2. Shire, Canada. New Safety information regarding Permax and occurrence of cardiac valvulopathy/fibrosis: update on the use of Permax (pergolide mesylate) (issued 12/10/04). Available at: http://www.hc-sc.gc.ca/dhp-mps/alt_formats/hpfb-dgpsa/pdf/medeff/shire_permax_2_hpc-cps-eng.pdf (accessed 12/08/08)

3. EMEA. EMEA recommends new warnings and contraindications for ergot-derived dopamine agonists (issued 26th June, 2008). Available at: http://www.emea.europa.eu/pdfs/human/press/pr/32239508en.pdf (accessed 08/08/08)

4. MHRA. Dopamine agonists for Parkinson's disease (issued 30th March 2007). Available at: http://www.mhra.gov.uk/Safetyinformation/Generalsafetyinformationandadvice/Product-specificinformationandadvice/DopamineagonistsforParkinson146sdisease/CON2030729 (accessed 03/06/08)

5. Schade R, *et al.* Dopamine agonists and the risk of cardiac-valve regurgitation. *N Engl J Med* 2007; **356:** 29–38.

6. Zanettini R, *et al.* Valvular heart disease and the use of dopamine agonists for Parkinson's disease. *N Engl J Med* 2007; **356:** 39–46.

7. FDA. FDA public health advisory: pergolide (marketed as Permax) (issued 29th March 2007). Available at: http://www.fda.gov/cder/drug/advisory/pergolide.htm (accessed 03/06/08)

8. Eli Lilly, Canada. Health Canada-mandated important safety information on Permax (pergolide mesylate) (issued 10th August 2007). Available at: http://www.lilly.ca/searchable/media/hcp/permax.pdf (accessed 25/06/08)

Interactions

As for Bromocriptine, p.800.

Pharmacokinetics

Pergolide mesilate is absorbed from the gastrointestinal tract. It is reported to be about 90% bound to plasma proteins. It is excreted mainly in the urine in the form of metabolites.

◊ References.

1. Blin O. The pharmacokinetics of pergolide in Parkinson's disease. *Curr Opin Neurol* 2003; **16** (suppl 1): S9–S12.

Uses and Administration

Pergolide mesilate, an ergot derivative, is a dopamine D_2-agonist with actions and uses similar to those of bromocriptine (p.800), but in contrast to bromocriptine (a dopamine D_2-agonist) it also has agonist properties at D_1 and D_3 receptors. Pergolide is used in the management of Parkinson's disease as monotherapy, or as an adjunct to levodopa therapy to reduce 'end-of-dose' or 'on-off' fluctuations in response; in the UK pergolide is restricted to patients who are intolerant of, or who fail to respond to, non-ergot drug treatment. Pergolide is given by mouth as the mesilate with doses expressed as the base. Pergolide mesilate 65.3 mg is equivalent to about 50 mg pergolide.

For use as *monotherapy* an initial dose equivalent to 50 micrograms of pergolide is given on the first evening of therapy. The dose is thereafter gradually increased: 50 micrograms twice daily is taken on days 2 to 4, then increased by 100 to 250 micrograms every 3 or 4 days, given in 3 divided doses, up to a daily dose of 1.5 mg at day 28. After day 30, the dose should be increased further by a maximum of 250 micrograms twice a week until an optimum response is achieved. Usual maintenance doses are 2.1 to 2.5 mg daily; doses above 3 mg daily are not recommended by the EMEA. The daily dose is usually given in 3 divided doses.

For use as *adjunctive therapy* with levodopa, pergolide should be introduced gradually and during this period patients can have their levodopa dosage decreased gradually until an optimum response is achieved. The initial dose of pergolide is the equivalent of 50 micrograms daily for the first 2 days, increased gradually by 100 or 150 micrograms every third day over the next 12 days of therapy. Further increases of 250 micrograms may then be made every third day until an optimum response is achieved. A usual maintenance dose is 3 mg daily; doses above 3 mg daily are not recommended by the EMEA. The daily dose is usually given in 3 divided doses.

Acromegaly. Dopaminergics can produce a paradoxical reduction in growth hormone secretion and may be used in the treatment of acromegaly as adjunctive therapy to surgery, radiotherapy, or somatostatin analogues to reduce circulating growth hormone levels, although they are less effective than somatostatin analogues (p.1798). While bromocriptine has been the main dopamine agonist used pergolide has also been tried.[1]

1. Kleinberg DL, *et al.* Pergolide for the treatment of pituitary tumors secreting prolactin or growth hormone. *N Engl J Med* 1983; **309:** 704–9.

Hyperprolactinaemia and prolactinomas. Dopamine agonists are widely used for the treatment of hyperprolactinaemia secondary to a prolactinoma (p.2079). Pergolide has been suggested as an alternative to bromocriptine in this condition.

Studies[1-3] of pergolide mesilate in patients with hyperprolactinaemia indicate that single doses reduce serum-prolactin concentrations for more than 24 hours. In most patients, the effective dose was between 50 and 150 micrograms daily. Adverse effects were similar to those seen with bromocriptine, although some patients who could not take bromocriptine were able to tolerate pergolide (and *vice versa*). Pergolide reportedly lost favour for this indication after reports of an increased incidence of uterine neoplasms in *rodents* receiving high doses. However, licensed product information states that no cases of uterine malignancies have to date been reported in humans receiving pergolide. The long-term outcome of treatment of macroprolactinomas with pergolide has been examined in 23 patients,[4] and efficacy and relative safety of pergolide was demonstrated after an average of 27 months (range: 9 to 64 months) treatment.

1. Franks S, *et al.* Treatment of hyperprolactinaemia with pergolide mesylate: acute effects and preliminary evaluation of long-term treatment. *Lancet* 1981; **ii:** 659–61.

2. Franks S, *et al.* Effectiveness of pergolide mesylate in long-term treatment of hyperprolactinaemia. *BMJ* 1983; **286:** 1177–9.

3. Kleinberg DL, *et al.* Pergolide for the treatment of pituitary tumors secreting prolactin or growth hormone. *N Engl J Med* 1983; **309:** 704–9.

4. Freda PU, *et al.* Long-term treatment of prolactin-secreting macroadenomas with pergolide. *J Clin Endocrinol Metab* 2000; **85:** 8–13.

Parkinsonism. Dopamine agonists such as pergolide are often used to begin the treatment of parkinsonism (p.791) in an attempt to delay therapy with levodopa, particularly in younger patients. They also have an adjunctive use when levodopa is no longer effective alone or cannot be tolerated, and may sometimes be useful in reducing 'off' periods with levodopa and in ameliorating other fluctuations in mobility in the later stages of the disease. Pergolide has a relatively long duration of action compared with other dopamine agonists commonly used. Although the duration of the clinical antiparkinsonian effect of pergolide remains to be determined, studies suggest it is of the order of 5 to 8 hours. Depending on the dose used the response to other dopamine agonists in late parkinsonism is 1 to 4 hours for levodopa, 2 to 4 hours for lisuride, and 4 to 6 hours for bromocriptine.

References.

1. Anonymous. Pergolide (Celance)—a third dopamine agonist. *Drug Ther Bull* 1991; **29:** 79.

2. Markham A, Benfield P. Pergolide: a review of its pharmacology and therapeutic use in Parkinson's disease. *CNS Drugs* 1997; **7:** 328–40.

3. Barone P, *et al.* Pergolide monotherapy in the treatment of early PD: a randomized, controlled study. *Neurology* 1999; **53:** 573–9.

4. Clarke CE, Speller JM. Pergolide for levodopa-induced complications in Parkinson's disease. Available in The Cochrane Database of Systematic Reviews, Issue 2. Chichester: John Wiley; 1999 (accessed 16/02/06).

5. Clarke CE, Speller JM. Pergolide versus bromocriptine for levodopa-induced complications in Parkinson's disease. Available in The Cochrane Database of Systematic Reviews; Issue 2. Chichester: John Wiley; 1999 (accessed 16/02/06).

Restless legs syndrome. The aetiology of restless legs syndrome (see Sleep-associated Movement Disorders, p.958) is obscure and treatment has largely been empirical although dopaminergic therapy has emerged as a common first-line choice. Pergolide has produced some benefit in a number of studies.[1-6] In a randomised placebo-controlled study,[6] therapeutic effects were observed with mean daily doses of pergolide 400 micrograms after 6 weeks and maintained after 12 months with doses of up to 720 micrograms daily.

1. Silber MH, *et al.* Pergolide in the management of restless legs syndrome: an extended study. *Sleep* 1997; **20:** 878–82.

2. Winkelmann J, *et al.* Treatment of restless legs syndrome with pergolide—an open clinical trial. *Mov Disord* 1998; **13:** 566–9.

3. Earley CJ, *et al.* Randomized, double-blind, placebo-controlled trial of pergolide in restless legs syndrome. *Neurology* 1998; **51:** 1599–1602.

4. Wetter TC, *et al.*. A randomized controlled study of pergolide in patients with restless legs syndrome. *Neurology* 1999; **52:** 944–50.

5. Stiasny K, *et al.* Long-term effects of pergolide in the treatment of restless legs syndrome. *Neurology* 2001; **56:** 1399–1402.

6. Trenkwalder C, *et al.* Efficacy of pergolide in treatment of restless legs syndrome: the PEARLS study. *Neurology* 2004; **62:** 1391–7.

Tourette's syndrome. Pergolide has been studied in the management of Tourette's syndrome (see Tics, p.954). A preliminary study[1] produced encouraging results, subsequently confirmed by placebo-controlled trials in children and adolescents.[2,3]

1. Lipinski JF, *et al.* Dopamine agonist treatment of Tourette disorder in children: results of an open-label trial of pergolide. *Mov Disord* 1997; **12:** 402–7.

2. Gilbert DL, *et al.* Tourette's syndrome improvement with pergolide in a randomized, double-blind, crossover trial. *Neurology* 2000; **54:** 1310–15.

3. Gilbert DL, *et al.* Tic reduction with pergolide in a randomized controlled trial in children. *Neurology* 2003; **60:** 606–11.

Preparations

USP 31: Pergolide Tablets.

Proprietary Preparations (details are given in Part 3)

Arg.: Aroltex; Breator†; Celance; Geranil†; Parlide†; ***Austral.:*** Permax; ***Austria:*** Permax; ***Belg.:*** Permax; ***Braz.:*** Celance; ***Canad.:*** Permax; ***Chile:*** Celance†; ***Cz.:*** Permax; ***Denm.:*** Permax; ***Fin.:*** Permax; ***Fr.:*** Celance; ***Ger.:*** Parkotil; ***Gr.:*** Celance; ***Hong Kong:*** Celance†; ***Hung.:*** Parkotil†; ***Irl.:*** Celance; ***Ital.:*** Nopar; ***Mex.:*** Permax; ***Neth.:*** Permax; ***NZ:*** Permax; ***Pol.:*** Hizest†; ***Port.:*** Permax; ***S.Afr.:*** Permax; ***Singapore:*** Celance†; ***Spain:*** Pharken; ***Switz.:*** Permax; ***Thai.:*** Celance†; ***Turk.:*** Permax; ***UK:*** Celance; ***USA:*** Permax†.

Piribedil *(rINN)*

ET-495; EU-4200; Piribédil; Piribedilum. 2-(4-Piperonylpiperazin-1-yl)pyrimidine.

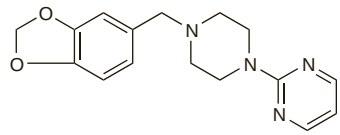
Пирибедил

$C_{16}H_{18}N_4O_2$ = 298.3.

CAS — 3605-01-4.

ATC — N04BC08.

ATC Vet — QN04BC08.



Profile

Piribedil is a non-ergot dopamine agonist that has been given orally in the treatment of parkinsonism and in circulatory disorders. Piribedil mesilate has been given by injection for circulatory disorders. Preparations of piribedil are licensed in some countries for the management of impaired memory and cognitive function in the elderly.

Adverse effects reported include nausea and vomiting, dizziness, hallucinations, confusion, drowsiness, hypothermia, dyskinesias, and occasional changes in liver function.

When used as monotherapy in Parkinson's disease a usual daily oral dose of piribedil is 150 to 250 mg in divided doses; a daily dose of 80 to 140 mg may be suitable when used as an adjunct to levodopa.

Effects on mental function. For reports of daytime somnolence occurring in patients receiving dopamine agonists including piribedil, see under Adverse Effects of Levodopa, p.805.

Parkinsonism. Piribedil is a dopamine D_2-agonist while its metabolite is reported to act on D_1 receptors. It has been mainly used as an adjunct to levodopa therapy in the treatment of Parkinson's disease (p.791).

References.

1. Montastruc JL, *et al.* Current status of dopamine agonists in Parkinson's disease management. *Drugs* 1993; **46:** 384–93.

2. Montastruc JL, *et al.* A randomized, double-blind study of a skin patch of a dopaminergic agonist, piribedil, in Parkinson's disease. *Mov Disord* 1999; **14:** 336–41.

3. Ziegler M, Rondot P. Activité du piribédil dans la maladie de Parkinson: étude multicentrique. *Presse Med* 1999; **28:** 1414–18.

4. Ziegler M, *et al.* Efficacy of piribedil as early combination to levodopa in patients with stable Parkinson's disease: a 6-month, randomized, placebo-controlled study. *Mov Disord* 2003; **18:** 418–25.

Preparations

Proprietary Preparations (details are given in Part 3)

Arg.: Trivastal; ***Braz.:*** Trivastal; ***Fr.:*** Trivastal; ***Ger.:*** Trivastal; ***Gr.:*** Trivastal; ***India:*** Trivastal; ***Ital.:*** Trivastan; ***Malaysia:*** Trivastal; ***Philipp.:*** Trivastal; ***Pol.:*** Pronoran; ***Port.:*** Trivastal; ***Rus.:*** Pronoran (Пронoран); ***Singapore:*** Trivastal; ***Thai.:*** Trivastal; ***Turk.:*** Trivastal; ***Venez.:*** Trivastal.

Pramipexole Hydrochloride

(BANM, rINNM)

Hidrocloruro de pramipexol; PNU-98528-E; Pramipexole, Chlorhydrate de; Pramipexole, dichlorhydrate de; Pramipexole Dihydrochloride *(USAN)*; Pramipexoli dihydrochloridum; Pramipexoli Hydrochloridum; SND-919-CL-2Y (pramipexole hydrochloride); SUD-919Y (pramipexole). (*S*)-2-Amino-4,5,6,7-tetrahydro-6-(propylamino)benzothiazole dihydrochloride monohydrate.

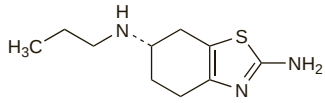
Прамипексола Гидрохлорид

$C_{10}H_{17}N_3S,2HCl, H_2O$ = 302.3.

CAS — 104632-26-0 (pramipexole); 104632-25-9 (anhydrous pramipexole hydrochloride); 191217-81-9 (pramipexole hydrochloride monohydrate).

ATC — N04BC05.

ATC Vet — QN04BC05.



(pramipexole)

Adverse Effects and Precautions

As for Bromocriptine, p.798.

Pramipexole should be used with caution in patients with renal impairment and reduced doses are recommended.

Ophthalmological monitoring is recommended at regular intervals or if vision abnormalities occur.

Incidence of adverse effects. References.

1. Etminan M, *et al.* Comparison of the risk of adverse events with pramipexole and ropinirole in patients with Parkinson's disease: a meta-analysis. *Drug Safety* 2003; **26:** 439–44.

Effects on mental function. Pramipexole has been associated with attacks of sudden onset of sleep, sometimes without any prior feeling of drowsiness, that can occur at any time during treatment. Licensed product information states that the incidence of daytime **somnolence** is increased at daily doses of pramipexole hydrochloride higher than 1.5 mg. A retrospective analysis[1] of data to evaluate the incidence and nature of somnolence in patients receiving pramipexole in clinical studies showed that for patients with moderate or severe somnolence, the onset of worst-reported somnolence occurred at a mean daily dose of around 4 mg (range: 0.75 to 4.5 mg).

For further reports of daytime somnolence occurring in patients receiving dopamine agonists including pramipexole, see under Adverse Effects of Levodopa, p.805.

For reference to **pathological gambling** reported in patients with Parkinson's disease receiving dopamine agonists, including pramipexole, see under Levodopa, p.805.

1. Hauser RA, *et al.* Pramipexole-induced somnolence and episodes of daytime sleep. *Mov Disord* 2000; **15:** 658–63.

Interactions

As for Bromocriptine, p.800. Cimetidine is reported to reduce the renal clearance of pramipexole.

Caution is advised when other sedating drugs or alcohol are used with pramipexole because of possible additive effects and the risk of precipitating sudden onset of sleep (see above).

◊ References.

1. Wright CE, *et al.* Influence of probenecid and cimetidine on pramipexole pharmacokinetics. *Clin Pharmacol Ther* 1996; **59:** 183.

Pharmacokinetics

Pramipexole is readily absorbed from the gastrointestinal tract and peak plasma concentrations have been reached within about 2 hours in fasting patients and in about 3 hours when given with food. Oral bioavailability is reported to be about 90%. Pramipexole is widely distributed throughout the body and plasma-protein binding is less than 20%. Metabolism is minimal and more than 90% of a dose is excreted via renal tubular secretion unchanged into the urine. Elimination half-lives of 8 to 12 hours have been reported. On the basis of studies in *rats*, it is thought to be distributed into breast milk.

◊ References.

1. Wright CE, *et al.* Steady-state pharmacokinetic properties of pramipexole in healthy volunteers. *J Clin Pharmacol* 1997; **37:** 520–5.

Uses and Administration

Pramipexole is a non-ergot dopamine agonist with actions similar to those of bromocriptine (p.800). It is used similarly in the management of Parkinson's disease, either alone or as an adjunct to levodopa therapy to reduce 'end-of-dose' or 'on-off' fluctuations in response. Pramipexole is also used for the treatment of moderate to severe restless legs syndrome. It is given orally as the hydrochloride; doses have been described in terms of the hydrochloride (as below) or of the base. In terms of equivalency:

- pramipexole hydrochloride 125 micrograms is equivalent to about 88 micrograms of pramipexole
- pramipexole hydrochloride 250 micrograms is equivalent to about 180 micrograms of pramipexole
- pramipexole hydrochloride 500 micrograms is equivalent to about 350 micrograms of pramipexole
- pramipexole hydrochloride 1 mg is equivalent to about 700 micrograms of pramipexole

In the treatment of **Parkinson's disease**, the dose of pramipexole should be increased gradually and the dose of levodopa gradually reduced during the dose-titration and maintenance phases until an optimum response is achieved. The initial dose of pramipexole *hydrochloride* is 125 micrograms three times daily increased to 250 micrograms three times daily in the second week and then to 500 micrograms three times daily in the third week according to response. Thereafter the daily dose may be increased if necessary by 750 micrograms at weekly intervals to a maximum of 4.5 mg daily. The dosage should be reduced in patients with renal impairment (see below).

If it is necessary to stop pramipexole therapy, it should be withdrawn gradually. UK licensed product information suggests tapering off the dose of pramipexole hydrochloride at a rate of 750 micrograms daily until a daily dose of 750 micrograms has been reached; thereafter, the dose should be reduced by 375 micrograms daily. US licensed product information suggests that withdrawal should be gradual over a period of 1 week.

Pramipexole is also given as a single daily dose, 2 to 3 hours before bedtime, in the treatment of **restless legs syndrome**. The initial dose of pramipexole *hydrochloride* is 125 micrograms daily. This may be increased if necessary after 4 to 7 days to 250 micrograms daily. Subsequent doses may be increased if necessary by 250 micrograms every 4 to 7 days to a maximum of 750 micrograms daily. Response to therapy should be evaluated after 3 months; if treatment is interrupted for more than a few days, it should be restarted at 125 micrograms daily, and then increased, if required, as described above. For this indication, pramipexole may be withdrawn without gradual tapering of the dose.

Administration in renal impairment. The elimination of pramipexole is dependent on renal function and the dosage of pramipexole hydrochloride should therefore be reduced in patients with renal impairment.

In the treatment of *Parkinson's disease*, the following dosage schedule has been suggested in UK licensed product information for initial therapy adjusted according to the patient's creatinine clearance (CC):

- CC 20 to 50 mL/minute: 125 micrograms given twice daily
- CC less than 20 mL/minute: 125 micrograms once daily

Similar reductions are suggested in US licensed information for patients with a CC of 35 to 59 mL/minute and 15 to 34 mL/minute, respectively.

If renal function declines during maintenance therapy it is recommended that the daily dose of pramipexole should be reduced by the same percentage as the decline in CC.

Lower doses are used in the treatment of *restless legs syndrome* and UK licensed information considers dosage reductions unnecessary in patients with CC of more than 20 mL/minute. US licensed information recommends that the interval between titration steps is increased to 14 days in those patients with CC of 20 to 60 mL/minute.

Parkinsonism. Pramipexole is a dopamine agonist used in the treatment of Parkinson's disease (p.791) as an adjunct to levodopa therapy to reduce 'off' periods with levodopa and ameliorate other fluctuations of mobility in the later stages of the disease. It is also used as monotherapy early in the course of the disease in an attempt to delay therapy with levodopa.

References.

1. Parkinson Study Group. Safety and efficacy of pramipexole in early Parkinson disease: a randomized dose-ranging study. *JAMA* 1997; **278:** 125–30.
2. Lieberman A, *et al.* Clinical evaluation of pramipexole in advanced Parkinson's disease: results of a double-blind, placebo-controlled, parallel-group study. *Neurology* 1997; **49:** 162–8.
3. Shannon KM, *et al.* Efficacy of pramipexole, a novel dopamine agonist, as monotherapy in mild to moderate Parkinson's disease. *Neurology* 1997; **49:** 724–8.
4. Guttman M. International Pramipexole-Bromocriptine Study Group. Double-blind comparison of pramipexole and bromocriptine treatment with placebo in advanced Parkinson's disease. *Neurology* 1997; **49:** 1060–5.
5. Parkinson Study Group. Pramipexole vs levodopa as initial treatment for Parkinson disease: a randomized controlled trial. *JAMA* 2000; **284:** 1931–8.
6. Clarke CE, *et al.* Pramipexole for levodopa-induced complications in Parkinson's disease. Available in The Cochrane Database of Systematic Reviews; Issue 2. Chichester: John Wiley; 2000 (accessed 16/02/06).
7. Clarke CE, *et al.* Pramipexole versus bromocriptine for levodopa-induced complications in Parkinson's disease. Available in The Cochrane Database of Systematic Reviews; Issue 2. Chichester: John Wiley; 2000 (accessed 16/02/06).
8. Parkinson Study Group. Pramipexole vs levodopa as initial treatment for Parkinson disease: a 4-year randomized controlled trial. *Arch Neurol* 2004; **61:** 1044–53. Correction. *ibid.* 2005; **62:** 430.
9. Moller JC, *et al.* Long-term efficacy and safety of pramipexole in advanced Parkinson's disease: results from a European multicenter trial. *Mov Disord* 2005; **20:** 602–10.

Restless legs syndrome. The aetiology of restless legs syndrome (RLS—see Sleep-associated Movement Disorders, p.958) is obscure and treatment has largely been empirical, although dopaminergic therapy has emerged as a common first-line choice. Low-dose pramipexole has produced some benefit in a number of studies[1-7] and is licensed for the treatment of moderate to severe RLS in some countries.

1. Lin S-C, *et al.* Effect of pramipexole in treatment of resistant restless legs syndrome. *Mayo Clin Proc* 1998; **73:** 497–500.
2. Montplaisir J, *et al.* Restless legs syndrome improved by pramipexole: a double-blind randomized trial. *Neurology* 1999; **52:** 938–43.
3. Montplaisir J, *et al.* Pramipexole in the treatment of restless legs syndrome: a follow-up study. *Eur J Neurol* 2000; **7** (suppl 1): 27–31.
4. Saletu M, *et al.* Acute placebo-controlled sleep laboratory studies and clinical follow-up with pramipexole in restless legs syndrome. *Eur Arch Psychiatry Clin Neurosci* 2002; **252:** 185–94.
5. Silber MH, *et al.* Pramipexole in the management of restless legs syndrome: an extended study. *Sleep* 2003; **26:** 819–21.
6. Stiasny-Kolster K, Oertel WH. Low-dose pramipexole in the management of restless legs syndrome: an open label trial. *Neuropsychobiology* 2004; **50:** 65–70.
7. Winkelman JW, *et al.* Efficacy and safety of pramipexole in restless legs syndrome. *Neurology* 2006; **67:** 1034–9.

Preparations

Proprietary Preparations (details are given in Part 3)

Arg.: Maxtenk; Mirapex†; Nixol; Parfeno; Parxium; Portiv; Sifrol; ***Austral.:*** Sifrol; ***Austria:*** Sifrol; ***Belg.:*** Mirapexin; ***Braz.:*** Mirapex; Sifrol; ***Canad.:*** Mirapex; ***Chile:*** Sifrol; ***Cz.:*** Mirapexin; Sifrol; ***Denm.:*** Mirapexin; Sifrol; ***Fin.:*** Sifrol; ***Fr.:*** Sifrol; ***Ger.:*** Sifrol; ***Gr.:*** Mirapexin; ***Hung.:*** Mirapexin; ***Indon.:*** Sifrol; ***Irl.:*** Mirapexin; ***Ital.:*** Mirapexin; ***Jpn:*** BI-Sifrol; ***Malaysia:*** Sifrol; ***Mex.:*** Sifrol; ***Neth.:*** Daquiran†; Sifrol; ***Norw.:*** Sifrol; ***NZ:*** Sifrol; ***Philipp.:*** Sifrol; ***Pol.:*** Mirapexin; ***Port.:*** Mirapexin; Sifrol; ***Rus.:*** Mirapex (Мирапекс); ***S.Afr.:*** Pexola; ***Singapore:*** Sifrol; ***Spain:*** Mirapexin; ***Swed.:*** Sifrol; ***Switz.:*** Sifrol; ***Thai.:*** Sifrol; ***Turk.:*** Pexola; ***UK:*** Mirapexin; ***USA:*** Mirapex; ***Venez.:*** Sifrol/Mirapex.

Procyclidine Hydrochloride

(BANM, rINNM)

Hidrocloruro de prociclidina; Procyclidine, Chlorhydrate de; Procyclidini Hydrochloridum. 1-Cyclohexyl-1-phenyl-3-(pyrrolidin-1-yl)propan-1-ol hydrochloride.

Проциклидина Гидрохлорид

$C_{19}H_{29}NO,HCl$ = 323.9.

CAS — 77-37-2 (procyclidine); 1508-76-5 (procyclidine hydrochloride).

ATC — N04AA04.

ATC Vet — QN04AA04.