Apomorphine is extensively metabolised in the liver, primarily by conjugation with glucuronic acid or sulfate; the major metabolite is apomorphine sulfate. It is also demethylated to produce norapomorphine. Most of a dose is excreted in urine, mainly as metabolites.

◊ References.

1. Neef C, van Laar T. Pharmacokinetic-pharmacodynamic relationships of apomorphine in patients with Parkinson's disease. *Clin Pharmacokinet* 1999; **37:** 257–71.
2. Argiolas A, Hedlund H. The pharmacology and clinical pharmacokinetics of apomorphine SL. *BJU Int* 2001; **88** (suppl 3): 18–21.
3. LeWitt PA. Subcutaneously administered apomorphine: pharmacokinetics and metabolism. *Neurology* 2004; **62** (suppl 4): S8–S11.

Uses and Administration

Apomorphine is a morphine derivative with structural similarities to dopamine. It is a potent dopamine D_1- and D_2-receptor agonist used in the management of parkinsonism, especially in the control of the 'on-off' effect. It has also been used in the management of erectile dysfunction. Apomorphine is given as the hydrochloride and doses are expressed in terms of this salt. The regimen for parkinsonism given below applies to the UK preparation and doses are give subcutaneously; a similar preparation is available in the USA although the licensed maximum single and daily doses are less than those in the UK.

The optimal dose of apomorphine in the management of 'off' periods in **parkinsonism** should be established individually under specialist care. At least 2 days of pretreatment with the antiemetic domperidone is advised before starting apomorphine. After withholding antiparkinsonian therapy overnight to provoke an 'off' period, a test dose of 1 mg is given initially, followed by a second dose of 2 mg after 30 minutes, if necessary. Subsequent incremental increases should then be given at intervals of at least 40 minutes, as necessary, to determine the lowest dose producing a satisfactory motor response. Once the patient's normal antiparkinsonian therapy is re-established, the effective dose of apomorphine hydrochloride is given at the first signs of an 'off' period.

The dose and frequency are further adjusted according to response; patients typically require 3 to 30 mg daily in divided doses but individual injections should not be greater than 10 mg. Patients who require more than 10 injections daily or those whose overall control of symptoms remains unsatisfactory with intermittent injections may benefit from continuous subcutaneous infusion. The infusion is started at a rate of 1 mg/hour and this may be increased in steps of up to 0.5 mg/hour at intervals of not less than 4 hours to a maximum rate of 4 mg/hour. It is advised that infusions should only be given during waking hours and that the infusion site should be changed every 12 hours; 24-hour infusions are not advised unless there are severe night-time symptoms. Patients usually need to supplement the infusion with intermittent bolus injections but the recommended maximum total daily dose given by infusion and/or injection is 100 mg.

In the management of **erectile dysfunction** the usual initial dose has been 2 mg taken sublingually about 20 minutes before sexual activity. A dose of 3 mg was used on subsequent occasions if necessary with a minimum of 8 hours between doses. Reduced dosage is needed in patients with renal impairment (see below).

Apomorphine stimulates the chemoreceptor trigger zone in the brain and can produce emesis within a few minutes of a dose. However, the use of apomorphine for the induction of emesis in poisoning is considered dangerous owing to the risk of inducing protracted vomiting and shock, and is not recommended.

Administration in renal impairment. In the management of erectile dysfunction, the maximum dose of apomorphine hydrochloride has been limited to 2 mg sublingually in patients with severe renal impairment.

Erectile dysfunction. Apomorphine is among a wide range of drugs that has been used in the management of erectile dysfunction[1-5] (p.2179) with some beneficial results. It is usually given sublingually although it has also been given subcutaneously.[3] Inhaled apomorphine is also under investigation.

1. Heaton JPW, *et al.* Recovery of erectile function by the oral administration of apomorphine. *Urology* 1995; **45:** 200–6.
2. Dula E, *et al.* Efficacy and safety of fixed-dose and dose-optimization regimens of sublingual apomorphine versus placebo in men with erectile dysfunction. *Urology* 2000; **56:** 130–5.
3. Segraves RT, *et al.* Effect of apomorphine on penile tumescence in men with psychogenic impotence. *J Urol (Baltimore)* 1991; **145:** 1174–5.
4. Martinez R, *et al.* Clinical experience with apomorphine hydrochloride: the first 107 patients. *J Urol (Baltimore)* 2003; **170:** 2352–5.
5. Gontero P, *et al.* Clinical efficacy of apomorphine SL in erectile dysfunction of diabetic men. *Int J Impot Res* 2005; **17:** 80–5.

DIAGNOSIS. Test doses of subcutaneous apomorphine have been used in the differential diagnosis of parkinsonian syndromes,[1-4] to distinguish forms responsive to dopaminergics from other parkinsonian syndromes such as Wilson's disease, corticobasal degeneration, and diffuse Lewy-body dementia. Oral challenge with levodopa is still the best test of dopaminergic responsiveness[5,6] but apomorphine has proved of value in re-assessing patients who have become less responsive to levodopa.[1,4]

1. Barker R, *et al.* Subcutaneous apomorphine as a diagnostic test for dopaminergic responsiveness in parkinsonian syndromes. *Lancet* 1989; **i:** 675.
2. Oertel WH, *et al.* Apomorphine test for dopaminergic responsiveness. *Lancet* 1989; **i:** 1262–3.
3. Frankel JP, *et al.* Use of apomorphine to test for dopamine responsiveness in Wilson's disease. *Lancet* 1989; **ii:** 801–2.
4. Hughes AJ, *et al.* Apomorphine test to predict dopaminergic responsiveness in parkinsonian syndromes. *Lancet* 1990; **336:** 32–4.
5. Steiger MJ, Quinn NP. Levodopa challenge test in Parkinson's disease. *Lancet* 1992; **339:** 751–2.
6. Müller T, *et al.* Repeated rating improves value of diagnostic dopaminergic challenge tests in Parkinson's disease. *J Neural Transm* 2003; **110:** 603–9.

Parkinsonism. ***TREATMENT.*** Although apomorphine has produced benefit in Parkinson's disease (p.791) when given orally, the high doses required to overcome extensive first-pass hepatic metabolism (up to 1.4 g daily in one study[1]), were associated with uraemia. The use of apomorphine in Parkinson's disease has therefore been limited by the need for parenteral dosage. The current main use of apomorphine in Parkinson's disease is for the stabilisation of patients with 'on-off' fluctuations unresponsive to other dopamine agonists. It is usually given subcutaneously either by injection or infusion but a review[2] of the use of apomorphine in Parkinson's disease also discussed studies of rectal, sublingual, and intranasal use. Inhaled apomorphine is also under investigation.

1. Cotzias GC, *et al.* Treatment of Parkinson's disease with aporphines. *N Engl J Med* 1976; **294:** 567–72.
2. Koller W, Stacy M. Other formulations and future considerations for apomorphine for subcutaneous injection therapy. *Neurology* 2004; **62** (suppl 4): S22–S26.

Preparations

USP 31: Apomorphine Hydrochloride Tablets.

Proprietary Preparations (details are given in Part 3)

Arg.: Apokinon†; Uprima†; ***Austral.:*** Apomine; ***Austria:*** APO-go; Ixense†; Uprima†; ***Belg.:*** Uprima†; ***Braz.:*** Uprima; ***Chile:*** Noc; Uprima†; ***Cz.:*** APO-go; Uprima†; ***Denm.:*** Uprima†; ***Fin.:*** Uprima†; ***Fr.:*** Apokinon†; Ixense†; Uprima†; ***Ger.:*** APO-go; Ixense†; Uprima†; ***Gr.:*** APO-go; Taluvian†; Uprima†; ***Hong Kong:*** Uprima†; ***Hung.:*** APO-go†; Uprima†; ***Irl.:*** Uprima†; ***Israel:*** APO-go; ***Ital.:*** Apofin; Ixense†; Taluvian†; Uprima†; ***Jpn:*** Ixense; ***Mex.:*** Taluvian; ***Neth.:*** APO-go; Uprima†; ***Norw.:*** Uprima†; ***NZ:*** Apomine; Uprima†; ***Port.:*** APO-go; Uprima†; ***S.Afr.:*** Uprima†; ***Spain:*** APO-go; Taluvian†; Uprima†; ***Swed.:*** Uprima†; ***Switz.:*** Uprima†; ***Thai.:*** Ixense†; ***Turk.:*** APO-go; ***UK:*** APO-go; Uprima†; ***USA:*** Apokyn; ***Venez.:*** Uprima†.

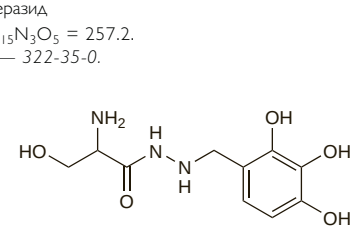
Benserazide *(BAN, USAN, rINN)*

Benseratsidi; Benserazid; Benserazida; Bensérazide; Benserazidum; Ro-4-4602. DL-Serine 2-(2,3,4-trihydroxybenzyl)hydrazide; 2-Amino-3-hydroxy-2'-(2,3,4-trihydroxybenzyl)propionohydrazide.

Бенсеразид

$C_{10}H_{15}N_3O_5$ = 257.2.

CAS — 322-35-0.



Benserazide Hydrochloride *(BANM, rINNM)*

Benseratsidihydrokloridi; Bensérazide, chlorhydrate de; Benserazid-hydrochlorid; Benserazidhydroklorid; Benserazidi hydrochloridum; Benserazido hidrochloridas; Benszerazid-hidroklorid; Hidrocloruro de benserazida; Serazide Hydrochloride.

Бенсеразида Гидрохлорид

$C_{10}H_{15}N_3O_5$,HCl = 293.7.

CAS — 14919-77-8; 14046-64-1.

NOTE. Compounded preparations of benserazide hydrochloride may be represented by the following names:

- Co-beneldopa *(BAN)*—benserazide 1 part and levodopa 4 parts (w/w).

Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), and *Jpn.*

Ph. Eur. 6.2 (Benserazide Hydrochloride). A white or yellowish-white or orange-white crystalline powder. It shows polymorphism. Freely soluble in water; very slightly soluble in dehydrated alcohol; practically insoluble in acetone. A 1% solution in water has a pH of 4.0 to 5.0. Protect from light.

Solubility. Benserazide is unstable in a neutral, alkaline, or strongly acidic medium.[1]

1. Schwartz DE, Brandt R. Pharmacokinetic and metabolic studies of the decarboxylase inhibitor benserazide in animals and man. *Arzneimittelforschung* 1978; **28:** 302–7.

Adverse Effects and Precautions

◊ Early reports[1] noted developmental abnormalities of the *rat* skeleton, but others found no evidence of any disorder involving bone metabolism in man.[2] Nevertheless licensed product information recommends that benserazide should not be given to patients under 25 years of age, nor to pregnant women or to women of child-bearing potential in the absence of adequate contraception.

1. Theiss E, Schärer K. Toxicity of L-dopa and a decarboxylase inhibitor in animal experiments. In: de Ajuriaguerra J, Gauthier G, eds. *Monoamines Noyaux Gris Centraux et Syndrome de Parkinson*. Geneva: Georg, 1971: 497–504.
2. Ziegler WH, *et al.* Toxicity of L-dopa and a dopa decarboxylase inhibitor in humans. In: de Ajuriaguerra J, Gauthier G, eds. *Monoamines Noyaux Gris Centraux et Syndrome de Parkinson*. Geneva: Georg, 1971: 505–16.

Pharmacokinetics

◊ Pharmacokinetic and metabolic studies[1,2] in *animals* and man have shown that, after oral doses in parkinsonian patients, benserazide was rapidly absorbed to the extent of about 58%; giving it with levodopa tended to increase this slightly. Benserazide was rapidly excreted in the urine in the form of metabolites, mostly within the first 6 hours; 85% of urinary excretion had occurred within 12 hours. It is mainly metabolised in the gut and appears to protect levodopa against decarboxylation primarily in the gut, but also in the rest of the organism, mainly by way of its metabolite trihydroxybenzylhydrazine. Benserazide did not cross the blood-brain barrier in *rats*.

1. Schwartz DE, *et al.* Pharmacokinetics of the decarboxylase benserazide in man: its tissue distribution in the rat. *Eur J Clin Pharmacol* 1974; **7:** 39–45.
2. Schwartz DE, Brandt R. Pharmacokinetic and metabolic studies of the decarboxylase inhibitor benserazide in animals and man. *Arzneimittelforschung* 1978; **28:** 302–7.

Uses and Administration

Benserazide hydrochloride is a peripheral dopa-decarboxylase inhibitor with actions similar to those of carbidopa (p.803) and is used similarly as an adjunct to levodopa in the treatment of parkinsonism (p.791). For details of dosage, see Levodopa, p.808.

◊ References.

1. Dingemanse J, *et al.* Pharmacodynamics of benserazide assessed by its effects on endogenous and exogenous levodopa pharmacokinetics. *Br J Clin Pharmacol* 1997; **44:** 41–8.

Preparations

BP 2008: Co-beneldopa Capsules; Dispersible Co-beneldopa Tablets.

Proprietary Preparations (details are given in Part 3)

Ger.: Restex.

Multi-ingredient: ***Arg.:*** Madopar; ***Austral.:*** Madopar; ***Austria:*** Dopamed; Levobens; Madopar; Restex; ***Belg.:*** Prolopa; ***Braz.:*** Prolopa; ***Canad.:*** Prolopa; ***Chile:*** Melitase; Prolopa; ***Cz.:*** Madopar; ***Denm.:*** Madopar; ***Fin.:*** Madopar; ***Fr.:*** Modopar; ***Ger.:*** Levodopa comp B; Levopar; Madopar; PK-Levo; ***Gr.:*** Madopar; ***Hong Kong:*** Madopar; ***Hung.:*** Madopar; ***Indon.:*** Leparson; Levazide; Levopar; Madopar; Pardoz; ***Irl.:*** Madopar; ***Israel:*** Levopar Plus; ***Ital.:*** Madopar; ***Malaysia:*** Madopar; ***Mex.:*** Madopar; ***Neth.:*** Madopar; Modopar; ***Norw.:*** Madopar; ***NZ:*** Madopar; ***Philipp.:*** Madopar; ***Pol.:*** Madopar; ***Port.:*** Madopar; ***Rus.:*** Madopar (Мадопар); ***S.Afr.:*** Madopar; ***Singapore:*** Madopar; ***Spain:*** Madopar; ***Swed.:*** Madopark; ***Switz.:*** Madopar; ***Thai.:*** Cenparkin†; Madopar; Vopar; ***Turk.:*** Madopar; ***UK:*** Madopar; ***Venez.:*** Madopar.

Benzatropine Mesilate *(BANM, rINNM)*

Benzatropine, Mésilate de; Benzatropine Methanesulfonate; Benzatropini Mesilas; Benztropine Mesylate; Mesilato de benzatropina. (1*R*,3*r*,5*S*)-3-Benzhydryloxytropane methanesulphonate.

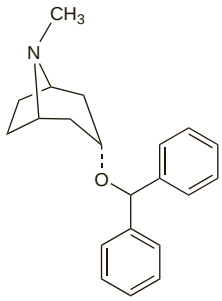
Бензатропина Мезилат

$C_{21}H_{25}NO,CH_4O_3S$ = 403.5.

CAS — 86-13-5 (benzatropine); 132-17-2 (benzatropine mesilate).

ATC — N04AC01.

ATC Vet — QN04AC01.



(benzatropine)

Pharmacopoeias. In *Br.* and *US*.

BP 2008 (Benzatropine Mesilate). A white, odourless or almost odourless, crystalline powder. Very soluble in water; freely soluble in alcohol; practically insoluble in ether.

USP 31 (Benztropine Mesylate). A white, slightly hygroscopic, crystalline powder. Very soluble in water; freely soluble in alcohol; very slightly soluble in ether. Store in airtight containers.

Adverse Effects, Treatment, and Precautions

As for Atropine Sulfate, p.1219. Drowsiness may be severe in some patients and patients so affected should not drive or operate machinery. Mental disturbances and excitement may occur with large doses or in susceptible patients.

Abuse. For mention of abuse of benzatropine see under Trihexyphenidyl Hydrochloride, p.820.

Effects on the heart. Paradoxical sinus bradycardia in a patient with depression and psychotic symptoms was attributed to benzatropine since it persisted despite modification to other treatment and resolved only when benzatropine was withdrawn.[1]

1. Voinov H, *et al.* Sinus bradycardia related to the use of benztropine mesylate. *Am J Psychiatry* 1992; **149:** 711.

Interactions

As for antimuscarinics in general (see Atropine Sulfate, p.1220).

Antidepressants. A report[1] of 5 patients who developed delirium while taking an antipsychotic, an *SSRI*, and benzatropine suggested that there might be an interaction between SSRIs and benzatropine.

1. Roth A, *et al.* Delirium associated with the combination of a neuroleptic, an SSRI, and benztropine. *J Clin Psychiatry* 1994; **55:** 492–5.

Antipsychotics. Fatal heat stroke after exposure to an ambient temperature of over 29° has been reported[1,2] in patients receiving benzatropine with antipsychotics. Paralytic ileus, sometimes fatal, has also been seen in patients taking benzatropine with antipsychotics.[3]

1. Stadnyk AN, Glezos JD. Drug-induced heat stroke. *Can Med Assoc J* 1983; **128:** 957–9.
2. Tyndel F, Labonté R. Drug-facilitated heat stroke. *Can Med Assoc J* 1983; **129:** 680.
3. Wade LC, Ellenor GL. Combination mesoridazine- and benztropine mesylate-induced paralytic ileus: two case reports. *Drug Intell Clin Pharm* 1980; **14:** 17–22.

Uses and Administration

Benzatropine mesilate is a tertiary amine antimuscarinic with actions and uses similar to those of trihexyphenidyl (p.820); it also has antihistaminic properties.

Benzatropine is used for the symptomatic treatment of parkinsonism (p.791), including the alleviation of the extrapyramidal syndrome induced by drugs such as phenothiazines, but, like other antimuscarinics, is of no value against tardive dyskinesias. It has been used in the treatment of dystonias (see under Uses and Administration of Levodopa, p.809).

Benzatropine mesilate is given orally or, if necessary, by intramuscular or intravenous injection.

In idiopathic **parkinsonism** benzatropine mesilate is usually given orally in an initial daily dose of 0.5 to 1 mg at bedtime. Its actions are cumulative, and may not be manifest for several days after beginning therapy. Patients with post-encephalitic parkinsonism often tolerate an initial daily dose of 2 mg. The dose may be gradually increased by 500 micrograms every 5 to 6 days to a maximum of 6 mg daily until the optimum dose is reached. Maintenance therapy may be given as a single daily dose at bedtime or in divided doses 2 to 4 times daily.

In the management of **drug-induced extrapyramidal symptoms** doses of 1 to 4 mg once or twice daily have been given orally or parenterally. Therapy may be withdrawn after 1 to 2 weeks to assess whether it is still necessary.

In an emergency, benzatropine mesilate may be injected intramuscularly or intravenously in a dose of 1 to 2 mg; intramuscular injection is reported to produce an effect as quickly as intravenous dosage so the latter is rarely necessary.

For management of **dystonias in children**, the *BNFC* suggests that in an emergency, single doses of 20 to 100 micrograms/kg (maximum of 2 mg) may be given by intravenous or intramuscular injection to children aged 3 to 12 years, and 1 to 2 mg to those aged 12 to 18 years.

Benzatropine has also been given as the hydrochloride.

Preparations

BP 2008: Benzatropine Injection; Benzatropine Tablets;
USP 31: Benztropine Mesylate Injection; Benztropine Mesylate Tablets.

Proprietary Preparations (details are given in Part 3)
Austral.: Benztrop; Cogentin; ***Austria:*** Cogentin; ***Canad.:*** Cogentin†; ***Cz.:*** Apo-Benztropine; ***Denm.:*** Cogentin†; ***Hong Kong:*** Cogentin; ***Irl.:*** Cogentin†; ***Norw.:*** Cogentin†; ***NZ:*** Benztrop; Cogentin; ***Port.:*** Cogentin†; ***Thai.:*** Cogentin; ***UK:*** Cogentin; ***USA:*** Cogentin.

Biperiden *(BAN, rINN)*

Biperideeni; Bipéridène; Biperideno; Biperidenum. 1-(Bicyclo-[2.2.1]hept-5-en-2-yl)-1-phenyl-3-piperidinopropan-1-ol.

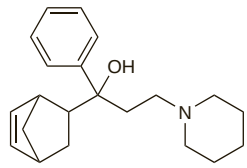
Бипериден

$C_{21}H_{29}NO$ = 311.5.

CAS — 514-65-8.

ATC — N04AA02.

ATC Vet — QN04AA02.



Pharmacopoeias. In *Int.* and *US*.

USP 31 (Biperiden). A white, practically odourless, crystalline powder. Practically insoluble in water; sparingly soluble in alcohol; freely soluble in chloroform. Protect from light.

Biperiden Hydrochloride *(BANM, rINNM)*

Biperideenihydrokloridi; Biperiden Hidroklorür; Bipéridène, chlorhydrate de; Biperidén-hidroklorid; Biperiden-hydrochlorid; Biperidenhydroklorid; Biperideni hydrochloridum; Biperideno hidrochloridas; Hidrocloruro de biperideno.

Бипериден Гидрохлорид

$C_{21}H_{29}NO,HCl$ = 347.9.

CAS — 1235-82-1.

ATC — N04AA02.

ATC Vet — QN04AA02.

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

Ph. Eur. 6.2 (Biperiden Hydrochloride). A white or almost white, crystalline powder. Slightly soluble in water and in alcohol; very slightly soluble in dichloromethane. A 0.2% solution in water has a pH of 5.0 to 6.5. Store in airtight containers. Protect from light.

USP 31 (Biperiden Hydrochloride). A white, practically odourless, crystalline powder. Slightly soluble in water, in alcohol, in chloroform, and in ether; sparingly soluble in methyl alcohol. Protect from light.

Biperiden Lactate *(BANM, rINNM)*

Biperiden Laktat; Bipéridène, Lactate de; Biperideni Lactas; Lactato de biperideno.

Бипериден Лактат

$C_{21}H_{29}NO,C_3H_6O_3$ = 401.5.

CAS — 7085-45-2.

ATC — N04AA02.

ATC Vet — QN04AA02.

Pharmacopoeias. *US* includes Biperiden Lactate Injection.

Adverse Effects, Treatment, and Precautions

As for Atropine Sulfate, p.1219.

Parenteral use may be followed by slight transient hypotension.

Abuse. Abuse of biperiden has been reported in psychiatric patients.[1]

1. Pullen GP, *et al.* Anticholinergic drug abuse: a common problem? *BMJ* 1984; **289**: 612–13.

Interactions

As for antimuscarinics in general (see Atropine Sulfate, p.1220).

Pharmacokinetics

Biperiden is readily absorbed from the gastrointestinal tract, but bioavailability is only about 30% suggesting that it undergoes extensive first-pass metabolism. Biperiden has an elimination half-life of about 20 hours.

◊ References.

1. Hollmann M, *et al.* Biperiden effects and plasma levels in volunteers. *Eur J Clin Pharmacol* 1984; **27:** 619–21.
2. Grimaldi R, *et al.* Pharmacokinetic and pharmacodynamic studies following the intravenous and oral administration of the antiparkinsonian drug biperiden to normal subjects. *Eur J Clin Pharmacol* 1986; **29:** 735–7.

Uses and Administration

Biperiden is a tertiary amine antimuscarinic with actions and uses similar to those of trihexyphenidyl (p.820) but with more potent antinicotinic properties.

Biperiden is used in the symptomatic treatment of parkinsonism (p.791), including the alleviation of the extrapyramidal syndrome induced by drugs such as phenothiazines, but, like other antimuscarinics, is of no value against tardive dyskinesias.

Biperiden is given orally as the hydrochloride and by injection as the lactate; doses are expressed in terms of the relevant salt. The initial oral dose for **Parkinson's disease** is 2 mg of the hydrochloride three or four times daily increased according to response to a maximum of 16 mg daily. The dose for **drug-induced extrapyramidal symptoms** is 2 mg of the hydrochloride orally one to three times daily; alternatively, 2 mg of biperiden lactate may be given by intramuscular or slow intravenous injection and repeated every 30 minutes if needed up to a maximum of 4 doses in 24 hours.

Preparations

USP 31: Biperiden Hydrochloride Tablets; Biperiden Lactate Injection.

Proprietary Preparations (details are given in Part 3)
Arg.: Akineton; Berofin; Darcipireno; Sinekin; ***Austral.:*** Akineton; ***Austria:*** Akineton; ***Belg.:*** Akineton; ***Braz.:*** Akineton; Cinetol; Parkinsol; ***Canad.:*** Akineton; ***Chile:*** Akineton; ***Cz.:*** Akineton; ***Denm.:*** Akineton; ***Fin.:*** Akineton; Ipsatol; ***Fr.:*** Akineton; ***Ger.:*** Akineton; Norakin N†; ***Gr.:*** Akineton; ***Hung.:*** Akineton; ***India:*** Dyskinon†; ***Irl.:*** Akineton; ***Israel:*** Dekinet; ***Ital.:*** Akineton; ***Mex.:*** Akineton; Bikipen; Kinex; ***Neth.:*** Akineton; ***Norw.:*** Akineton†; ***Philipp.:*** Akineton; ***Pol.:*** Akineton; ***Port.:*** Akineton; ***Rus.:*** Akineton (Акинетон)†; ***S.Afr.:*** Akineton; ***Spain:*** Akineton; ***Swed.:*** Akineton†; ***Switz.:*** Akineton; ***Turk.:*** Akineton; ***USA:*** Akineton; ***Venez.:*** Akineton.

Bornaprine Hydrochloride *(BANM, rINNM)*

Bornaprin Hidroklorür; Bornaprine, Chlorhydrate de; Bornaprini Hydrochloridum; Hidrocloruro de bornaprina. 3-Diethylaminopropyl 2-phenylbicyclo[2.2.1]heptane-2-carboxylate hydrochloride.

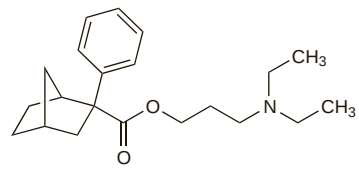
Борнаприна Гидрохлорид

$C_{21}H_{31}NO_2,HCl$ = 365.9.

CAS — 20448-86-6 (bornaprine); 26908-91-8 (bornaprine hydrochloride).

ATC — N04AA11.

ATC Vet — QN04AA11.



(bornaprine)

Profile

Bornaprine hydrochloride is a quaternary ammonium antimuscarinic with actions and uses similar to those of trihexyphenidyl (p.820). It is used in the symptomatic treatment of **parkinsonism** (p.791), including the alleviation of the extrapyramidal syndrome induced by drugs such as phenothiazines, but, like other antimuscarinics, is of no value against tardive dyskinesias; it is