Moracizine undergoes metabolism in the liver and its activity may be influenced by other drugs affecting the enzymes responsible for its metabolism; it is an enzyme inducer and may also affect the activity of other hepatically-metabolised drugs.

Pharmacokinetics

Moracizine is readily and almost completely absorbed from the gastrointestinal tract. It undergoes significant first-pass hepatic metabolism so that the oral bioavailability is about 38%. Moracizine is extensively metabolised and some of the numerous metabolites may be active. It induces its own metabolism; the plasma elimination half-life is about 2 hours after multiple doses. Although plasma concentrations are reduced with multiple dosing, clinical response is not affected. It is about 95% bound to plasma proteins. Moracizine is distributed into breast milk. About 56% of a dose is excreted in the faeces and about 39% in the urine.

◊ References.

1. Benedek IH, *et al.* Enzyme induction by moricizine: time course and extent in healthy subjects. *J Clin Pharmacol* 1994; **34:** 167–75.

Uses and Administration

Moracizine is a phenothiazine compound that has class I antiarrhythmic activity (p.1153) but does not readily fall into the subclasses a, b, or c. It is used as the hydrochloride in the treatment of serious symptomatic ventricular arrhythmias. Moracizine hydrochloride is given orally in a usual dose of 600 to 900 mg daily in 2 or 3 divided doses. Treatment should be started in hospital, and doses should be adjusted at intervals of not less than 3 days. Doses should be reduced in patients with hepatic or renal impairment (see below).

◊ Reviews.

1. Clyne CA, *et al.* Moricizine. *N Engl J Med* 1992; **327:** 255–60.

Administration in hepatic or renal impairment. The initial dose of moracizine hydrochloride in hepatic or renal impairment should be 600 mg or less daily and patients should be monitored closely (including ECG) before any adjustment of dose is made.

Cardiac arrhythmias. Moracizine is effective in various arrhythmias but is usually reserved for life-threatening ventricular arrhythmias; like other class I antiarrhythmics (see Cardiac Arrhythmias under Flecainide, p.1289), it has been associated with increased mortality when used prophylactically after myocardial infarction,[1] and is not recommended in such patients. However, there is limited evidence that it may be useful in some patients with supraventricular arrhythmias.[2,3]

1. The Cardiac Arrhythmia Suppression Trial II Investigators. Effect of the antiarrhythmic agent moricizine on survival after myocardial infarction. *N Engl J Med* 1992; **327:** 227–33.
2. Mehta AV, *et al.* Experience with moricizine HCl in children with supraventricular tachycardia. *Int J Cardiol* 1996; **57:** 31–5.
3. Geller JC, *et al.* Efficacy and safety of moricizine in the maintenance of sinus rhythm in patients with recurrent atrial fibrillation. *Am J Cardiol* 2001; **87:** 172–7.

Preparations

USP 31: Moricizine Hydrochloride Tablets.

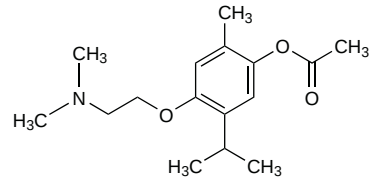
Proprietary Preparations (details are given in Part 3)
USA: Ethmozine†.

Moxisylyte Hydrochloride *(BANM, rINNM)*

Hidrocloruro de moxisilita; Moksisylyyttihydrokloridi; Moxisilita Clorhidrato; Moxisylyte, Chlorhydrate de; Moxisylythydroklorid; Moxisylyti Hydrochloridum; Moxisylytum Hydrochloridum; Thymoxamine Hydrochloride. 4-(2-Dimethylaminoethoxy)-5-isopropyl-2-methylphenyl acetate hydrochloride.

Моксизилита Гидрохлорид

$C_{16}H_{25}NO_3$,HCl = 315.8.
CAS — 54-32-0 (moxisylyte); 964-52-3 (moxisylyte hydrochloride).
ATC — C04AX10; G04BE06.
ATC Vet — QC04AX10; QG04BE06.



(moxisylyte)

NOTE. MOX, formerly THY, is a code approved by the BP 2008 for use on single unit doses of eye drops containing moxisylyte hydrochloride where the individual container may be too small to bear all the appropriate labelling information.

Pharmacopoeias. In *Br.*
BP 2008 (Moxisylyte Hydrochloride). A white, odourless or almost odourless, crystalline powder. Freely soluble in water and in chloroform; soluble in alcohol; practically insoluble in ether and in petroleum spirit. A 5% solution in water has a pH of 4.5 to 5.5. Protect from light.

Adverse Effects

Moxisylyte hydrochloride may cause nausea, diarrhoea, headache, vertigo, flushing of the skin, dry mouth, and nasal congestion. Hepatotoxicity has been reported. Overdosage may cause hypotension.

Transient ptosis has occurred occasionally after ophthalmic application. Prolonged erections or priapism have occurred rarely after intracavernosal injection and systemic effects may also occur.

Effects on the liver. Hepatic adverse reactions with moxisylyte first appeared in France after its use in benign prostatic hyperplasia, a condition in which relatively high doses were used (up to 480 mg daily compared with up to 320 mg daily for peripheral vascular disease). Since then the UK CSM has received reports associated with lower doses.[1] Thirteen hepatic reactions, accounting for 17% of all reports of suspected adverse reactions to moxisylyte, had been received. These comprised 3 cases of hepatic function abnormalities, 3 of jaundice, 4 of cholestatic jaundice, 2 of hepatitis, and 1 of hepatitis with jaundice. In most cases the reaction occurred within 5 weeks of the start of treatment and resolved on drug withdrawal. In 9 cases the dosage of moxisylyte was known and varied from 80 to 320 mg daily with 7 patients receiving 160 mg or less daily.

1. Committee on Safety of Medicines/Medicines Control Agency. Hepatic reactions with thymoxamine (Opilon). *Current Problems* 1993; **19:** 11–12. Also available at: http://www.mhra.gov.uk/home/idcplg?IdcService=GET_FILE&dDocName=CON2024456&RevisionSelectionMethod=LatestReleased (accessed 10/04/08)

Precautions

Moxisylyte hydrochloride should not be given to patients with active liver disease and should be given with care to patients with diabetes mellitus as it may theoretically decrease insulin requirements. Monitoring of liver function is recommended, especially if therapy is prolonged or if high doses are being used. Intracavernosal injection of moxisylyte is contra-indicated in patients with conditions that predispose to priapism.

Interactions

Moxisylyte may enhance the effects of antihypertensives and the hypotensive effect of moxisylyte may be enhanced by tricyclic antidepressants.

Uses and Administration

Moxisylyte is an alpha-adrenoceptor blocker with vasodilating activity. It is used by mouth in the treatment of peripheral vascular disease (p.1178) and has been self-administered by intracavernosal injection in erectile dysfunction (p.2179).