daily dose is 150 mg taken as a single dose 1 hour before starting work. Reduced doses are recommended in the elderly and in patients with severe hepatic impairment.

◊ References.

1. Harsh JR, *et al.* The efficacy and safety of armodafinil as treatment for adults with excessive sleepiness associated with narcolepsy. *Curr Med Res Opin* 2006; **22:** 761–74.
2. Roth T, *et al.* Effects of armodafinil in the treatment of residual excessive sleepiness associated with obstructive sleep apnea/hypopnea syndrome: a 12-week, multicenter, double-blind, randomized, placebo-controlled study in nCPAP-adherent adults. *Clin Ther* 2006; **28:** 689–706.
3. Hirshkowitz M, *et al.* Adjunct armodafinil improves wakefulness and memory in obstructive sleep apnea/hypopnea syndrome. *Respir Med* 2007; **101:** 616–27.

Preparations

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

Atomoxetine Hydrochloride *(BANM, USAN, rINNM)*

Atomoxétine, Chlorhydrate d'; Atomoxetini Hydrochloridum; Hidrocloruro de tomoxetina; LY-135252; LY-139602; LY-139603; Tomoxetine Hydrochloride. (–)-*N*-Methyl-γ-(2-methylphenoxy)-benzenepropanamine hydrochloride.

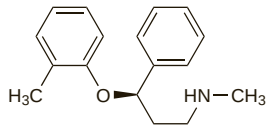
Томоксетина Гидрохлорид

$C_{17}H_{21}NO,HCl$ = 291.8.

CAS — 83015-26-3 (atomoxetine); 82248-59-7 (atomoxetine hydrochloride).

ATC — N06BA09.

ATC Vet — QN06BA09.



(atomoxetine)

Adverse Effects and Precautions

Adverse effects reported in patients receiving atomoxetine include dyspepsia and other gastrointestinal disturbances, anorexia and weight loss, fatigue, sleep disturbances, dizziness, irritability and emotional lability, cough, sinusitis or rhinorrhoea, urinary hesitancy or retention, decreased libido and sexual dysfunction, priapism, skin rashes, increased sweating, and hot flushes. Suicidal behaviour has been reported in children (see Effects on Mental State, below). Hypersensitivity reactions have occurred rarely. There have also been rare reports of severe hepatotoxicity (see Effects on the Liver, below).

There may be increases in blood pressure and heart rate, and atomoxetine should be given with caution to patients with hypertension, tachycardia, or cardiovascular or cerebrovascular disease (see also Effects on the Cardiovascular System, under Dexamfetamine Sulfate, p.2153). Orthostatic hypotension and syncope have also been reported. QT prolongation has been associated with atomoxetine therapy and it should be used with caution in patients with known or suspected prolonged QT interval. Use with drugs liable to inhibit the cytochrome P450 isoenzyme CYP2D6, other drugs that may prolong the QT interval, or drugs likely to cause electrolyte imbalance may also increase the risk (see Interactions, below). Sudden death, stroke, and myocardial infarction have also been reported in patients given atomoxetine. All patients should be assessed for cardiovascular disease before treatment is started. Atomoxetine should generally not be used in those with known serious structural cardiac abnormalities, cardiomyopathy, serious heart rhythm abnormalities, or other serious cardiac problems

Seizures are a potential risk with atomoxetine therapy and it should be used with caution in patients with a history of seizures; treatment may need to be stopped in those who develop seizures or have an increase in seizure frequency.

Atomoxetine is contra-indicated in patients with angle-closure glaucoma as it may increase the risk of mydriasis.

The weight and height gain of children receiving atomoxetine has been reported to lag behind that of the predicted norm for about the first 9 to 12 months of treatment and generally normalises within about 3 years of treatment; licensed product information recommends that growth be monitored and consideration given to dose reduction or interrupting treatment in patients who are not growing or gaining weight satisfactorily.

Poor metabolisers of atomoxetine (see Pharmacokinetics, below) may have an increased risk of adverse reactions.

Effects on the liver. In December 2004, the US manufacturer stated that 2 cases of severe hepatotoxicity had been reported with atomoxetine treatment since the drug was launched in 2002.[1] In both cases the patients recovered; however, because of the risk of acute hepatic failure resulting in death or the need for transplantation, it was recommended that atomoxetine should be permanently discontinued in patients with jaundice or markedly increased liver enzyme values.

Similar advice has also been issued in the UK by the CSM.[2] Up to February 2005, the CSM were aware of 3 reports of hepatic disorders (one each of hepatitis, jaundice, and increased bilirubin) with atomoxetine treatment in the UK; a total of 41 reports of hepatic disorders had been received worldwide.

1. Eisenberg P. Safety data on Strattera (atomoxetine hydrochloride)–hepatic effects. Available at: http://www.strattera.com/pdf/dear_hcp.pdf (accessed 31/01/05)
2. MHRA. Strattera (atomoxetine)–risk of hepatic disorders. Message from Professor G Duff, Chairman of Committee on Safety of Medicines (issued 02/02/05). Available at: http://www.mhra.gov.uk/home/idcplg?IdcService=GET_FILE&dDocName=CON019459&RevisionSelectionMethod=LatestReleased (accessed 08/08/08)

Effects on mental state. Irritability and mood swings have been reported with the use of atomoxetine in children. In an observational study of 153 children, irritability, aggression, mania, or hypomania were associated with atomoxetine in 51 cases. Of these, 31 had a family history, and 41 a personal history, of mood disorders; 27 had both and 6 had neither. The authors therefore recommended that all patients receiving atomoxetine should be monitored closely.[1]

Subsequently, the FDA[2] requested the US manufacturer (*Eli Lilly*) to conduct an analysis of adverse events from 12 clinical trials involving 2208 patients. The risk of suicidal behaviour during the first few months of treatment was found to be 0.4% in children receiving atomoxetine compared with no risk in those on placebo; no suicides were reported although there was one unsuccessful suicide attempt in the atomoxetine-treated group. This finding resulted in the inclusion of a warning in US labelling about the increased risk of suicidal ideation in children and adolescents being treated with atomoxetine; it was also recommended that changes in behaviour must be closely monitored, particularly during the initial months of therapy or when the dose is changed. Similar warnings have been issued by the UK CSM[3] and regulatory authorities in other countries.[4]

In light of these concerns a review of available data in Europe was conducted; it was considered that the overall risk to benefit ratio of atomoxetine in children remained favourable.[5]

1. Henderson TA, Hartman K. Aggression, mania, and hypomania induction associated with atomoxetine. *Pediatrics* 2004; **114:** 895–6.
2. FDA. FDA alert for healthcare professionals: atomoxetine (marketed as Strattera)–suicidal thinking in children and adolescents (issued 29th September, 2005). Available at: http://www.fda.gov/cder/drug/InfoSheets/HCP/atomoxetinehcp.pdf (accessed 19/04/06)
3. MHRA. Strattera (atomoxetine): risk of suicidal thoughts/behaviour. Message from Professor G Duff, Chairman of Committee on Safety of Medicines (issued 29th September, 2005). Available at: http://www.mhra.gov.uk/home/groups/pl-p/documents/websiteresources/con2018039.pdf (accessed 11/08/08)
4. Lilly, Canada. WARNING for atomoxetine regarding the potential for behavioural and emotional changes, including risk of self-harm (issued 28th September 2005). Available at: http://www.hc-sc.gc.ca/dhp-mps/alt_formats/hpfb-dgpsa/pdf/mede-ff/strattera_hpc-cps-eng.pdf (accessed 08/08/08)
5. MHRA. Updated warnings on the attention deficit hyperactivity disorder drug Strattera: information for healthcare professionals. Message from Professor G Duff, Chairman of Commission on Human Medicines (issued 16th February, 2006). Available at: http://www.mhra.gov.uk/home/idcplg?IdcService=SS_GET_PAGE&useSecondary=true&ssDocName=CON2023222&ssTargetNodeId=221 (accessed 19/04/06)

Handling. Atomoxetine is an ocular irritant and atomoxetine capsules should not therefore be opened. If the capsule contents do accidentally come into contact with the eye, the affected eye must be flushed immediately with water; hands and other potentially contaminated surfaces should be washed as soon as possible.

Interactions

Atomoxetine should not be taken with an MAOI, or within 2 weeks of stopping MAOI therapy, nor should MAOI therapy be started for 2 weeks after stopping atomoxetine. Care should be taken if given with other drugs that raise blood pressure, because of a possible additive affect; the actions of salbutamol on the cardiovascular system may be potentiated. In addition, there is a risk of cardiac events in patients receiving atomoxetine who are also taking drugs that affect cardiac conduction or electrolyte balance, or that inhibit the cytochrome P450 isoenzyme CYP2D6 (see below).

Seizures have been noted with atomoxetine and caution is advised when used with drugs known to lower the seizure threshold.

Atomoxetine is metabolised via the isoenzyme CYP2D6 and inhibitors of this enzyme such as paroxetine, fluoxetine, and quinidine may increase plasma concentrations of atomoxetine in extensive, but not poor, metabolisers.

Antidepressants. *Paroxetine* was found to inhibit atomoxetine's metabolism by cytochrome P450 isoenzyme CYP2D6 in extensive metabolisers resulting in pharmacokinetics for atomoxetine similar to those in poor metabolisers.[1]

1. Belle DJ, *et al.* Effect of potent CYP2D6 inhibition by paroxetine on atomoxetine pharmacokinetics. *J Clin Pharmacol* 2002; **42:** 1219–27.

Pharmacokinetics

Atomoxetine is well absorbed after oral doses, with peak plasma concentrations being achieved 1 to 2 hours later. Bioavailability is about 94% in poor metabolisers but only 63% in extensive metabolisers. Atomoxetine is about 98% bound to plasma proteins. Atomoxetine is metabolised primarily via the cytochrome P450 isoenzyme CYP2D6 to the active metabolite 4-hydroxyatomoxetine; a minority of the population are poor metabolisers and experience plasma concentrations about 5 times those in extensive metabolisers. It is excreted in the urine as glucuronide metabolites and a small amount of unchanged drug; less than 17% of a dose is excreted in the faeces. The half-life of atomoxetine is about 5.2 hours in extensive and 21.6 hours in poor metabolisers.

◊ References.

1. Sauer J-M, *et al.* Clinical pharmacokinetics of atomoxetine. *Clin Pharmacokinet* 2005; **44:** 571–90.
2. Cui YM, *et al.* Atomoxetine pharmacokinetics in healthy Chinese subjects and effect of the CYP2D6*10 allele. *Br J Clin Pharmacol* 2007; **64:** 445–9.

Uses and Administration

Atomoxetine hydrochloride is a selective noradrenaline reuptake inhibitor used in the treatment of attention deficit hyperactivity disorder (p.2148) in adults and children aged 6 years and over. It is given as the hydrochloride although doses are expressed in terms of the base; atomoxetine hydrochloride 11.4 mg is equivalent to about 10 mg of atomoxetine.

In *adults and adolescents and children weighing over 70 kg*, the initial dose is the equivalent of 40 mg daily, gradually increased after at least 7 days to 80 mg daily; in the USA an increase in dose may be made after a minimum of 3 days. A further increase to a maximum of 100 mg daily may be made after 2 to 4 weeks. In *children and adolescents of 70 kg and under*, the initial dose is the equivalent of about 500 micrograms/kg daily; this may be gradually increased to about 1.2 mg/kg daily. The total daily dose in this group should not exceed 1.4 mg/kg or 100 mg, whichever is less. Doses may be given as either a single dose in the morning or as equally divided doses in the morning and late afternoon or early evening.

Reduced doses are recommended in patients with hepatic impairment, see below. A lower initial dose and slower titration of atomoxetine may be required in patients who are poor CYP2D6 metabolisers (see Adverse Effects and Precautions, and Pharmacokinetics, above) and in those also taking CYP2D6 inhibitors (see Interactions, above). US licensed product information recommends increasing to the usual target dose only if symptoms fail to improve after 4 weeks and the initial dose is well tolerated in these patients.

◊ References.

1. Michelson D, *et al.* Atomoxetine in the treatment of children and adolescents with attention-deficit/hyperactivity disorder: a randomized, placebo-controlled, dose-response study. Abstract: *Pediatrics* 2001; **108:** 1197. Full version: http://pediatrics.aappublications.org/cgi/content/full/108/5/e83 (accessed 15/04/04)
2. Simpson D, Plosker GL. Atomoxetine: a review of its use in adults with attention deficit hyperactivity disorder. *Drugs* 2004; **64:** 205–22.
3. Kelsey DK, *et al.* Once-daily atomoxetine treatment for children with attention-deficit/hyperactivity disorder, including an assessment of evening and morning behavior: a double-blind, placebo-controlled trial. Abstract: *Pediatrics* 2004; **114:** 240. Full version: http://pediatrics.aappublications.org/cgi/reprint/114/1/e1 (accessed 14/05/08)
4. Eiland LS, Guest AL. Atomoxetine treatment of attention-deficit/hyperactivity disorder. *Ann Pharmacother* 2004; **38:** 86–90.
5. Corman SL, *et al.* Atomoxetine: the first nonstimulant for the management of attention-deficit/hyperactivity disorder. *Am J Health-Syst Pharm* 2004; **61:** 2391–9.
6. Barton J. Atomoxetine: a new pharmacotherapeutic approach in the management of attention deficit/hyperactivity disorder. *Arch Dis Child* 2005; **90** (Suppl 1): i26–i29.
7. Gibson AP, *et al.* Atomoxetine versus stimulants for treatment of attention deficit/hyperactivity disorder. *Ann Pharmacother* 2006; **40:** 1134–41.
8. Wilens TE, *et al.* Long-term atomoxetine treatment in adolescents with attention-deficit/hyperactivity disorder. *J Pediatr* 2006; **149:** 112–19.
9. Newcorn JH, *et al.* Low-dose atomoxetine for maintenance treatment of attention-deficit/hyperactivity disorder. Abstract: *Pediatrics* 2006; **118:** 2527. Full version: http://pediatrics.aappublications.org/cgi/reprint/118/6/e1701 (accessed 14/05/08)

Administration in hepatic impairment. In patients with moderate hepatic impairment the dose of atomoxetine (see above) should be reduced by 50%, while in those with severe impairment it should be reduced by 75%.

References.

1. Chalon SA, *et al.* Effect of hepatic impairment on the pharmacokinetics of atomoxetine and its metabolites. *Clin Pharmacol Ther* 2003; **73:** 178–91.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Recit; Strattera; ***Austral.:*** Strattera; ***Belg.:*** Strattera; ***Canad.:*** Strattera; ***Chile:*** Deaten; Strattera; ***Cz.:*** Strattera; ***Ger.:*** Strattera; ***Gr.:*** Strattera; ***Hong Kong:*** Strattera; ***Malaysia:*** Strattera; ***Mex.:*** Strattera; ***Neth.:*** Strattera; ***Norw.:*** Strattera; ***NZ:*** Strattera; ***Philipp.:*** Strattera; ***Port.:*** Strattera; ***Rus.:*** Strattera (Страттера); ***S.Afr.:*** Strattera; ***Singapore:*** Strattera; ***Thai.:*** Strattera; ***UK:*** Strattera; ***USA:*** Strattera.

Bemegride *(BAN, rINN)*

Bemegrida; Bémégride; Bemegridum. 3-Ethyl-3-methylglutarimide; 4-Ethyl-4-methylpiperidine-2,6-dione.

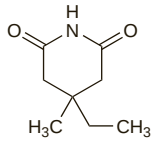
Бемегрид

$C_8H_{13}NO_2$ = 155.2.

CAS — 64-65-3.

ATC — R07AB05.

ATC Vet — QR07AB05.



Profile

Bemegride has properties similar to those of doxapram (p.2155). It has been given intravenously as a respiratory stimulant.

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

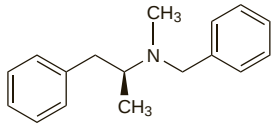
Benzfetamine Hydrochloride *(BANM, rINNM)* ⊗

Benzfétamine, Chlorhydrate de; Benzfetamini Hydrochloridum; Benzphetamine Hydrochloride; Hidrocloruro de benzfetamina. (+)-*N*-Benzyl-*N*,α-dimethylphenethylamine hydrochloride.

Бензфетамина Гидрохлорид

$C_{17}H_{21}N,HCl$ = 275.8.

CAS — 156-08-1 (benzfetamine); 5411-22-3 (benzfetamine hydrochloride).



(benzfetamine)

Profile

Benzfetamine hydrochloride is a central stimulant and sympathomimetic with properties similar to those of dexamfetamine (below). It has been used as an anorectic in the treatment of obesity (p.2149), although amfetamines are no longer recommended for this indication. The usual initial oral dose is 25 to 50 mg given once daily, subsequently adjusted, according to requirements, to a dose of 25 to 50 mg up to three times daily.

Preparations

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

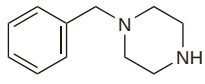
Benzylpiperazine ⊗

N-Benzylpiperazine; 1-Benzylpiperazine. 1-(Phenylmethyl)piperazine.

Бензилпиперазин

$C_{11}H_{16}N_2$ = 176.3.

CAS — 2759-28-6.



NOTE. The following terms have been used as 'street names' (see p.vi) or slang names for various forms of benzylpiperazine:
A2; Blast; BZP; Charge; Charlie; Cosmic Kelly; ESP; Euphoria; Exodus; Frenzy; Legal E; Legal X; Nemesis; Pep; Pep Love; Pep Stoned; Pep Twisted; Rapture; The Good Stuff.

Profile

Benzylpiperazine is reported to produce CNS stimulant effects similar to those of the amfetamines (see Dexamfetamine, p.2153) and is subject to abuse.

Other piperazine derivatives subject to abuse include:

- 1-(3,4-methylenedioxybenzyl)piperazine (MDBP)
- 1-(3-trifluoromethylphenyl)piperazine (TFMPP)
- 1-(3-chlorophenyl)piperazine (mCPP)
- 1-(4-methoxyphenyl)piperazine (MeOPP)

◊ References.

1. Gee P, *et al.* Toxic effects of BZP-based herbal party pills in humans: a prospective study in Christchurch, New Zealand. *N Z Med J* 2005; **118:** U1784.
2. Staack RF. Piperazine designer drugs of abuse. *Lancet* 2007; **369:** 1411–13.
3. Johnstone AC, *et al.* Benzylpiperazine: a drug of abuse? *J Psychopharmacol* 2007; **21:** 888–94.
4. Wood DM, *et al.* Collapse, reported seizure—and an unexpected pill. *Lancet* 2007; **369:** 1490.

Catha ⊗

Abyssinian, African, or Arabian Tea; Kat; Kath; Khat; Miraa; Qat; Somali Tea.

Description. Catha consists of the leaves of *Catha edulis* (Celastraceae), and contains cathine, cathinone, celastrin, choline, tannins, and inorganic salts.
The following terms have been used as 'street names' (see p.vi) or slang names for various forms of catha:
Cat; Chat; Feline; Kat; Miraa; Pootie; Qat; Quaadka.

Cathine *(pINN)* ⊗

Cathinum; Catina; (+)-Norpseudoephedrine. *threo*-2-Amino-1-phenylpropan-1-ol.

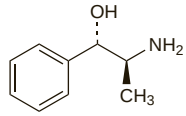
Катин

$C_9H_{13}NO$ = 151.2.

CAS — 492-39-7; 36393-56-3.

ATC — A08AA07.

ATC Vet — QA08AA07.



Cathinone *(pINN)* ⊗

Cathinonum; Catinona. (*S*)-2-Aminopropiophenone.

Катинон

$C_9H_{11}NO$ = 149.2.

CAS — 71031-15-7.

Profile

Catha, the leaves of *Catha edulis* (Celastraceae), is used for its stimulant properties among some cultures of Africa and the Middle East, usually by chewing the leaves. Its effects are reported to resemble those of the amfetamines (see Dexamfetamine Sulfate, below), and are thought to be largely due to the content of cathinone. Dependence and psychotic reactions have been reported. Cathine, another constituent, has been used as the hydrochloride as an anorectic.

◊ References to the pharmacology and pharmacokinetics of catha and its constituents[1-8] and reports of adverse effects.[9-15]

1. Brenneisen R, *et al.* Metabolism of cathinone to (–)-norephedrine and (–)-norpseudoephedrine. *J Pharm Pharmacol* 1986; **38:** 298–300.
2. Brenneisen R, *et al.* Amphetamine-like effects in humans of the khat alkaloid cathinone. *Br J Clin Pharmacol* 1990; **30:** 825–8.
3. Kalix P. Pharmacological properties of the stimulant khat. *Pharmacol Ther* 1990; **48:** 397–416.
4. Kalix P. Chewing khat, an old drug habit that is new in Europe. *Int J Risk Safety Med* 1992; **3:** 143–56.
5. Kalix P. Cathinone, a natural amphetamine. *Pharmacol Toxicol* 1992; **70:** 77–86.
6. Widler P, *et al.* Pharmacodynamics and pharmacokinetics of khat: a controlled study. *Clin Pharmacol Ther* 1994; **55:** 556–62.
7. Kalix P. Catha edulis, a plant that has amphetamine effects. *Pharm World Sci* 1996; **18:** 69–73.
8. Toennes SW, *et al.* Pharmacokinetics of cathinone, cathine and norephedrine after the chewing of khat leaves. *Br J Clin Pharmacol* 2003; **56:** 125–30.
9. Rumpf KW, *et al.* Rhabdomyolysis after ingestion of an appetite suppressant. *JAMA* 1983; **250:** 2112.
10. Gough SP, Cookson IB. Khat-induced schizophreniform psychosis in UK. *Lancet* 1984; **i:** 455.
11. Roper JP. The presumed neurotoxic effects of Catha edulis—an exotic plant now available in the United Kingdom. *Br J Ophthalmol* 1986; **70:** 779–81.
12. Zureikat N, *et al.* Chewing khat slows the orocaecal transit time. *Gut* 1992; **33** (suppl): S23.
13. Yousef G, *et al.* Khat chewing as a cause of psychosis. *Br J Hosp Med* 1995; **54:** 322–6.
14. Al-Motarreb A, *et al.* Khat chewing and acute myocardial infarction. *Heart* 2002; **87:** 279–280.
15. Al-Motarreb A, *et al.* Khat chewing is a risk factor for acute myocardial infarction: a case-control study. *Br J Clin Pharmacol* 2005; **59:** 574–81.

Preparations

Proprietary Preparations (details are given in Part 3)
Ger.: Antiadipositum X-112 T; ***S.Afr.:*** Dietene; Eetless; Leanor; Nobese No. 1; Slim 'n Trim; Thinz; ***Switz.:*** Antiadipositum X-112; Belloform†; Limit-X†; Miniscap†; ***Thai.:*** Mirapront N†.

Multi-ingredient: ***Mex.:*** Redotex; Redotex NF.

Clobenzorex Hydrochloride *(rINNM)* ⊗

Clobenzorex, Chlorhydrate de; Clobenzorexi Hydrochloridum; Hidrocloruro de clobenzorex; SD-271-12. (+)-*N*-(2-Chlorobenzyl)-α-methylphenethylamine hydrochloride.

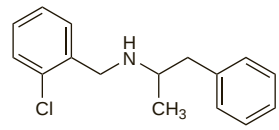
Клобензорекса Гидрохлорид

$C_{16}H_{18}ClN,HCl$ = 296.2.

CAS — 13364-32-4 (clobenzorex); 5843-53-8 (clobenzorex hydrochloride).

ATC — A08AA08.

ATC Vet — QA08AA08.



(clobenzorex)

NOTE. The following terms have been used as 'street names' (see p.vi) or slang names for various forms of clobenzorex:
Dinintels.

Profile

Clobenzorex hydrochloride is a central stimulant and sympathomimetic with properties similar to those of dexamfetamine (below). It has been used as an anorectic in the treatment of obesity (p.2149) but regulatory authorities in the EU have called for the withdrawal of all anorectics from the market (see under Effects on the Cardiovascular System in Fenfluramine, p.2156).

Preparations

Proprietary Preparations (details are given in Part 3)
Mex.: Asenlix; Itravil; Obeclox; Redicres.

Deanol *(BAN)* ⊗

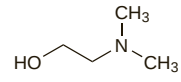
Démanol. 2-Dimethylaminoethanol.

$C_4H_{11}NO$ = 89.14.

CAS — 108-01-0 (deanol); 3342-61-8 (deanol aceglumate); 3635-74-3 (deanol acetamidobenzoate); 968-46-7 (deanol benzilate); 71-79-4 (deanol benzilate hydrochloride); 15585-86-1 (deanol cyclohexylpropionate); 5988-51-2 (deanol tartrate).

ATC — N06BX04.

ATC Vet — QN06BX04.



NOTE. Deanol Aceglumate is *pINN*.

Profile

Deanol, a precursor of choline, may enhance central acetylcholine formation. It has been used as a central stimulant in the treatment of hyperactivity in children but its efficacy is not proven. It has been included in preparations used as tonics and for the management of impaired mental function.

It has been used as a variety of salts and esters including deanol aceglumate, deanol acetamidobenzoate, deanol bisorcate, deanol cyclohexylpropionate (cyprodenate; cyprodemanol), deanol hemisuccinate, deanol pidolate, and deanol tartrate. Deanol benzilate (deanol diphenylglycolate; benzacine) has been used as the hydrochloride in antispasmodic preparations.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: DM Active; ***Belg.:*** Actebral†; ***Fr.:*** Astyl†; ***Ger.:*** Risataran; ***Ital.:*** Rischiani†; ***Pol.:*** Bimanol; ***Port.:*** Tonibral†; ***Rus.:*** Nooclerin (Ноокперин).

Multi-ingredient: ***Fr.:*** Acti 5; Debrumyl; ***Ger.:*** Rowachol comp†; ***Port.:*** Actilam; Debrumyl; Forticol; Tonice; ***Spain:*** Anti Anorex Triple; Denubil; ***Switz.:*** Vigoran†.