Withdrawal. Rebound haemodynamic changes, including hypertension and increased heart rate, occurred 10 to 30 minutes after stopping intravenous sodium nitroprusside infusion in 20 patients with heart failure.[1] The changes generally resolved spontaneously within 1 to 3 hours and produced only minimal exacerbation of symptoms in most patients, although 3 developed pulmonary oedema 20 to 30 minutes after stopping the infusion, needing restarting of nitroprusside in 2 cases. A study[2] investigating a possible mechanism for this effect found that plasma-renin concentrations were increased during infusion of nitroprusside and remained elevated for 30 minutes after the infusion was stopped. It was suggested that this persistence of elevated plasma-renin concentrations after clearance of short-lived nitroprusside may be responsible for the rebound effects.

1. Packer M, *et al*. Rebound hemodynamic events after the abrupt withdrawal of nitroprusside in patients with severe chronic heart failure. *N Engl J Med* 1979; **301:** 1193–7.
2. Cottrell JE, *et al*. Rebound hypertension after sodium nitroprusside-induced hypotension. *Clin Pharmacol Ther* 1980; **27:** 32–6.

Interactions

Enhanced hypotension should be expected if sodium nitroprusside is used with other antihypertensives or drugs that produce hypotension.

Alteplase. Sodium nitroprusside infusion prolonged the fibrinolytic activity of alteplase when given to *animals*; use of nitrovasodilators with alteplase may be responsible for the enhanced bleeding tendency seen in some patients on thrombolytic therapy.[1]

1. Korbut R, *et al*. Prolongation of fibrinolytic activity of tissue plasminogen activator by nitrovasodilators. *Lancet* 1990; **335:** 669.

Pharmacokinetics

Sodium nitroprusside is rapidly metabolised to cyanide in erythrocytes and smooth muscle and, *in vivo*, this is followed by the release of nitric oxide, the active metabolite. Cyanide is further metabolised in the liver to thiocyanate, which is slowly excreted in the urine; this metabolism is mediated by the enzyme rhodanase and requires the presence of thiosulfate. The plasma half-life of thiocyanate is reported to be about 3 days, but may be much longer in patients with renal impairment.

◊ Reviews.

1. Schulz V. Clinical pharmacokinetics of nitroprusside, cyanide, thiosulphate and thiocyanate. *Clin Pharmacokinet* 1984; **9:** 239–51.

Uses and Administration

Sodium nitroprusside is a short-acting hypotensive drug with a duration of action of 1 to 10 minutes. It produces peripheral vasodilatation and reduces peripheral resistance by a direct action on both veins and arterioles. It has been termed a nitrovasodilator because it releases nitric oxide *in vivo*. Its effects appear within a few seconds of intravenous infusion. Sodium nitroprusside is used in the treatment of hypertensive crises (p.1171) and to produce controlled hypotension during general anaesthesia. It has also been used to reduce preload and afterload in severe heart failure (p.1165) including that associated with myocardial infarction (p.1175).

It is given by continuous intravenous infusion of a solution containing 50 to 200 micrograms/mL. A controlled infusion device must be used. The solution should be prepared immediately before use by dissolving sodium nitroprusside in glucose 5% and then diluting with glucose 5%; the solution must be protected from light during infusion. Blood pressure should be monitored closely and care should be taken to prevent extravasation. In general, treatment should not continue for more than 72 hours. If required for several days concentrations of cyanide should be monitored; the blood concentration should not exceed 1 microgram/mL and the serum concentration should not exceed 80 nanograms/mL. Thiocyanate concentrations in blood should also be measured if infusion continues for more than 72 hours and should not exceed 100 micrograms/mL. Since rebound hypertension has been reported when sodium nitroprusside is withdrawn, the infusion should be tailed off gradually over 10 to 30 minutes.

For **hypertensive crises** in patients not receiving antihypertensive drugs, an initial dose of 0.3 to 1.5 micrograms/kg per minute may be given, increasing gradually under close supervision until the desired reduction in blood pressure is achieved. The average dose required to maintain the blood pressure 30 to 40% below the pretreatment diastolic blood pressure is 3 micrograms/kg per minute and the usual dose range is 0.5 to 6 micrograms/kg per minute. Lower doses should be used in patients already receiving other antihypertensives. The maximum recommended rate is about 8 micrograms/kg per minute in the UK, and 10 micrograms/kg per minute in the USA; infusions at these rates should be used for no longer than 10 minutes and should be stopped after 10 minutes if there is no response. If there is a response, sodium nitroprusside should ideally be given for only a few hours to avoid the risk of cyanide toxicity. Treatment with an oral antihypertensive should be introduced as soon as possible.

For the **induction of hypotension** during anaesthesia a maximum dose of 1.5 micrograms/kg per minute is recommended.

In **heart failure** an initial dose of 10 to 15 micrograms/minute has been used, increasing by 10 to 15 micrograms/minute every 5 to 10 minutes according to response. The usual dosage range is 10 to 200 micrograms/minute and the dose should not exceed 280 micrograms/minute (or 4 micrograms/kg per minute).

Sodium nitroprusside has also been used as a reagent for detecting ketones in urine.

Administration in children. Although experience is more limited than with adults, sodium nitroprusside has been successfully used in infants and children. Continuous infusion of nitroprusside at a rate of 2 to 4 micrograms/kg per minute for 28 days was reported[1] in an 11-year-old child with refractory hypertension, without any signs of thiocyanate toxicity. In a series of 58 neonates with cardiovascular disorders or respiratory distress syndrome,[2] sodium nitroprusside was given in a usual initial dose of 250 to 500 nanograms/kg per minute, and the rate was then repeatedly doubled at intervals of 15 to 20 minutes until the desired effect was achieved, adverse effects supervened, or it was judged ineffective. The maximum rate did not exceed 6 micrograms/kg per minute. Infusion of sodium nitroprusside in doses of 0.5 to 8 micrograms/kg per minute to produce controlled reduction of blood pressure has also been reported[3] in 28 children with hypertensive crises; 16 had also received labetalol.[3]

1. Luderer JR, *et al*. Long-term administration of sodium nitroprusside in childhood. *J Pediatr* 1977; **91:** 490–1.
2. Benitz WE, *et al*. Use of sodium nitroprusside in neonates: efficacy and safety. *J Pediatr* 1985; **106:** 102–10.
3. Deal JE, *et al*. Management of hypertensive emergencies. *Arch Dis Child* 1992; **67:** 1089–92.

Ergotamine poisoning. For the use of sodium nitroprusside in the treatment of cyanosis of the extremities due to ergotamine overdosage, see Cardiovascular Effects, p.620.

Preparations

BP 2008: Sodium Nitroprusside Intravenous Infusion;
USP 31: Sodium Nitroprusside for Injection.

Proprietary Preparations (details are given in Part 3)

Arg.: Doketrol; Niprusodio; Nitroprus; ***Braz.:*** Nipride; Nitropresabbott; Nitroprus; ***Canad.:*** Nipride; ***Cz.:*** Nipruss; ***Fr.:*** Nitriate; ***Ger.:*** Nipruss; ***Gr.:*** Nitriate; ***India:*** Sonide; ***Irl.:*** Nipride†; ***Israel:*** Nipruss; ***Jpn:*** Nitopro; ***Mex.:*** Nitan†; ***Rus.:*** Naniprus (Нанипрус); ***S.Afr.:*** Hypoten; SNP; ***Spain:*** Nitroprussiat; ***Turk.:*** Nipruss; ***USA:*** Nitropress.

Sotalol Hydrochloride

(BANM, USAN, rINNM) ⊗

Hidrocloruro de sotalol; MJ-1999; Sotalol, chlorhydrate de; Sotalol Hidroklorür; *d,l*-Sotalol Hydrochloride; Sotalol-hydrochlorid; Sotalolhydroklorid; Sotaloli hydrochloridum; Sotalolihydrokloridi; Sotalolio hidrochloridas; Szotalol-hidroklorid. 4′-(1-Hydroxy-2-isopropylaminoethyl)methanesulphonanilide hydrochloride.

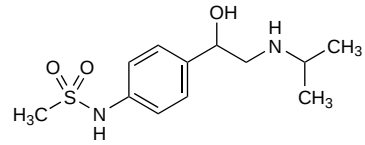
Соталола Гидрохлорид

$C_{12}H_{20}N_2O_3S,HCl$ = 308.8.

CAS — 3930-20-9 (sotalol); 959-24-0 (sotalol hydrochloride).

ATC — C07AA07.

ATC Vet — QC07AA07.



(sotalol)

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

Ph. Eur. 6.2 (Sotalol Hydrochloride). A white or almost white powder. Freely soluble in water; soluble in alcohol; practically insoluble in dichloromethane. A 5% solution in water has a pH of 4.0 to 5.0. Protect from light.

USP 31 (Sotalol Hydrochloride). A white to off-white powder. Freely soluble in water; soluble in alcohol; very slightly soluble in chloroform.

Stability. Suspensions of sotalol hydrochloride 5 mg/mL made using either commercially available or extemporaneously prepared vehicles were found[1] to be stable for up to 3 months when stored at 4° or 25°. Prolonged storage at 25° was not recommended, however, because of the risk of microbial growth.

1. Nahata MC, Morosco RS. Stability of sotalol in two liquid formulations at two temperatures. *Ann Pharmacother* 2003; **37:** 506–9.

Adverse Effects, Treatment, and Precautions

As for Beta Blockers, p.1226.

Torsade de pointes has been reported in patients given sotalol, usually due to prolongation of the QT interval. The QT interval should be monitored: extreme caution is required if the QT interval exceeds 500 milliseconds and sotalol should be stopped or the dose reduced if the QT interval exceeds 550 milliseconds. As hypokalaemia or hypomagnesaemia may predispose patients to arrhythmias, serum-electrolyte concentrations should be monitored before and during treatment with sotalol.

Sotalol should be used with caution in renal impairment (see under Uses and Administration, below) and is contra-indicated in patients whose creatinine clearance is less than 10 mL/minute.

Breast feeding. Sotalol is distributed into breast milk and milk to serum ratios have been reported[1-3] to range from 2.2 to 8.8. In one report[2] it was calculated that a breast-fed infant might ingest 20 to 23% of a maternal dose; however, no bradycardia was noted in the infant in this study. The American Academy of Pediatrics states[4] that there have been no reports of clinical effects in breast-fed infants whose mothers were receiving sotalol and that therefore it may be considered to be usually compatible with breast feeding.

1. O'Hare MF, *et al*. Sotalol as a hypotensive agent in pregnancy. *Br J Obstet Gynaecol* 1980; **87:** 814–20.
2. Hackett LP, *et al*. Excretion of sotalol in breast milk. *Br J Clin Pharmacol* 1990; **29:** 277–8.
3. Wagner X, *et al*. Coadministration of flecainide acetate and sotalol during pregnancy: lack of teratogenic effects, passage across the placenta, and excretion in human breast milk. *Am Heart J* 1990; **119:** 700–2.
4. American Academy of Pediatrics. The transfer of drugs and other chemicals into human milk. *Pediatrics* 2001; **108:** 776–89. Correction. *ibid.*; 1029. Also available at: http://aappolicy.aappublications.org/cgi/content/full/pediatrics%3b108/3/776 (accessed 10/07/07)

Interactions

There is an increased risk of precipitating ventricular arrhythmias if sotalol is given with other drugs that prolong the QT interval, and use with the following drugs is therefore not recommended: class Ia antiarrhythmics (including disopyramide, procainamide, and quinidine), amiodarone, phenothiazine antipsychotics, tricyclic antidepressants, certain antihistamines (astemizole or terfenadine), cisapride, erythromycin, halofantrine, pentamidine, quinolones, sultopride, and vincamine. Caution is required if sotalol is given with drugs that cause electrolyte disturbances, such as diuretics, since this also increases the risk of arrhythmias.

Other interactions associated with beta blockers are discussed on p.1228.

Pharmacokinetics

Sotalol is almost completely absorbed from the gastrointestinal tract and peak plasma concentrations are

obtained about 2 to 4 hours after a dose. The plasma elimination half-life is about 10 to 20 hours. Sotalol has low lipid solubility. Very little is metabolised and it is excreted unchanged in the urine. Binding to plasma proteins is reported to be low. It crosses the placenta and is distributed into breast milk; concentrations in milk may be higher than those in maternal serum (see Breast Feeding, above). Only small amounts are reported to cross the blood-brain barrier and enter the CSF. Sotalol is removed by dialysis.

◊ General references.

1. Singh BN, *et al.* Sotalol: a review of its pharmacodynamic and pharmacokinetic properties, and therapeutic use. *Drugs* 1987; **34:** 311–49.
2. Fitton A, Sorkin EM. Sotalol: an updated review of its pharmacological properties and therapeutic use in cardiac arrhythmias. *Drugs* 1993; **46:** 678–719.

Pregnancy. The systemic clearance of sotalol in 6 healthy women after an intravenous dose was significantly higher during pregnancy than in the postnatal period, and the mean elimination half-life was shorter (6.6 versus 9.3 hours), although the latter difference was not significant.[1] Clearance after an oral dose was also higher during pregnancy than afterwards, but half-lives (10.9 versus 10.3 hours) and mean bioavailability were similar. The changes were probably due to alterations in renal function in the antenatal period.

In a study[2] of transplacental therapy, sotalol was found to cross the placenta easily and completely, with steady-state plasma concentrations similar in mother and fetus. Sotalol accumulated in the amniotic fluid but not in the fetus; it was not associated with fetal growth restriction.

1. O'Hare MF, *et al.* Pharmacokinetics of sotalol during pregnancy. *Eur J Clin Pharmacol* 1983; **24:** 521–4.
2. Oudijk MA, *et al.* Treatment of fetal tachycardia with sotalol: transplacental pharmacokinetics and pharmacodynamics. *J Am Coll Cardiol* 2003; **42:** 765–70.

Uses and Administration

Sotalol is a non-cardioselective beta blocker (p.1225). It is reported to lack both intrinsic sympathomimetic and membrane-stabilising properties. In addition to the class II antiarrhythmic activity of beta blockers, sotalol lengthens the duration of the action potential resulting in class III antiarrhythmic activity. For a classification and explanation of antiarrhythmic activity, see p.1153.

Sotalol is used in the management of ventricular and supraventricular arrhythmias (p.1160). Because of its proarrhythmic effects, it is usually reserved for severe or life-threatening arrhythmias, and it should not be used in patients with asymptomatic ventricular arrhythmias. Although it was formerly used for its beta-blocking effects in the management of angina pectoris, hypertension, and myocardial infarction, it is no longer recommended for these indications because of the risk of precipitating arrhythmias.

Sotalol is given as the hydrochloride. Treatment should be started in hospital with suitable monitoring facilities. The QT interval should be assessed before the start of treatment and whenever the dosage is adjusted (see Precautions above); plasma-electrolyte concentrations and renal function should also be monitored. The dose should be reduced in patients with renal impairment (see below).

The usual initial oral dose of sotalol hydrochloride is 80 mg daily, as a single dose or in two divided doses. The dosage is then individualised according to response, and doses are increased gradually allowing 2 or 3 days between increments. US licensed product information recommends a higher initial dose of 80 mg twice daily and this should not be increased for at least 3 days. Most patients respond to doses of 160 to 320 mg daily (usually given in two divided doses). Some patients with ventricular arrhythmias may require doses as high as 640 mg daily.

Sotalol may be given intravenously to control acute arrhythmias, to substitute for oral therapy, and for programmed electrical stimulation. To control acute arrhythmias, sotalol hydrochloride is given in a dose of 20 to 120 mg (500 to 1500 micrograms/kg) intravenously over 10 minutes. This dose may be repeated every 6 hours if necessary. To substitute for oral therapy an intravenous infusion of 200 to 500 micrograms/kg per hour may be used. The total daily dose should not exceed 640 mg. For programmed electrical stimulation (to test antiarrhythmic efficacy) an initial dose of 1.5 mg/kg is given over 10 to 20 minutes, followed by an intravenous infusion of 200 to 500 micrograms/kg per hour.

Sotalol is used as a racemic mixture; *d*-sotalol (dexsotalol; (+)-sotalol) has also been investigated as an antiarrhythmic but development was stopped when it was found to increase mortality (see Action, below).

◊ General references.

1. Fitton A, Sorkin EM. Sotalol: an updated review of its pharmacological properties and therapeutic use in cardiac arrhythmias. *Drugs* 1993; **46:** 678–719.
2. Nappi JM, McCollam PL. Sotalol: a breakthrough antiarrhythmic? *Ann Pharmacother* 1993; **27:** 1359–68.
3. Zanetti LAF. Sotalol: a new class III antiarrhythmic agent. *Clin Pharm* 1993; **12:** 883–91.
4. Hohnloser SH, Woosley RL. Sotalol. *N Engl J Med* 1994; **331:** 31–8.
5. Anderson JL, Prystowsky EN. Sotalol: an important new antiarrhythmic. *Am Heart J* 1999; **137:** 388–409.

Action. Sotalol is used as the racemic mixture of the two stereoisomers, *d*-sotalol (dexsotalol; (+)-sotalol) and *l*-sotalol ((−)-sotalol). A comparison of the effects of *d*-sotalol and racemic sotalol in 6 healthy subjects[1] showed that the beta-blocking activity resided almost entirely in the *l*-isomer, while the effects on the QT interval, which are consistent with type III antiarrhythmic activity, appear to be due to both isomers. A study in 8 healthy subjects also showed a lack of beta blockade by *d*-sotalol.[2] This would suggest that the electrophysiological effects of sotalol are unrelated to its beta-blocking properties. *d*-Sotalol has been investigated as an antiarrhythmic.[3] However, a preliminary placebo-controlled study in patients with myocardial infarction at high risk of arrhythmia due to impaired left ventricular function was terminated early when increased mortality was seen in the treatment group.[4,5]

1. Johnston GD, *et al.* A comparison of the cardiovascular effects of (+)-sotalol and (±)-sotalol following intravenous administration in normal volunteers. *Br J Clin Pharmacol* 1985; **20:** 507–10.
2. Yasuda SU, *et al.* d-Sotalol reduces heart rate in vivo through a β-adrenergic receptor-independent mechanism. *Clin Pharmacol Ther* 1993; **53:** 436–42.
3. Advani SV, Singh BN. Pharmacodynamic, pharmacokinetic and antiarrhythmic properties of d-sotalol, the dextro-isomer of sotalol. *Drugs* 1995; **49:** 664–79.
4. Choo V. SWORD slashed. *Lancet* 1994; **344:** 1358.
5. Waldo AL, *et al.* Effect of d-sotalol on mortality in patients with left ventricular dysfunction after recent and remote myocardial infarction. *Lancet* 1996; **348:** 7–12. Correction. *ibid.*; 416.

Administration in children. Sotalol has been used to treat both ventricular and supraventricular arrhythmias in children aged from newborn to adolescent;[1-3] it appears to be effective and well-tolerated, although proarrhythmic effects may occur. Neonates may be more sensitive to the QT-prolonging effects of sotalol[3] and lower doses may be appropriate. In the UK, the *BNFC* recommends the following oral doses of sotalol hydrochloride:

- Neonates: initial dose 1 mg/kg twice daily, increased as necessary every 3 to 4 days to a maximum of 4 mg/kg twice daily
- Children aged 1 month to 12 years: initial dose 1 mg/kg twice daily, increased as necessary every 2 to 3 days to a maximum of 4 mg/kg twice daily (maximum total dose 80 mg twice daily)

Licensed product information in the USA recommends doses of sotalol hydrochloride based on body surface area. Children aged 2 years and over may be given an initial dose of 30 mg/m^2 three times daily, increased as necessary at intervals of at least 36 hours to a maximum of 60 mg/m^2 three times daily. For children under 2 years of age the dose should be further reduced, and nomograms are available providing age-specific recommendations.

In children with refractory supraventricular tachycardia, sotalol has been given with flecainide; in a study[4] in children aged under 1 year, doses used ranged from 100 to 250 mg/m^2 daily of sotalol and from 40 to 150 mg/m^2 daily of flecainide.

Sotalol has also been used transplacentally to treat fetal tachycardias, including atrial flutter and supraventricular tachycardia. It may be effective as second-line therapy in addition to digoxin,[5] and has also been used first-line.[6,7] However, one retrospective study[6] of 21 fetuses given sotalol transplacentally found that it was more effective in atrial flutter than in supraventricular tachycardia; mortality was also higher in fetuses with supraventricular tachycardia, and the authors therefore suggested that sotalol should only be used in resistant cases.

1. Çeliker A, *et al.* Sotalol in treatment of pediatric cardiac arrhythmias. *Pediatr Int* 2001; **43:** 624–30.
2. Beaufort-Krol GCM, Bink-Boelkens MTE. Effectiveness of sotalol for atrial flutter in children after surgery for congenital heart disease. *Am J Cardiol* 1997; **79:** 92–4.
3. Läer S, *et al.* Development of a safe and effective pediatric dosing regimen for sotalol based on population pharmacokinetics and pharmacodynamics in children with supraventricular tachycardia. *J Am Coll Cardiol* 2005; **46:** 1322–30.
4. Price JF, *et al.* Flecainide and sotalol: a new combination therapy for refractory supraventricular tachycardia in children <1 year of age. *J Am Coll Cardiol* 2002; **39:** 517–20.
5. Sonesson S-E, *et al.* Foetal supraventricular tachycardia treated with sotalol. *Acta Paediatr* 1998; **87:** 584–7.
6. Oudijk MA, *et al.* Sotalol in the treatment of fetal dysrhythmias. *Circulation* 2000; **101:** 2721–6.
7. Rebelo M, *et al.* Sotalol in the treatment of fetal tachyarrhythmia. *Rev Port Cardiol* 2006; **25:** 477–81.

Administration in renal impairment. Sotalol is excreted mainly unchanged by the kidneys and may accumulate in renal impairment. The usual daily dosage (see above) should therefore be reduced, either by decreasing the size of each dose, or by increasing the interval between doses. UK licensed product information for oral or intravenous sotalol recommends the following doses based on creatinine clearance (CC):

- CC 30 to 60 mL/minute: half usual dose
- CC 10 to 30 mL/minute: quarter usual dose
- CC less than 10 mL/minute: not recommended

Dosage recommendations in the USA depend on both the indication and CC, and incremental increases should not be made until 5 or 6 doses have been given. In the treatment of ventricular arrhythmias, licensed product information for oral sotalol recommends that in renal impairment doses should be given at the following intervals:

- CC 30 to 59 mL/minute: every 24 hours
- CC 10 to 29 mL/minute: every 36 to 48 hours
- CC less than 10 mL/minute: dosage should be individualised

For the treatment of atrial fibrillation, the same dosage intervals are recommended but sotalol is contra-indicated if CC is less than 40 mL/minute.

In a study of 10 hypertensive patients with varying degrees of renal impairment,[1] the apparent first-order elimination rate constant and plasma clearance of sotalol correlated with glomerular filtration rate. Another study[2] compared kinetics in patients with normal renal function, renal impairment, and renal failure. Elimination half-lives of 8.1 and 24.2 hours were reported in patients with CC above 39 mL/minute and between 8 and 38 mL/minute, respectively. It was suggested that an increase in the dosage interval to 48 or 72 hours may be necessary to compensate for longer half-lives. Caution is required when sotalol is used in patients on dialysis; a half-life of 33.9 hours was reported in patients with renal failure but this fell to 5.8 hours during dialysis which removed about 43% of sotalol.

1. Berglund G, *et al.* Pharmacokinetics of sotalol after chronic administration to patients with renal insufficiency. *Eur J Clin Pharmacol* 1980; **18:** 321–6.
2. Blair AD, *et al.* Sotalol kinetics in renal insufficiency. *Clin Pharmacol Ther* 1981; **29:** 457–63.

Preparations

BP 2008: Sotalol Injection; Sotalol Tablets;
USP 31: Sotalol Hydrochloride Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Darob†; Sotacor; ***Austral.:*** Cardol; Solavert; Sotab†; Sotacor; Sotahexal; ***Austria:*** Darob; Sotacor; Sotahexal; Sotamed; Sotanorm; Sotastad; Ventricor†; ***Belg.:*** Sotalex; ***Braz.:*** Sotacor; ***Canad.:*** Sotacor†; ***Chile:*** Hipecor; ***Cz.:*** Darob†; Rentibloc†; Sotahexal; Sotalex; ***Denm.:*** Dutacor; Sotabet†; Sotacor; ***Fin.:*** Sotacor; Sotalin; ***Fr.:*** Sotalex; ***Ger.:*** CorSotalol†; Darob; Favorex; Gilucor; Jutalex; Rentibloc; Sota; Sota Lich; Sota-Puren; Sota-saar; Sotabeta; Sotagamma; Sotahexal; Sotalex; Sotalodoc; Sotaryt†; Sotastad; ***Hong Kong:*** Sotacor; ***Hung.:*** Sotahexal; Sotalex; ***Irl.:*** Sotacor; Sotoger; ***Israel:*** Sotacor†; ***Ital.:*** Rytmobeta; Sotalex; ***Jpn:*** Sotacor†; ***Malaysia:*** Sotacor; ***Mex.:*** Sotaper; ***Neth.:*** Sotacor; ***Norw.:*** Sotacor; ***NZ:*** Sotacor; Sotahexal; ***Philipp.:*** Sotalex; ***Pol.:*** Biosotal; Darob; Sotahexal; ***Port.:*** Darob; ***Rus.:*** Sotahexal (Сотагексал); Sotalex (Сталекс); ***S.Afr.:*** Sotacor; Sotahexal; ***Singapore:*** Sotacor; ***Spain:*** Sotapor; ***Swed.:*** Sotabet†; Sotacor; ***Switz.:*** Sotalex; ***Turk.:*** Darob; Sotarit; Talozin; ***UK:*** Beta-Cardone; Sotacor; ***USA:*** Betapace.

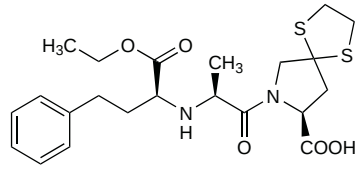
Multi-ingredient: ***S.Afr.:*** Sotazide.

Spirapril Hydrochloride *(BANM, USAN, rINNM)*

Hidrocloruro de espirapril; Sch-33844; Spirapriilihydrokloridi; Spirapril, chlorhydrate de; Spirapril-hydrochlorid; Spiraprilhydroklorid; Spiraprili hydrochloridum; Spiraprilio hidrochloridas; TI-211-950. (*S*)-7-{*N*-[(*S*)-1-Ethoxycarbonyl-3-phenylpropyl]-L-alanyl}-1,4-dithia-7-azaspiro[4.4]nonane-8-carboxylic acid hydrochloride.

Спираприла Гидрохлорид

$C_{22}H_{30}N_2O_5S_2,HCl$ = 503.1.
CAS — 83647-97-6 (spirapril); 94841-17-5 (spirapril hydrochloride).
ATC — C09AA11.
ATC Vet — QC09AA11.



(spirapril)