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

Interactions
As for Amobarbital, p.962.

Pharmacokinetics
Secobarbital is well absorbed from the gastrointestinal tract after oral doses and is reported to be about 46 to 70% bound to plasma proteins. The mean elimination half-life is reported to be 28 hours. It is metabolised in the liver, mainly by hydroxylation, and excreted in urine as metabolites and a small amount of unchanged drug.

Uses and Administration
Secobarbital is a barbiturate that has been used as a hypnotic and sedative. It has general properties similar to those of amobarbital (p.962). As a hypnotic in the short-term management of insomnia (p.957) it was usually given in an oral dose of 100 mg of the sodium salt at night, but barbiturates are no longer considered appropriate for such use.

Secobarbital sodium has also been given orally or by intramuscular or intravenous injection for premedication in anaesthetic procedures (p.1780) but barbiturates for pre-operative sedation have been replaced by other drugs.

Preparations
USP 31: Secobarbital Elixir; Secobarbital Sodium and Amobarbital Sodium Capsules; Secobarbital Sodium Capsules; Secobarbital Sodium for Injection; Secobarbital Sodium Injection.

Proprietary Preparations (details are given in Part 3)
UK: Seconal; ***USA:*** Seconal.

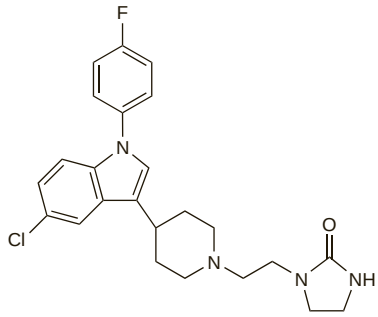
Multi-ingredient: ***Port.:*** Vesparax†; ***UK:*** Tuinal; ***USA:*** Tuinal.

Sertindole *(BAN, USAN, rINN)*

Lu-23-174; Sertindol; Sertindoli; Sertindolum. 1-(2-{4-[5-Chloro-1-(*p*-fluorophenyl)indol-3-yl]piperidino}ethyl)-2-imidazolidinone.

Сертиндол

$C_{24}H_{26}ClFN_4O$ = 440.9.
CAS — 106516-24-9.
ATC — N05AE03.
ATC Vet — QN05AE03.



Adverse Effects, Treatment, and Precautions
Although sertindole may share some of the adverse effects seen with the classical antipsychotics (see Chlorpromazine, p.969), the incidence and severity of such effects may vary. Sertindole is associated with a low incidence of extrapyramidal adverse effects and does not appear to cause sedation. Prolactin elevation may be less frequent. The most common adverse effects with sertindole are peripheral oedema, rhinitis, dyspnoea, sexual dysfunction, dizziness, dry mouth, orthostatic hypotension, weight gain, and paraesthesia. Hyperglycaemia, convulsions, and tardive dyskinesia are uncommon.

Marketing of sertindole has been restricted because of cardiac arrhythmias and sudden cardiac deaths associated with its use (see below). Since sertindole has been associated with prolongation of the QT interval, usually during the first 3 to 6 weeks of treatment, it is recommended that patients should have an ECG before the start of therapy and periodically during treatment. Patients with pre-existing prolongation of the QT interval or a family history of congenital QT prolongation should not be given sertindole and sertindole should be stopped if such prolongation occurs during treatment. In addition, sertindole is contra-indicated in patients with a history of cardiovascular disease, heart failure, cardiac hypertrophy, arrhythmias, or bradycardia. Certain medications may also increase the risk (see Interactions, below).

Sertindole should not be given to patients with uncorrected hypokalaemia or hypomagnesaemia. Baseline serum potassium and magnesium screening should be performed before starting sertindole therapy in patients who are at risk of significant electrolyte disturbances. Serum potassium should be monitored in patients with electrolyte disturbances, vomiting or diarrhoea, or receiving diuretics during sertindole treatment. It is also recommended that blood pressure should be monitored during dose titration and in early maintenance therapy.

Sertindole is contra-indicated in patients with severe hepatic impairment. It should be used with caution in the elderly and in patients with Parkinson's disease, mild to moderate hepatic impairment, or a history of seizures.

Sertindole may affect the performance of skilled tasks including driving.

Gradual withdrawal of sertindole is recommended because of the risk of withdrawal symptoms such as sweating, nausea and vomiting, and rebound psychosis, with abrupt cessation.

Dementia. The FDA has issued advice against the use of atypical antipsychotics in the treatment of behavioural problems in elderly patients with dementia after analysis of placebo-controlled studies showed an increased risk of mortality with certain drugs in this class. See under Risperidone, p.1024.

Effects on body-weight. The increased risk of weight gain with some atypical antipsychotics is discussed under Adverse Effects of Clozapine, p.981.

Effects on carbohydrate metabolism. The increased risk of glucose intolerance and diabetes mellitus with some atypical antipsychotics, and recommendations on monitoring, are discussed under Adverse Effects of Clozapine, p.981.

Effects on the cardiovascular system. Prolongation of the QT interval is said by the manufacturer to be common in patients given sertindole, with the effect being greater at the upper end of the dose range. In addition, the QT interval is prolonged to a greater extent than that seen with some other antipsychotics. QT interval prolongation is a known risk factor for the development of serious arrhythmias such as torsade de pointes although such arrhythmias are uncommon with sertindole.

In evidence presented to the FDA it was reported that as of 1st June 1996 there had been 27 deaths, 16 due to adverse cardiac events, among the 2194 patients given sertindole in clinical studies.[1] By the end of November 1998, the UK CSM was aware of 36 suspected adverse drug reactions with a fatal outcome, 9 of which originated in the UK.[2] There had also been 13 reports of serious but non-fatal cardiac arrhythmias in the UK. Although not all the fatalities were related to sudden cardiac events, at the time the CSM considered that, given the number of serious arrhythmias and sudden cardiac deaths, the risk-benefit ratio of sertindole was no longer favourable. The drug was withdrawn from the market in the UK and subsequently in a number of other countries, although it remained available on a named-patient basis. However, in 2001, the issue was re-evaluated by the CSM and the European advisory body, the Committee on Proprietary Medicinal Products, and it was recommended that sertindole could be reintroduced in Europe under certain restrictions.[3] Initially sertindole should only be prescribed to patients enrolled in clinical studies to ensure that they are carefully selected and monitored. In the UK, sertindole was remarketed in September 2002.

1. Barnett AA. Safety concerns over antipsychotic drug, sertindole. *Lancet* 1996; **348:** 256.
2. CSM/MCA. Suspension of availability of sertindole (Serdolect). *Current Problems* 1999; **25:** 1. Also available at: http://www.mhra.gov.uk/home/idcplg?IdcService=GET_FILE&dDocName=CON2023233&RevisionSelectionMethod=LatestReleased (accessed 16/05/06)
3. CSM/MCA. Restricted re-introduction of the atypical antipsychotic sertindole (Serdolect) (issued 10th September, 2002). Available at: http://www.mhra.gov.uk/Safetyinformation/Safetywarningsalertsandrecalls/Safetywarningsandmessagesformedicines/CON019523 (accessed 21/08/08)

Effects on lipid metabolism. The increased risk of hyperlipidaemia with some atypical antipsychotics is discussed under Adverse Effects of Chlorpromazine, p.970. See also Effects on Carbohydrate Metabolism under Adverse Effects of Clozapine, p.981.

Pregnancy. For comments on the use of some atypical antipsychotics during pregnancy, see under Precautions of Clozapine, p.983.

Interactions
The risk of arrhythmias with sertindole may be increased by other drugs that prolong the QT interval and use together should be avoided. Sertindole should be given with caution with drugs that produce electrolyte disturbances; monitoring of serum potassium is recommended if given with potassium-depleting diuretics.

Sertindole may antagonise the effects of dopaminergics.

Sertindole is extensively metabolised by the cytochrome P450 isoenzymes of the group CYP3A and by CYP2D6. The use of potent inhibitors of CYP3A such as indinavir, itraconazole, and ketoconazole with sertindole is contra-indicated. Minor increases in sertindole plasma concentrations have been noted in patients also given macrolide antibacterials or calcium-channel blockers which also inhibit CYP3A; however, despite the small increase, the use of these CYP3A4 inhibitors with sertindole is not recommended. Fluoxetine and paroxetine, potent inhibitors of CYP2D6, have increased plasma concentrations of sertindole by a factor of 2 to 3 and lower maintenance doses of sertindole may be required. In contrast, enzyme inducers such as rifampicin, carbamazepine, phenytoin, and phenobarbital may decrease sertindole plasma levels by a factor of 2 to 3; in such cases, higher doses of sertindole may be required.

Pharmacokinetics
Sertindole is slowly absorbed with peak concentrations occurring about 10 hours after oral doses. It is about 99.5% bound to plasma proteins and readily crosses the placenta. Sertindole is extensively metabolised in the liver by the cytochrome P450 isoenzymes CYP2D6 and CYP3A. There is moderate interindividual variation in the pharmacokinetics of sertindole due to polymorphism in the isoenzyme CYP2D6. Poor metabolisers, deficient in this isoenzyme, may have plasma concentrations of sertindole 2 to 3 times higher than other patients. The two major metabolites, dehydrosertindole and norsertindole, appear to be inactive. Sertindole and its metabolites are excreted slowly, mainly in the faeces with a minor amount appearing in the urine. The mean terminal half-life is about 3 days.

Uses and Administration
Sertindole is an atypical antipsychotic that is an antagonist at central dopamine (D_2), serotonin ($5\text{-}HT_2$), and adrenergic (α_1) receptors. It is used in the treatment of schizophrenia (p.955) in patients who are unable to tolerate at least one other antipsychotic. In addition, sertindole should only be prescribed to patients enrolled in clinical studies to ensure adequate monitoring, especially regular ECG measurements (see Adverse Effects, above).

Sertindole is given in an initial oral dose of 4 mg once daily, increased gradually in steps of 4 mg every 4 or 5 days to a usual maintenance dose of 12 to 20 mg once daily. The maximum dose is 24 mg daily. Slower dose titration and lower maintenance doses are advisable for the elderly and patients with mild to moderate hepatic impairment.

If therapy is interrupted for 1 week or more, the dose of sertindole should be re-titrated. An ECG should also be undertaken before re-starting sertindole.

◊ References.

1. Lewis R. *et al.* Sertindole for schizophrenia. Available in The Cochrane Database of Systematic Reviews; Issue 3. Chichester: John Wiley; 2005 (accessed 16/05/06).

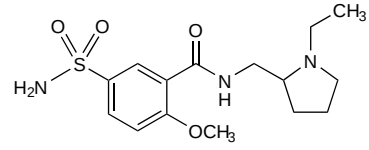
Preparations
Proprietary Preparations (details are given in Part 3)
Austria: Serdolect; ***Cz.:*** Serdolect; ***Fr.:*** Serdolect; ***Gr.:*** Serdolect; ***Hung.:*** Serdolect; ***Neth.:*** Serdolect; ***Port.:*** Serdolect; ***Rus.:*** Serdolect (Сердолект); ***Switz.:*** Serdolect; ***UK:*** Serdolect.

Sulpiride *(BAN, USAN, rINN)*

Sulpirid; Sulpirida; Sulpiridas; Sulpiridi; Sulpiridum; Sülprid; Szulpirid. *N*-(1-Ethylpyrrolidin-2-ylmethyl)-2-methoxy-5-sulphamoylbenzamide.

Сульпирид

$C_{15}H_{23}N_3O_4S$ = 341.4.
CAS — 15676-16-1 (sulpiride).
ATC — N05AL01.
ATC Vet — QN05AL01.



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

Ph. Eur. 6.2 (Sulpiride). A white or almost white crystalline powder. Practically insoluble in water; slightly soluble in alcohol and in dichloromethane; sparingly soluble in methyl alcohol. It dissolves in dilute solutions of mineral acids and in alkali hydroxides.

Levosulpiride *(rINN)*

Levosulpirida; Lévosulpiride; Levosulpiridum; Levosulpride; L-Sulpiride.

Левосульпирид

$C_{15}H_{23}N_3O_4S$ = 341.4.
CAS — 23672-07-3.
ATC — N05AL07.
ATC Vet — QN05AL07.

Adverse Effects, Treatment, and Precautions
As for Chlorpromazine, p.969.

Sleep disturbances, overstimulation, and agitation may occur. Extrapyramidal effects appear to be as frequent as with chlorpromazine but have usually been mild. It has been suggested that sulpiride is less likely to cause tardive dyskinesia but good evidence of any important difference is lacking. Sulpiride is less likely to cause sedation than chlorpromazine and antimuscarinic ef-

fects are minimal. Cardiovascular effects such as hypotension are generally rare although they may occur with overdosage.

Sulpiride should be given with care to manic or hypomanic patients in whom it may exacerbate symptoms.

Breast feeding. Sulpiride may be distributed into breast milk and the *BNF* recommends that its use should be avoided in mothers wishing to breast feed.

On the fifth day after starting D-sulpiride, DL-sulpiride, or L-sulpiride in a dose of 50 mg twice daily, mean concentrations of sulpiride in breast milk from 45 women were 840, 850, and 810 nanograms/mL respectively.[1]

1. Polatti F. Sulpiride isomers and milk secretion in puerperium. *Clin Exp Obstet Gynecol* 1982; **9:** 144–7.

Effects on the cardiovascular system. Sulpiride 100 mg by mouth caused an attack of hypertension in 6 of 26 hypertensive patients; in 4 it induced a rise in urinary excretion of vanillylmandelic acid and catecholamines.[1] A transient rise in blood pressure and catecholamines after sulpiride occurred in 3 patients who were found to have a phaeochromocytoma; another patient probably had a phaeochromocytoma. The means by which sulpiride provoked hypertension were not known but appeared to be due to a noradrenergic effect. Sulpiride should be avoided during the treatment of phaeochromocytoma, and prescribed with great care in hypertensive patients.

1. Corvol P, *et al*. Poussées hypertensives déclenchées par le sulpiride. *Sem Hop Paris* 1974; **50:** 1265–9.

Porphyria. Sulpiride is considered to be unsafe in patients with porphyria because it has been shown to be porphyrinogenic in *animals*.

Renal impairment. For the precautions to be observed in patients with impaired renal function, see under Uses and Administration, below.

Interactions

As for Chlorpromazine, p.973

Gastrointestinal drugs. Giving sulpiride with therapeutic doses of *sucralfate* or an *antacid* containing aluminium and magnesium hydroxides to 6 healthy subjects reduced the mean oral bioavailability of sulpiride by 40 and 32%, respectively.[1] When sulpiride was given 2 hours after the antacid or sucralfate (each in 2 subjects), bioavailability was reduced by about 25%. This interaction was expected to be clinically significant, and it was recommended that sulpiride should be given before, rather than with or after, sucralfate or antacids.

1. Gouda MW, *et al*. Effect of sucralfate and antacids on the bioavailability of sulpiride in humans. *Int J Pharmaceutics* 1984; **22:** 257–63.

Pharmacokinetics

Sulpiride is slowly absorbed from the gastrointestinal tract; peak plasma concentrations are attained 3 to 6 hours after ingestion. Bioavailability is low and subject to interindividual variation. It is rapidly distributed to the tissues but passage across the blood-brain barrier is poor. Sulpiride is about 40% bound to plasma proteins and is reported to have a plasma half-life of about 8 to 9 hours. It is excreted in the urine and faeces, mainly as unchanged drug. Sulpiride is distributed into breast milk.

◊ References.

1. Wiesel F-A, *et al*. The pharmacokinetics of intravenous and oral sulpiride in healthy human subjects. *Eur J Clin Pharmacol* 1980; **17:** 385–91.
2. Bressolle F, *et al*. Sulpiride pharmacokinetics in humans after intramuscular administration at three dose levels. *J Pharm Sci* 1984; **73:** 1128–36.
3. Bressolle F, *et al*. Absolute bioavailability, rate of absorption, and dose proportionality of sulpiride in humans. *J Pharm Sci* 1992; **81:** 26–32.
4. Mauri MC, *et al*. L-sulpiride in young and elderly negative schizophrenics: clinical and pharmacokinetic variables. *Prog Neuropsychopharmacol Biol Psychiatry* 1994; **18:** 355–6.
5. Muller MJ, *et al*. Serum levels of sulpiride enantiomers after oral treatment with racemic sulpiride in psychiatric patients: a pilot study. *Pharmacopsychiatry* 2001; **34:** 27–32.

Uses and Administration

Sulpiride is a substituted benzamide antipsychotic that is reported to be a selective antagonist of central dopamine (D_2, D_3, and D_4) receptors. It is also claimed to have mood elevating properties.

Sulpiride is mainly used in the treatment of psychoses such as schizophrenia (below). It has also been given in the management of Tourette's syndrome (below), anxiety disorders (p.952), depression (p.373), vertigo (p.565), and benign peptic ulceration (below). Levosulpiride, the L-isomer of sulpiride, has been used similarly to sulpiride.

In the treatment of **schizophrenia** in adults and children aged 14 years and over, initial oral doses of 200 to 400 mg of sulpiride are given twice daily, increased if necessary up to a maximum of 1.2 g twice daily in patients with mainly positive symptoms or up to a total of 800 mg daily in patients with mainly negative symptoms. Patients with mixed positive and negative symptoms, with neither predominating, are given usual doses of 400 to 600 mg twice daily. Lower initial doses have been recommended in elderly patients, subsequently adjusted as required.

Sulpiride is also given in some countries by intramuscular injection, in usual doses ranging from 200 to 800 mg daily.

Dosage adjustment is advised in patients with renal impairment (see below).

Administration in renal impairment. A single intravenous dose of sulpiride 100 mg was given to 6 healthy subjects with normal renal function (creatinine clearance greater than 90 mL/minute) and to 3 groups of 6 patients each with creatinine clearances (CC) in the ranges of 30 to 60, 10 to 30, and less than 10 mL/minute.[1] There was a progressive diminution in the rate of elimination and an increase in half-life with decreasing renal function. The mean plasma elimination half-lives were 5.90, 11.02, 19.27, and 25.96 hours in the 4 groups, respectively.

In the UK it has been recommended that the oral dose be reduced according to CC as follows:

- CC 30 to 60 mL/minute: two-thirds of the usual dose, or prolong dosage interval by a factor of 1.5
- CC 10 to 30 mL/minute: half the usual dose, or double dosage interval
- CC less than 10 mL/minute: one-third of the usual dose, or triple dosage interval

However, the *BNF* suggests that sulpiride should be avoided if CC is less than 10 mL/minute.

1. Bressolle F, *et al*. Pharmacokinetics of sulpiride after intravenous administration in patients with impaired renal function. *Clin Pharmacokinet* 1989; **17:** 367–73.

Chorea. Antipsychotics have some action against choreiform movements (p.953) as well as being of use to control the behavioural disturbances of Huntington's chorea. Although sulpiride was found to have produced an overall reduction in abnormal movements in 11 patients with Huntington's chorea when compared with placebo in a double-blind study[1] there was generally no accompanying functional improvement and patients with mild disease tended to worsen when taking sulpiride.

1. Quinn N, Marsden CD. A double blind trial of sulpiride in Huntington's disease and tardive dyskinesia. *J Neurol Neurosurg Psychiatry* 1984; **47:** 844–7.

Gastrointestinal disorders. Although sulpiride is used in some countries as an adjunct in the treatment of peptic ulcer disease (p.1702) it is not among the more usual drugs used for this indication. Efficacy has also been claimed for sulpiride or levosulpiride in a variety of other gastrointestinal disorders, including irritable bowel disease (p.1699), decreased gastrointestinal motility (p.1694), and nausea and vomiting (p.1700), but again they are not among the drugs usually considered for use in these conditions.

Lactation induction. Drug therapy has been used occasionally to stimulate lactation in breast-feeding mothers, although mechanical stimulation of the nipple remains the primary method. Dopamine antagonists such as sulpiride can produce modest increases in breast milk production[1-3] although metoclopramide has been more widely used (see p.2003). However, there is concern about the adverse effects of these drugs. As sulpiride appears in breast milk and may be associated with adverse effects in the infant it has been recommended that it should not be used to enhance milk production.[4,5]

1. Aono T, *et al*. Effect of sulpiride on poor puerperal lactation. *Am J Obstet Gynecol* 1982; **143:** 927–32.
2. Ylikorkala O, *et al*. Sulpiride improves inadequate lactation. *BMJ* 1982; **285:** 249–51.
3. Ylikorkala O, *et al*. Treatment of inadequate lactation with oral sulpiride and buccal oxytocin. *Obstet Gynecol* 1984; **63:** 57–60.
4. Pons G, *et al*. Excretion of psychoactive drugs into breast milk: pharmacokinetic principles and recommendations. *Clin Pharmacokinet* 1994; **27:** 270–89.
5. Lasich AJ. Sulpiride and breastfeeding. *S Afr Med J* 2005; **95:** 624–6.

Schizophrenia. A systematic review[1] of the use of sulpiride for schizophrenia (p.955) or serious mental illness concluded that while sulpiride might be as effective as the classical antipsychotics for schizophrenia and appeared to produce few adverse effects, evidence of its value for treating negative symptoms was lacking. Comparisons with the atypical antipsychotic drugs were also lacking.

1. Soares B, *et al*. Sulpiride for schizophrenia. Available in The Cochrane Database of Systematic Reviews; Issue 1. Chichester: John Wiley; 1999 (accessed 21/08/08).

Tourette's syndrome. When drug treatment is needed for tics and behavioural disturbances in Tourette's syndrome (p.954) dopamine antagonists such as the antipsychotics haloperidol or pimozide are most commonly used but sulpiride has also been tried.[1] Although unlicensed in the UK for the treatment of Tourette's syndrome, the *BNFC* has suggested that oral doses of sulpiride 50 to 400 mg twice daily may be given to those aged from 2 to 12 years, and 100 to 400 mg twice daily to adolescents aged from 12 to 18 years.

1. Robertson MM, *et al*. Management of Gilles de la Tourette syndrome using sulpiride. *Clin Neuropharmacol* 1990; **13:** 229–35.

Preparations

BP 2008: Sulpiride Tablets.

Proprietary Preparations (details are given in Part 3)

Arg.: Dislep; Nivelan†; Vipral; ***Austria:*** Dogmatil; Meresa; ***Belg.:*** Docsulpiri; Dogmatil; Levopraid; ***Braz.:*** Dogmatil; Equilid; ***Chile:*** Aplacid; Dislep; Sanblex; Sedusen; Sulpilan; ***Cz.:*** Dogmatil; Eglonyl†; Prosulpin; Sulpirol; ***Denm.:*** Dogmatil; ***Fin.:*** Suprium; ***Fr.:*** Aiglonyl†; Dogmatil; Synedil; ***Ger.:*** Arminol; Dogmatil; Meresa; neogama; neogama D novo†; Sulp†; Sulpivert; Vertigo-Meresa; vertigo-neogama; ***Gr.:*** Calmoflorine; Darleton; Dogmatyl; Eclorion†; Nonestón†; Nufarol; Nylipark†; Restful; Stamonevrol; Valirem; ***Hong Kong:*** Dogmatil; ***Hung.:*** Depral; ***Indon.:*** Dogmatil; ***Irl.:*** Dolmatil; ***Israel:*** Modal; ***Ital.:*** Championyl; Dobren; Equilid; Levobren; Levopraid; ***Jpn:*** Dogmatyl; ***Malaysia:*** Dogmatil; ***Mex.:*** Dislep; Ekilid; Pontiride; Rimastine; ***Neth.:*** Dogmatil; ***Philipp.:*** Dogmatil; ***Port.:*** Dogmatil; Gustamax†; Lisopride†; ***Rus.:*** Betamaks (Бетамакс); Eglek (Эглек); Eglonil (Эвитол); Prosulpin (Просульпин); ***S.Afr.:*** Eglonyl; Espiride; ***Singapore:*** Dogmatil†; ***Spain:*** Digton; Dogmatil; Guastil; Lebopride; Levogastrol; Pausedal; Psicocen; Sulkine†; Tepavil; ***Switz.:*** Dogmatil; ***Turk.:*** Dogmatil; Meresa; Sulpir; Zeprid; ***UK:*** Dolmatil; Sulpitil†; Sulpor; ***Venez.:*** Dislep; Guven†.

Multi-ingredient: ***Arg.:*** Alplax Digest; Novo Vegestabil†; Tranquinal Soma; Tranxilium Digest; Vegestabil; ***Braz.:*** Bromopirin; Sulpan; ***Mex.:*** Numencial; ***Spain:*** Ansium; Sirodina†; Tepazepan; ***Venez.:*** Tepazepam†.

Sultopride Hydrochloride *(rINNM)*

Hidrocloruro de sultoprida; LIN-1418; Sultopride, Chlorhydrate de; Sultopridi Hydrochloridum. *N*-(1-Ethylpyrrolidin-2-ylmethyl)-5-ethylsulphonyl-2-methoxybenzamide hydrochloride.

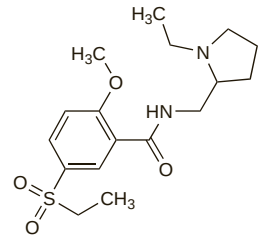
Сультоприда Гидрохлорид

$C_{17}H_{26}N_2O_4S,HCl$ = 390.9.

CAS — 53583-79-2 (sultopride); 23694-17-9 (sultopride hydrochloride).

ATC — N05AL02.

ATC Vet — QN05AL02.



(sultopride)

Profile

Sultopride is a substituted benzamide with general properties similar to those of sulpiride (above). It is used in psychoses such as schizophrenia (p.955). It has also been used in the emergency management of agitation in psychotic or aggressive patients. It is given as the hydrochloride but doses are expressed in terms of the base; sultopride hydrochloride 441 mg is equivalent to about 400 mg of sultopride.

For acute and chronic psychoses it may be given in oral doses of 400 to 800 mg daily; it may also be given intramuscularly.

Ventricular arrhythmias, including torsade de pointes, have been reported. It has been recommended that sultopride should not be used in patients with bradycardia.

Porphyria. Sultopride is considered to be unsafe in patients with porphyria because it has been shown to be porphyrinogenic in *in-vitro* systems.

Preparations

Proprietary Preparations (details are given in Part 3)

Belg.: Barnetil†; ***Fr.:*** Barnetil†; ***Ital.:*** Barnotil†; ***Port.:*** Barnetil†.