with saturable binding to the angiotensin-converting enzyme. The clearance of ramiprilat is reduced in renal impairment.

◊ Reviews.

1. Meisel S, *et al*. Clinical pharmacokinetics of ramipril. *Clin Pharmacokinet* 1994; **26:** 7–15.
2. van Griensven JMT, *et al*. Pharmacokinetics, pharmacodynamics and bioavailability of the ACE inhibitor ramipril. *Eur J Clin Pharmacol* 1995; **47:** 513–8.
3. Fillastre JP, *et al*. Kinetics, safety, and efficacy of ramipril after long-term administration in hemodialyzed patients. *J Cardiovasc Pharmacol* 1996; **27:** 269–74.

Uses and Administration

Ramipril is an ACE inhibitor (p.1193). It is used in the treatment of hypertension (p.1171), heart failure (p.1165), and after myocardial infarction (p.1175) to improve survival in patients with clinical evidence of heart failure. It is also used to reduce the risk of cardiovascular events in patients with certain risk factors (see Cardiovascular Risk Reduction, p.1164).

Ramipril owes its activity to ramiprilat to which it is converted after oral doses. The haemodynamic effects are seen within 1 to 2 hours of a single oral dose and the maximum effect occurs after about 3 to 6 hours, although the full effect may not develop for several weeks during chronic dosing. The haemodynamic effect is maintained for at least 24 hours, allowing once-daily dosing.

In the treatment of **hypertension** an initial oral dose of 1.25 mg once daily is given. Since there may be a precipitous fall in blood pressure when starting therapy with an ACE inhibitor, the first dose should preferably be given at bedtime. Patients taking diuretics should, if possible, have the diuretic stopped 2 to 3 days before starting ramipril, and resumed later if necessary. The usual maintenance dose is 2.5 to 5 mg daily as a single dose, although up to 10 mg daily may be required. In the USA an initial dose of 2.5 mg once daily in hypertensive patients not taking a diuretic and a maintenance dose of 2.5 to 20 mg daily, as a single dose or in two divided doses, have been suggested.

In the management of **heart failure**, severe first-dose hypotension on introduction of an ACE inhibitor is common in patients on loop diuretics, but their temporary withdrawal may cause rebound pulmonary oedema. Thus treatment should begin with a low dose under close medical supervision; high doses of diuretics should be reduced before starting ramipril. Ramipril is given in an initial dose of 1.25 mg once daily. The usual maximum dose is 10 mg daily; doses of 2.5 mg or more daily may be taken in 1 or 2 divided doses.

After **myocardial infarction**, treatment with ramipril may be started in hospital 3 to 10 days after the infarction at a usual initial dose of 2.5 mg twice daily, increased after two days to 5 mg twice daily. The usual maintenance dose is 2.5 to 5 mg twice daily.

For the **prophylaxis of cardiovascular events** in patients considered to be at high risk, ramipril is given in an initial dose of 2.5 mg once daily. The dose should be increased, if tolerated, to 5 mg once daily after 1 week, then to the usual maintenance dose of 10 mg once daily after a further 3 weeks. In patients with hypertension or recent myocardial infarction it may also be given in divided doses.

A reduction in dosage of ramipril may be necessary in patients with impaired hepatic or renal function (see below).

◊ References.

1. Todd PA, Benfield P. Ramipril: a review of its pharmacological properties and therapeutic efficacy in cardiovascular disorders. *Drugs* 1990; **39:** 110–35.
2. The Acute Infarction Ramipril Efficacy (AIRE) Study Investigators. Effect of ramipril on mortality and morbidity of survivors of acute myocardial infarction with clinical evidence of heart failure. *Lancet* 1993; **342:** 821–8.
3. Frampton JE, Peters DH. Ramipril: an updated review of its therapeutic use in essential hypertension and heart failure. *Drugs* 1995; **49:** 440–66.
4. The Heart Outcomes Prevention Evaluation Study Investigators. Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. *N Engl J Med* 2000; **342:** 145–53.
5. Warner GT, Perry CM. Ramipril: a review of its use in the prevention of cardiovascular outcomes. *Drugs* 2002; **62:** 1381–1405.
6. Vuong AD, Annis LG. Ramipril for the prevention and treatment of cardiovascular disease. *Ann Pharmacother* 2003; **37:** 412–19.
7. Rokoss MJ, Teo KK. Ramipril in the treatment of vascular diseases. *Expert Opin Pharmacother* 2005; **6:** 1911–19.
8. Anderson VR, *et al*. Ramipril: a review of its use in preventing cardiovascular outcomes in high-risk patients. *Am J Cardiovasc Drugs* 2006; **6:** 417–32.
9. Lüders S, *et al*.. The PHARAO study: prevention of hypertension with the angiotensin-converting enzyme inhibitor ramipril in patients with high-normal blood pressure - a prospective, randomized, controlled prevention trial of the German Hypertension League. *J Hypertens* 2008; **26:** 1487–1496.

Administration in hepatic or renal impairment. The UK licensed product information states that in patients with hepatic impairment, or renal impairment with a creatinine clearance of less than 30 mL/minute, the initial dose of ramipril should not exceed 1.25 mg daily. In hepatic impairment higher doses should be used with caution. In renal impairment the maintenance dose should not exceed 5 mg daily; for those with a creatinine clearance of less than 10 mL/minute, the maintenance dose should not exceed 2.5 mg daily.

Preparations

BP 2008: Ramipril Capsules; Ramipril Tablets.

Proprietary Preparations (details are given in Part 3)

Arg.: Lostapres; Tritace; ***Austral.:*** Prilace; Ramace; Tritace; ***Austria:*** Hypren; Lannapril; Ramipharm; Tritace; ***Belg.:*** Ramace; Tritace; ***Braz.:*** Atensec; Ecator; Naprix; Triatec; ***Canad.:*** Altace; ***Chile:*** Ramipres; Triatec; ***Cz.:*** Acesial; Amprilan; Hartil; Miril; Piramil; Ramicard; Ramil; Ramistada; Ramitren; Tritace; ***Denm.:*** Ramace; Triatec; ***Fin.:*** Cardace; Ramace†; ***Fr.:*** Triatec; ***Ger.:*** Delix; Ramicard; Ramigamma; RamiLich; Vesdil; ***Gr.:*** Stibenyl; Triatec; ***Hong Kong:*** Tritace; ***Hung.:*** Amprilan; Corpril; Emren; Hartil; Meramyl; Piramil; Ramace; Ramicard; Ramiwin; Tritace; ***India:*** Cardace; Hopecard; Preface; R-Pril; Ramcor; Ramipres; Sclerace; ***Indon.:*** Cardace; Hyperil; Ramixal; Tenapril; Triatec; ***Irl.:*** ByTrite; Ramic; Ramilo; Tritace; ***Israel:*** Ramitens; Tritace; ***Ital.:*** Quark; Triatec; Unipril; ***Malaysia:*** Tritace; ***Mex.:*** Intempril; Ramace†; Tritace; ***Neth.:*** Remik†; Tritace; ***Norw.:*** Triatec; ***NZ:*** Tritace; ***Philipp.:*** Tritace; ***Pol.:*** Axtil; Mitrip; Piramil; Ramicor; Tritace; Vivace; ***Port.:*** Ramik; Triatec; Verzatec; ***Rus.:*** Hartil (Хартил); ***S.Afr.:*** Ramace; Ramiwin; Rampil; Tritace; ***Singapore:*** Tritace; ***Spain:*** Acovil; Carasel; ***Swed.:*** Pramace; Triatec; ***Switz.:*** Triatec; Vesdil; ***Thai.:*** Corpril; Piramil; Ramiril; Ramtace; Tritace; ***Turk.:*** Delix; ***UK:*** Tritace; ***USA:*** Altace; ***Venez.:*** Altace; Piramil.

Multi-ingredient: *Arg.:* Triacor; Tritace-HCT; ***Austral.:*** Triasyn; ***Austria:*** Hypren plus; Lannapril plus; Lasitace; Ramicomp; Ramipharm comb; Trialix; Triapin; Tritazide; Unimax; ***Belg.:*** Tritazide; ***Braz.:*** Ecator H; Naprix A; Naprix D; Triatec D; ***Cz.:*** Amprilan H; Hartil-H; Medoram plus H; Miril plus H; Ramil H; Ramixa Plus H; Triasyn; Tritazide; Unimax†; ***Denm.:*** Triatec Comp; ***Fin.:*** Cardace Comp; Unimax; ***Fr.:*** Cotriatec; ***Ger.:*** Arelix ACE; Aretensin; Delix Plus; Delmuno; Rami-Q comp; Ramicard Plus; Ramigamma HCT; RamiLich comp; Ramiplus; Ramipril comp; Ramipril HCT; Ramipril HCTad; Ramipril Plus; Unimax; Vesdil plus; ***Gr.:*** Stibenyl HCT; Triacor; Triatec Plus; Unitens; ***Hung.:*** Amprilan HD; Amprilan HL; Hartil HCT; Meramyl HCT; Ramace Plusz; Ramiwin HCT; Triasyn; Tritace-HCT; ***India:*** Ramcor H; Ramipres H; ***Irl.:*** Trialix; Triapin; ***Israel:*** Tritace Comp; ***Ital.:*** Idroquark; Prilace; Triatec HCT; Uniprildiur; ***Mex.:*** Triacor; Tritazide; ***Neth.:*** Delitab-HCT; Prilitab-HCT; Prilitaril-HCT; Ramitab-HCT; Rataril-HCT; Triapin; Tritazide; Unimax; ***Philipp.:*** Triapin; ***Pol.:*** Ramicor Comb; ***Port.:*** Ramicor D; Triatec Composto; Unimax; ***S.Afr.:*** Tri-Plen; ***Swed.:*** Triatec Comp; ***Switz.:*** Trialix; Triatec Comp; Unimax; ***Turk.:*** Delix Plus; ***UK:*** Triapin; ***Venez.:*** Altace Plus.

Ranolazine *(USAN, rINN)*

CVT-303; Ranolazina; Ranolazinum; RS-43285-003. (±)-*N*-(2,6-Dimethylphenyl)-4-[2-hydroxy-3-(2-methoxyphenoxy)propyl]-1-piperazineacetamide.

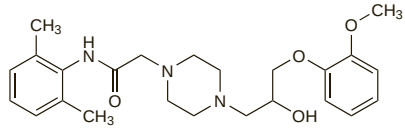
Ранолазин

$C_{24}H_{33}N_3O_4$ = 427.5.

CAS — 95635-55-5.

ATC — C01EB18.

ATC Vet — QC01EB18.



Ranolazine Hydrochloride *(USAN, rINNM)*

Hidrocloruro de ranolazina; Ranolazine, Chlorhydrate de; Ranolazini Hydrochloridum; RS-43285.

Ранолазина Гидрохлорид

$C_{24}H_{33}N_3O_4,2HCl$ = 500.5.

CAS — 95635-56-6.

ATC — C01EB18.

ATC Vet — QC01EB18.

Adverse Effects and Precautions

Adverse effects most commonly seen with ranolazine are nausea, constipation, dizziness, and headache. Palpitations, tinnitus, vertigo, dry mouth, abdominal pain, vomiting, peripheral oedema, and dyspnoea have also been reported. Rarely reported effects include bradycardia, haematuria, paraesthesia, hypotension, and blurred vision.

Dose-related prolongation of the QT interval may occur; ranolazine is therefore contra-indicated in patients with pre-existing QT prolongation, and in those at increased risk of QT prolongation, including patients with hepatic impairment (Child-Pugh classes A to C) and those taking interacting drugs (see Interactions, below). Blood pressure may be increased in patients with severe renal impairment and should be monitored regularly.

Interactions

Ranolazine is primarily metabolised by the P450 isoenzyme CYP3A and should not be given with other drugs that are potent or moderately potent inhibitors of this enzyme, such as ketoconazole, diltiazem, macrolide antibacterials, HIV protease inhibitors, and grapefruit juice or grapefruit products. Simvastatin is also metabolised by this enzyme and plasma concentrations are reported to be doubled when given with ranolazine. Ranolazine is an inhibitor of CYP2D6 and drugs metabolised by this enzyme, such as tricyclic antidepressants or some antipsychotics, may need dose reductions. Ranolazine is both a substrate for, and inhibitor of, P-glycoprotein, and the dose of other substrates, such as digoxin, may need reducing.

Pharmacokinetics

Absorption of ranolazine is highly variable with peak plasma concentrations occurring about 2 to 5 hours after an oral dose of the modified-release preparation. Ranolazine is extensively metabolised in the gastrointestinal tract and liver. Four main metabolites have been identified. Protein binding of ranolazine is about 62%. About 75% of a dose is excreted in the urine with the remainder in the faeces, with less than 5% as unchanged drug. The apparent terminal half-life for the modified-release preparation of ranolazine is 7 hours, and steady state occurs within 3 days.

◊ Reviews.

1. Jerling M. Clinical pharmacokinetics of ranolazine. *Clin Pharmacokinet* 2006; **45:** 469–91.

Uses and Administration

Ranolazine is an antianginal drug. Its mechanism of action is unclear but may involve inhibition of the late sodium current in cardiac myocytes; it also inhibits fatty acid oxidation, but this does not appear to occur at therapeutic plasma concentrations. It is used for the treatment of angina pectoris (p.1157) in patients who have not responded satisfactorily to other antianginals and should be given as an adjunct to standard therapy. It is given in a modified-release form in an initial oral dose of 500 mg twice daily, increasing to a maximum of 1 g twice daily if necessary.

◊ Reviews.

1. Siddiqui MAA, Keam SJ. Ranolazine: a review of its use in chronic stable angina pectoris. *Drugs* 2006; **66:** 693–710.
2. Tafreshi MJ, Fisher E. Ranolazine: a new approach to management of patients with angina. *Ann Pharmacother* 2006; **40:** 689–93.
3. Chaitman BR. Ranolazine for the treatment of chronic angina and potential use in other cardiovascular conditions. *Circulation* 2006; **113:** 2462–72.
4. Zerumsky K, McBride BF. Ranolazine in the management of chronic stable angina. *Am J Health-Syst Pharm* 2006; **63:** 2331–8.

Preparations

Proprietary Preparations (details are given in Part 3)

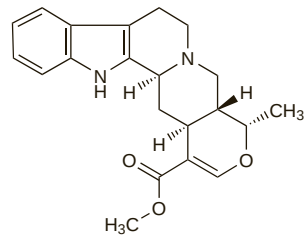
USA: Ranexa.

Raubasine

Ajmalicine; Alkaloid F; Raubasiini; Raubasin; Raubasina; Raubasinum; δ-Yohimbine. Methyl 16,17-didehydro-19α-methyl-18-oxayohimban-16-carboxylate.

$C_{21}H_{24}N_2O_3$ = 352.4.

CAS — 483-04-5.



Pharmacopoeias. In *Chin.*

Profile

Raubasine is an alkaloid obtained from *Rauwolfia serpentina* (Apocynaceae). It is a vasodilator related chemically to reserpine (p.1387) and has been given orally and by injection in peripheral and cerebral vascular disorders.

Preparations

Proprietary Preparations (details are given in Part 3)
Ital.: Lamuran.

Multi-ingredient: ***Austria:*** Defluina; ***Fr.:*** Duxil†; Iskedyl; ***Hong Kong:*** Duxaril; ***Philipp.:*** Duxaril; ***Port.:*** Duxil; Transoxyl†; ***Singapore:*** Duxaril; ***Spain:*** Duxor†; ***Thai.:*** Duxaril; Iso-Triraupin†.

Rauwolfia Serpentina

Chotachand; Rauvolfia; Rauwolfia; Rauwolfiae Radix; Rauwolfiawurzel.
CAS — 8063-17-0 (rauwolfia).
ATC — C02AA04.

Pharmacopoeias. In *Ger.* and *US.*
USP 31 (Rauwolfia Serpentina). The dried roots of *Rauwolfia serpentina* (Apocynaceae). It contains not less than 0.15% of reserpine-rescinnamine group alkaloids calculated as reserpine. Store at 15° to 30° in a dry place.

Profile

Rauwolfia serpentina contains numerous alkaloids, the most active as hypotensive agents being the ester alkaloids, reserpine and rescinnamine. Other alkaloids present have structures related to reserpic acid, but are not esterified, and include ajmaline (rauwolfine), ajmalinine, ajmalicine, isoajmaline (isorauwolfine), serpentine, rauwolfinine, and sarpagine. The actions of rauwolfia serpentina are those of its alkaloids and it has been used for the same purposes as reserpine, p.1387. It has been given orally as the powdered whole root.

Rauwolfia vomitoria has also been used.

A crude form of rauwolfia serpentina has been used in India for centuries as preparations such as Sarpagandha, in the treatment of insomnia and certain forms of mental illness.

Preparations

USP 31: Rauwolfia Serpentina Tablets.

Proprietary Preparations (details are given in Part 3)

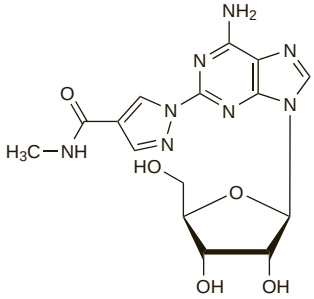
Multi-ingredient: ***Ger.:*** Hyperforat-forte†; ***Rus.:*** Speman Forte (Спеман Форте); ***Spain:*** Rulun; ***USA:*** Rauzide†.

Regadenoson *(BAN, USAN, rINN)*

CVT-3146; Régadénoson; Regadenosón; Regadenosonum. 1-(6-Amino-9-β-D-ribofuranosyl-9*H*-purin-2-yl)-*N*-methyl-1*H*-pyrazole-4-carboxamide monohydrate.

Регаденозон

$C_{15}H_{18}N_8O_5$, H_2O = 408.4.
CAS — 313348-27-5 (regadenoson); 875148-45-1 (regadenoson monohydrate).



Adverse Effects, Treatment, and Precautions

As for Adenosine, p.1202. Regadenoson may be used with caution in patients with asthma or chronic obstructive pulmonary disease.

Interactions

As for Adenosine, p.1202.

Pharmacokinetics

After intravenous injection of regadenoson, peak plasma concentrations are reached within 1 to 4 minutes and decline in a multi-exponential fashion. The initial half-life is about 2 to 4 minutes, followed by an intermediate stage with a half-life of about 30 minutes, during which the pharmacodynamic effect is lost; the half-life during the terminal phase is about 2 hours. Regadenoson does not appear to be metabolised; about 57% of a dose is excreted unchanged in the urine.

◊ References.

1. Gordi T, *et al.* A population pharmacokinetic/pharmacodynamic analysis of regadenoson, an adenosine A -receptor agonist, in healthy male volunteers. *Clin Pharmacokinet* 2006; **45:** 1201–12.
2. Gordi T, *et al.* Regadenoson pharmacokinetics and tolerability in subjects with impaired renal function. *J Clin Pharmacol* 2007; **47:** 825–33.

Uses and Administration

Regadenoson has similar properties to adenosine (p.1202) but has a greater selectivity for the adenosine A_{2A}-receptor. It is a coronary vasodilator and increases coronary blood flow and is used to provide a pharmacological stress as an adjunct to radionuclide myocardial perfusion imaging. It is given intravenously in a single dose of 400 micrograms by rapid injection over about 10 seconds, followed by 5 mL of sodium chloride 0.9%; the radionuclide should be given 10 to 20 seconds after the sodium chloride.

◊ References.

1. Hendel RC, *et al.* Initial clinical experience with regadenoson, a novel selective A agonist for pharmacologic stress single-photon emission computed tomography myocardial perfusion imaging. *J Am Coll Cardiol* 2005; **46:** 2069–75.
2. Iskandrian AE, *et al.* Adenosine versus regadenoson comparative evaluation in myocardial perfusion imaging: results of the ADVANCE phase 3 multicenter international trial. *J Nucl Cardiol* 2007; **14:** 645–58.

Preparations

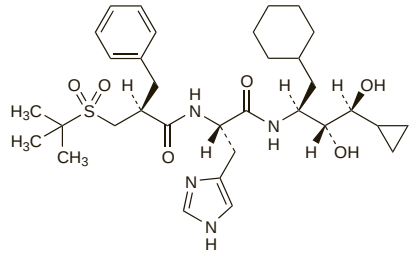
Proprietary Preparations (details are given in Part 3)
USA: Lexiscan.

Remikiren *(rINN)*

Rémikirène; Remikireno; Remikirenum; Ro-42-5892. (α*S*)-α-[(α*S*)-α-[(*tert*-Butylsulfonyl)methyl]hydrocinnamamido]-*N*-[(1*S*,2*R*,3*S*)-1-(cyclohexylmethyl)-3-cyclopropyl-2,3-dihydroxypropyl]imidazole-4-propionamide.

Ремикирен

$C_{33}H_{50}N_4O_6S$ = 630.8.
CAS — 126222-34-2.
ATC — C09XA01.
ATC Vet — QC09XA01.



Profile

Remikiren inhibits the actions of renin and thus prevents the conversion of angiotensinogen into angiotensin I. It is orally active and has been investigated in the management of hypertension and heart failure.

◊ A number of renin antagonists have been investigated as specific inhibitors of the renin-angiotensin system.[1,2] Remikiren is a nonpeptide that is active intravenously and orally, but it is reported to have a very low oral bioavailability.

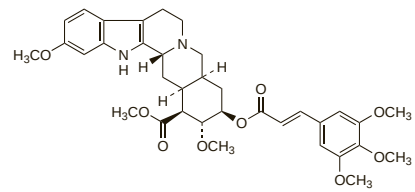
1. Frishman WH, *et al.* Renin inhibition: a new approach to cardiovascular therapy. *J Clin Pharmacol* 1994; **34:** 873–80.
2. Rongen GA, *et al.* Clinical pharmacokinetics and efficacy of renin inhibitors. *Clin Pharmacokinet* 1995; **29:** 6–14.

Rescinnamine *(BAN, rINN)*

Rescinamina; Rescinnamin; Rescinnaminum; Resinnamiini. Methyl-*O*-(3,4,5-trimethoxycinnamoyl)reserpate.

Ресциннамин

$C_{35}H_{42}N_2O_9$ = 634.7.
CAS — 24815-24-5.
ATC — C02AA01.
ATC Vet — QC02AA01.



Profile

Rescinnamine is an ester alkaloid isolated from the root of *Rauwolfia serpentina* or *R. vomitoria*. It has properties similar to those described under reserpine (below) and has been used in the treatment of hypertension.

Preparations

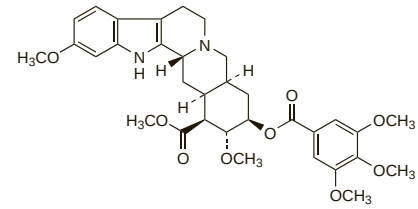
Proprietary Preparations (details are given in Part 3)
Multi-ingredient: ***Thai.:*** Iso-Triraupin†.

Reserpine *(BAN, rINN)*

Reserpiini; Reserpin; Reserpina; Réserpine; Reserpinum; Reszerpin; Rezerpin; Rezerpina; Rezerpinas. Methyl 11,17α-dimethoxy-18β-(3,4,5-trimethoxybenzoyloxy)-3β,20α-yohimbane-16β-carboxylate; Methyl *O*-(3,4,5-trimethoxybenzoyl)reserpate.

Резерпин

$C_{33}H_{40}N_2O_9$ = 608.7.
CAS — 50-55-5.
ATC — C02AA02.
ATC Vet — QC02AA02.



Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), *Int.*, *Jpn*, *US*, and *Viet.*
Ph. Eur. 6.2 (Reserpine). It occurs as small, white to slightly yellow crystals or a crystalline powder. It darkens slowly on exposure to light. Practically insoluble in water; very slightly soluble in alcohol. Protect from light.
USP 31 (Reserpine). A white or pale buff to slightly yellowish, odourless, crystalline powder. It darkens slowly on exposure to light, but more rapidly when in solution. Insoluble in water; soluble 1 in 1800 of alcohol and 1 in 6 of chloroform; freely soluble in acetic acid; very slightly soluble in ether; slightly soluble in benzene. Store in airtight containers at a temperature of 25°, excursions permitted between 15° and 30°. Protect from light.

Stability. Reserpine is unstable in the presence of alkalis, particularly when the drug is in solution.

Adverse Effects

Adverse effects commonly include nasal congestion, headache and CNS symptoms including depression, drowsiness, dizziness, lethargy, nightmares, and symptoms of increased gastrointestinal tract motility including diarrhoea, abdominal cramps, and, at higher doses, increased gastric acid secretion. Respiratory distress, cyanosis, anorexia, and lethargy may occur in infants whose mothers have taken reserpine before delivery.

Higher doses may cause flushing, bradycardia, severe depression which may lead to suicide, and extrapyramidal effects. Hypotension, coma, convulsions, respiratory depression, and hypothermia also occur in overdosage. Hypotension is also more common in patients after a cerebrovascular accident.

Breast engorgement and galactorrhoea, gynaecomastia, increased prolactin concentrations, decreased libido, impotence, sodium retention, oedema, decreased or increased appetite, weight gain, miosis, dry mouth, sialorrhoea, dysuria, rashes, pruritus, and thrombocytopenic purpura have also been reported.

Large doses of reserpine have been shown to be tumorigenic in *rodents*. Several reports have suggested an association between reserpine and the development of neoplasms of the breast (see below) but other surveys have failed to confirm this.

Neoplasms of the breast. Although early studies suggested that the incidence of breast cancer was up to 3 to 4 times greater in hypertensive women treated with rauwolfia preparations than in control groups, analysis[1] of both prospective trials and case-control studies found only a low-grade association between use of rauwolfia preparations and risk of malignancy.

1. Grossman E, *et al.* Antihypertensive therapy and the risk of malignancies. *Eur Heart J* 2001; **22:** 1343–52.

Treatment of Adverse Effects

Withdrawal of reserpine or reduction of the dosage causes the reversal of many adverse effects although mental disorders may persist for months and hypotensive effects may persist for weeks after the cessation of treatment. If overdosage occurs activated charcoal may be considered within 1 hour of ingestion. Treatment is generally supportive and symptomatic. Severe hypotension may respond to placing the patient in the supine position with the feet raised. Direct-acting sympathomimetics may be effective for treatment of severe hypotension, but should be given with caution. The patient must be observed for at least 72 hours.

Precautions

Reserpine should not be used in patients with depression or a history of depression, with active peptic ulcer disease or ulcerative colitis, or in patients with Parkinson's disease. It should also be avoided in phaeochromocytoma.

It should be used with caution in debilitated or elderly patients, and in the presence of cardiac arrhythmias, myocardial infarction, renal insufficiency, gallstones, epilepsy, or allergic conditions such as bronchial asthma.

Reserpine is contra-indicated in patients having ECT and an interval of at least 7 to 14 days should be allowed between the last dose of reserpine and the start of any ECT.