AUSTRALIAN PRODUCT INFORMATION

PRESOLOL™

(labetalol hydrochloride) film coated tablet

1 NAME OF THE MEDICINE

Labetalol hydrochloride

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Each film coated tablet contains 100 mg or 200 mg of labetalol hydrochloride as the active ingredient.

Excipients of known effect: sulfites, lactose and sugars.

For the full list of excipients, see section 6.1 List of Excipients.

3 PHARMACEUTICAL FORM

PRESOLOL 100: 8mm normal convex orange film coated tablet marked LL 100, G on reverse.

PRESOLOL 200: 10.5mm normal convex orange film coated tablet marked LL 200, G on reverse.

4 CLINICAL PARTICULARS

4.1 THERAPEUTIC INDICATIONS

Treatment of all grades of hypertension.

4.2 DOSE AND METHOD OF ADMINISTRATION

PRESOLOL tablets are to be taken orally, preferably after meals.

For all patients, the dosage of PRESOLOL must be titrated against blood pressure response.

The recommended starting dose for all patients is 100 mg to 200 mg twice daily. The dosage should then be increased at weekly intervals until satisfactory control of blood pressure is attained. It is important to follow these dosage instructions and to increase the dosage gradually, in order to minimise side effects.

Effective daily doses vary between 200 mg and 2.4 g. In those patients on high doses, a regimen of three to four doses a day may be necessary.

Hypertension may be controlled by PRESOLOL alone. If PRESOLOL tablets are prescribed with diuretics, or other antihypertensive agents, an additive effect can be expected.

When transferring patients from other treatment regimens, PRESOLOL should be introduced in the manner described above and the dosage of other treatments should be reduced gradually.

4.3 CONTRAINDICATIONS

Beta-blockers are usually contraindicated in a variety of conditions and as labetalol has not yet been fully investigated for its effect on these conditions, they should be treated as contraindications until further information is available.

1. Bronchospasm. Beta-adrenergic blockade of the smooth muscle of bronchi and bronchioles may result in an increased airways resistance. These drugs also reduce the effectiveness of asthma treatment. This may be dangerous in susceptible patients. Therefore, beta-blockers are contraindicated in any patient with a history of airways obstruction or a tendency to bronchospasm. Use of cardioselective beta-blockers can also result in severe bronchospasm. If such therapy must be used, great caution should be exercised. Alternative therapy should be considered

2. Allergic disorders (including allergic rhinitis) which may suggest a predisposition to bronchospasm.

3. Right ventricular failure secondary to pulmonary hypertension

4. Significant right ventricular hypertrophy

5. Sinus bradycardia (less than 45 to 50 beats/minute) or sick sinus syndrome

6. Second and third degree A-V block

7. Shock (including cardiogenic and hypovolaemic shock)

8. Known hypersensitivity to labetalol hydrochloride

9. Congestive heart failure

10. Anaesthesia with agents that produce myocardial depression (e.g. ether, chloroform, cyclopropane)

11. Lactation and early pregnancy (see section 4.6 Fertility, Pregnancy and Lactation – Use in Pregnancy and Use in Lactation)

4.4 SPECIAL WARNINGS AND PRECAUTIONS FOR USE

1. Cardiac failure. Beta-blockade depresses myocardial contractility and may precipitate cardiac failure in some patients with a history of cardiac failure, chronic myocardial insufficiency or unsuspected cardiomyopathy. In patients without a history of cardiac failure, continuing depression of the myocardium may lead to cardiac failure. If cardiac failure develops, the beta-blocker should be withdrawn (see Warning 2 below).

(Note. Although congestive heart failure has been considered to be a contraindication to the use of beta-blockers, there is a growing literature on the experimental use of beta-adrenergic blocking drugs in heart failure. As further trials are needed to identify which patients are most likely to respond to which drugs, beta-blockers should not normally be prescribed for heart failure outside of specialist centres.)

2. Abrupt withdrawal. Care should be taken if beta-blockers have to be discontinued abruptly in patients with coronary artery disease. Severe exacerbation of angina and precipitation of myocardial infarction and ventricular arrhythmias has occurred following abrupt discontinuation of beta-blockade in patients with ischaemic heart disease. Therefore, it is recommended that the dosage be reduced gradually over a period of about 8 to 14 days during which time the patient's progress should be assessed. The drug may be reinstituted temporarily if the angina worsens. If the drug must be withdrawn abruptly, close observation is required. In the peri-operative period, beta-blockers should not be withdrawn, unless there are strong clinical reasons to do so.

3. Concomitant therapy with calcium antagonists. The concomitant use of beta-blockers and calcium antagonists with myocardial depressant and sinus node activity, e.g. verapamil and, to a lesser extent, diltiazem, may cause hypotension, bradycardia and asystole. Extreme caution is required if these drugs have to be used together.

The dihydropyridine calcium antagonists (e.g. nifedipine) have a weaker myocardial depressant effect and can be administered cautiously with beta-blockers. If excessive hypotension develops, the calcium antagonist should be stopped or the dosage reduced.

4. Peripheral circulation. Beta-blockade may impair the peripheral circulation and exacerbate the symptoms of peripheral vascular disease.

5. Antiarrhythmic drugs. Care should be taken when prescribing beta-blockers with antiarrhythmic drugs. Interactions have been reported during concomitant beta-blocker therapy with the Class IA agents disopyramide, and less frequently quinidine; Class IB agents, tocainide, mexiletine and

lidocaine; Class IC agents, flecainide and propaferone (not available in Australia); the Class III agent, amiodarone; and the Class IV antiarrhythmic agents.

6. Prinzmetal angina. There is a risk of exacerbating coronary artery spasm if patients with Prinzmetal or variant angina are treated with a beta-blocker. If this treatment is essential, it should only be undertaken in a Coronary or Intensive Care Unit.

7. Euthyroid hyperthyroxinaemia. The effects of beta-blockers on thyroid hormone metabolism may result in elevations of serum free thyroxine (T4) levels. In the absence of any signs or symptoms of hyperthyroidism, additional investigation is necessary before a diagnosis of thyrotoxicosis can be made.

8. Hepatic injury. Severe hepatocellular injury, confirmed by rechallenge in at least one case, occurs rarely with labetalol therapy. The hepatic injury is usually reversible, but hepatic necrosis and death have been reported. Injury has occurred after both short- and long-term treatment and may be slowly progressive despite minimal symptomatology.

Appropriate laboratory testing should be done at the first sign or symptom of hepatic dysfunction. If there is laboratory evidence of hepatic injury or the patient is jaundiced, labetalol therapy should be stopped and not restarted.

9. Beta-blockers. Beta-blockers, even those with apparent cardioselectivity, should not be used in patients with asthma or a history of obstructive disease unless no alternative treatment is available. In such cases, the risk of inducing bronchospasm should be appreciated, and appropriate precautions taken. If bronchospasm should occur after the use of PRESOLOL, it can be treated with a beta 2-agonist by inhalation, e.g. salbutamol (the dose of which may need to be greater than the usual dose in asthma), and, if necessary, intravenous atropine 1 mg.

There have been reports of skin rashes and/or dry eyes associated with the use of beta-adrenoreceptor blocking drugs. The reported incidence is small and in most cases the symptoms have cleared when treatment was withdrawn. Gradual discontinuation of the drug should be considered if any such reaction is not otherwise explicable.

Anaesthesia and the Peri-operative Period

Beta-blockade may have beneficial effects in decreasing the incidence of arrhythmias and myocardial ischaemia during anaesthesia and the post-operative period. It is currently recommended that maintenance beta-blockade be continued peri-operatively. The anaesthetist must be made aware of beta-blockade because of the potential for interactions with other drugs, resulting in severe bradyarrhythmias and hypotension, the decreased reflex ability to compensate for blood loss, hypovolaemia and regional sympathetic blockade, and the increased propensity for vagal-induced bradycardia. Incidents of protracted severe hypotension or difficulty restoring normal cardiac rhythm during anaesthesia have been reported. Modern inhalational anaesthetic agents are generally well tolerated, although older agents (ether, cyclopropane, methoxyflurane, trichlorethylene) were sometimes associated with severe circulatory depression in the presence of beta-blockade.

Labetalol need not be discontinued prior to anaesthesia but patients should receive intravenous atropine prior to induction.

Synergistic effects of labetalol and halothane on cardiac output and blood pressure have been reported.

Diabetes

Beta-blockers affect glucose metabolism and may mask some important premonitory signs of acute hypoglycaemia, such as tachycardia.

In patients with insulin or non-insulin dependent diabetes, especially labile diabetes, or with a history of spontaneous hypoglycaemia, beta-blockade may result in the loss of diabetic control and delayed recovery from hypoglycaemia. The dose of insulin or oral hypoglycaemic agent may need adjustment.

In one study there was an increase in mean fasting glucose levels during labetalol treatment but no alteration in insulin activity or response to an oral glucose tolerance test.

Other Metabolic Effects

Beta-adrenoreceptors are involved in the regulation of lipid as well as carbohydrate metabolism. Some drugs affect the lipid profile adversely, although the long-term clinical significance of this change is unknown, and the effect appears to be less for drugs with intrinsic sympathomimetic activity.

Use of Catecholamine-depleting Agents

Concomitant use of drugs such as reserpine and guanethidine requires careful monitoring since the added effect of beta-blockade may produce an excessive reduction of the resting sympathetic nervous tone.

Clonidine

Concurrent use of beta-blockers and clonidine should be avoided because of the risk of adverse interaction and severe withdrawal symptoms. If administered concomitantly, the clonidine should not be discontinued until several days after the withdrawal of the beta-blocker.

Phaeochromocytoma

Labetalol has been shown to be effective in lowering blood pressure and relieving symptoms in patients with phaeochromocytoma. However, paradoxical hypertension responses have been reported in a few patients with this tumour; therefore, use caution when administering labetalol to patients with phaeochromocytoma.

In patients with this condition, an alpha-blocking drug (e.g. phentolamine/ phenoxybenzamine) should be considered before administration of labetalol, bearing in mind that this drug has alpha- and beta-blocking properties (see section 5.1 Pharmacodynamic Properties – Mechanism of Action).

Eye and Skin Reactions

Various skin rashes and conjunctival xeroses have been reported with beta-blockers. Cross reactions may occur between beta-blockers, therefore substitutions within the group may not necessarily preclude occurrence of symptoms.

During the long-term treatment with the beta-blocking drug, practolol, a specific rash bearing a superficial resemblance to psoriasis was occasionally described. In a number of patients affected, this rash was accompanied by adverse effects on the eye (xerophthalmia and/or keratoconjunctivitis) of varying severity. This condition is called the oculomucocutaneous syndrome or practolol syndrome. In a few patients, these eye changes occurred independently of a skin rash. On rare occasions, otitis media, sclerosing peritonitis, pericarditis and pleurisy have been reported. Although this syndrome has not been observed in patients taking other beta-blockers, the possibility of such side effects occurring should be borne in mind.

More recently an association between Peyronie's disease, a fibrosing induration of the penis, and various beta-blockers has been suggested but is not proven.

It has been found in animal studies that labetalol binds to the melanin pigment of the uveal tract. The significance of this in humans is not known but regular checks on visual function should be made as a precaution.

Extensive ophthalmological monitoring of 72 patients treated with labetalol at doses of 300 mg to 2.4 g daily for between 6 months and 3 years, and routine monitoring of eye complaints from over 6,000 patients has not revealed any adverse effects on the eye.

Sclerosing Peritonitis

Sclerosing peritonitis has been reported in association with practolol therapy but not with other beta-blockers or labetalol. Nevertheless, the possibility of such a reaction must be borne in mind.

Cross-sensitisation

Cross-sensitisation may occur between beta-blockers and substitution within the group may not necessarily preclude recurrence of symptoms.

Allergic Manifestations

These may be exaggerated by beta-blockade (e.g. allergic rhinitis during the pollen season and allergic reactions to bee and wasp stings). Beta-blockers should be avoided if there is a risk of bronchospasm.

Hyperthyroidism

Since beta-blockers may mask the clinical signs of developing or continuing hyperthyroidism, resulting in symptomatic improvement without any change in thyroid hormone status, special care should be exercised in those patients who are hyperthyroid and are also receiving labetalol as an antihypertensive agent.

Postural Hypotension

Severe postural hypotension has occurred in some patients (see section 4.8 Adverse Effects (Undesirable Effects)). Furthermore, this may be enhanced by the concurrent administration of other vasodilators.

If PRESOLOL tablets are prescribed with diuretics or other antihypertensive agents, an additive effect can be expected. When transferring patients from other treatment regimens, PRESOLOL should be introduced in the manner described in section 4.2 Dose and Method of Administration, and the dosage of other treatments should be reduced gradually.

Individual Variations

There is a large variation in hypotensive response between patients and this may be due to variable rate of absorption and first-pass metabolism during the passage of labetalol through the intestinal wall and liver.

Significant Cardiomegaly

Use in Renal Impairment

Labetalol does not adversely affect renal function and is a particularly suitable drug for use in hypertensive patients with renal disease (see section 5.2 Pharmacokinetic Properties – Pharmacokinetics in Special Populations). In patients with severe renal disease, haemodynamic changes following beta-blockade may impair renal function further.

Use in the Elderly

The bioavailability and half-life of labetalol hydrochloride are increased in the elderly. In addition, the hypotensive response is greater in this age group following administration. Therefore, lower doses of PRESOLOL are likely to be required in elderly patients.

Paediatric Use

There is little reported clinical experience of the use of labetalol in children. Thus, care should be taken in establishing individual dosage requirements in children. Safety and effectiveness in children have not been established.

Effects on Laboratory Tests

Labetalol fluoresces in alkaline solution at an excitation wavelength of 334 nm and a fluorescence wavelength of 412 nm and may, therefore, interfere with the assays of certain fluorescent substances including catecholamines.

The presence of labetalol metabolites in the urine may result in falsely elevated levels of urinary catecholamines, metanephrine, normetanephrine, and vanillylmandelic acid (VMA) when measured by fluorimetric or photometric methods. In screening patients suspected of having a phaeochromocytoma and being treated with labetalol hydrochloride, a specific method, such as high-performance liquid

chromatographic assay with solid phase extraction, should be employed in determining levels of catecholamines.

Labetalol may produce a false positive result when urine is screened for amphetamines. Care must be taken to corroborate any such result with a more specific method.

Labetalol has been shown to reduce the uptake of radioisotopes of metaiodobenzylguanidine (MIBG). Care should therefore be taken in interpreting results from MIBG scintigraphy.

4.5 INTERACTIONS WITH OTHER MEDICINES AND OTHER FORMS OF INTERACTIONS

Postural hypotension may be enhanced by the concurrent administration of other vasodilators.

The interaction of labetalol with methyldopa and with clonidine has been examined on blood pressure and heart rate in animals. The results indicate that labetalol, given together with methyldopa or clonidine, should exert an additional hypotensive effect in human beings who are sensitive to both drugs in the combined therapy.

Concomitant use of tricyclic antidepressants may increase the incidence of tremor.

Cimetidine increases the oral bioavailability of labetalol. Care is required in determining the dose of labetalol in patients who are also taking cimetidine.

Concurrent administration of some NSAIDs may impair the antihypertensive effects of labetalol, possibly due to their inhibition of renal synthesis of vasodilatory prostaglandins. Dosage adjustment may be necessary.

See section 4.4 Special Warnings and Precautions for Use.

4.6 FERTILITY, PREGNANCY AND LACTATION

Effects on Fertility

No data available.

Use in Pregnancy

Pregnancy Category: C

Beta-adrenergic blocking agents may cause bradycardia in the fetus and newborn infant. During the final part of pregnancy and parturition these drugs should therefore only be given after weighing the needs of the mother against the risk to the foetus.

Labetalol is known to cross the placental barrier and has been found to bind to the eyes of fetal animals. Labetalol has been used successfully in the treatment of hypertension arising in the second and third trimester of pregnancy. Labetalol crosses the placental barrier and the possibility of the consequences of alpha- and beta-adrenoreceptor blockade in the fetus and neonate should be borne in mind.

Perinatal and neonatal distress (bradycardia, hypotension, respiratory depression, hypoglycaemia, hypothermia) has been rarely reported. Sometimes these symptoms developed a day or two after birth. Response to supportive measures (e.g. intravenous fluids and glucose) is usually prompt but with severe pre eclampsia, particularly after prolonged intravenous labetalol, recovery may be slower. This may be related to diminished hepatic metabolism in premature babies. Intrauterine and neonatal deaths have been reported but other drugs (e.g. vasodilators, respiratory depressants) and the effects of pre-eclampsia, intrauterine growth retardation and prematurity were implicated. Such clinical experience warns against unduly prolonging high dose labetalol and delaying delivery and against co-administration of hydralazine.

Administration of labetalol in the first trimester of pregnancy is not recommended. Labetalol does not appear to be teratogenic in rats or rabbits, but it is embryolethal when given in a dose of 50 mg/kg orally.

Use in Lactation

Labetalol is excreted in breast milk. No adverse reactions in breastfeeding infants have been reported. It is not recommended for nursing mothers unless the expected benefits outweigh any potential risks.

4.7 EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

Labetalol hydrochloride may cause occasional dizziness or fatigue. Patients should therefore be warned of the possibility of impairment of the ability to drive or operate machinery.

4.8 ADVERSE EFFECTS (UNDESIRABLE EFFECTS)

Labetalol is normally well tolerated. Symptoms of postural hypotension may occur if the initial dosage is too high or if the dose is increased too rapidly.

Cardiovascular Disorders

Postural hypotension may occur if the initial dosage is too high or if the dose is increased too rapidly. Occasionally bradycardia and heart block have been reported.

Central and Peripheral Nervous System

Transient dizziness, headache, tiredness, depressed mood and lethargy may occur. There have been reports of a tingling sensation of the skin (especially of the scalp) associated with labetalol treatment usually occurring early in treatment and which is transient in nature.

Collagen Disorders

There have been occasional reports of positive anti¬nuclear antibodies unassociated with disease as well as the occasional case of systemic lupus erythematosus and very occasionally drug fever.

Ocular Disorders

Blurred vision, eye irritation and dry eyes have been reported.

Hepatic Disorders

Reports of raised liver function tests; jaundice (both hepatocellular and cholestatic) and hepatitis have on occasion been reported, the signs and symptoms of which are usually reversible on withdrawal of the drug. Hepatic necrosis has been reported (see Warnings).

Musculoskeletal Disorders

There has been one report of toxic myopathy. Tremor has been reported in the treatment of hypertension of pregnancy. Muscle cramps have been reported.

Respiratory Disorders

Bronchospasm may occur in susceptible individuals.

Skin and Appendages

Rashes of various types such as generalised maculopapular, lichenoid, urticarial, bullous, lichen planus, psoriasiform and facial erythema, Peyronie's disease and reversible alopecia.

Genitourinary Disorders

Acute retention of urine and difficulty in micturition have occurred during labetalol treatment.

Gastrointestinal Disorders

Epigastric pain, nausea and vomiting.

Hypersensitivity

Rash, pruritis, angioedema and dyspnoea.

Others

Ankle swelling; failure of ejaculation; nasal congestion and sweating.

Reporting Suspected Adverse Effects

Reporting suspected adverse reactions after registration of the medicinal product is important. It allows continued monitoring of the benefit-risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions at www.tga.gov.au/reporting-problems.

4.9 OVERDOSE

Overdosage with labetalol causes excessive hypotension which is posture sensitive and, sometimes, excessive bradycardia. Patients should be laid supine and, if necessary, their legs should be raised to improve the blood supply to the brain. Atropine 3 mg intravenously should be given to relieve the bradycardia. Gastric lavage or induced emesis is recommended if the overdosage is recent (2 to 3 hours). Oliguric renal failure has been reported after massive overdosage of labetalol hydrochloride tablets.

If further measures are required to obtain adequate circulatory function, intravenous noradrenaline may be preferable to isoprenaline, the established pharmacological treatment for excessive cardiac beta-blockade. The recommended starting dose of noradrenaline in patients is 5 to 10 micrograms by intravenous injection, repeated as required, according to the response. Alternatively, noradrenaline may be infused at a rate of 5 micrograms/minute until a satisfactory response is achieved.

For information on the management of overdose, contact the Poisons Information Centre on 13 11 26 (Australia).

5 PHARMACOLOGICAL PROPERTIES

5.1 PHARMACODYNAMIC PROPERTIES

Mechanism of Action

Labetalol antagonises alpha- and beta-adrenoreceptors concurrently by competitive inhibition; it has no intrinsic sympathomimetic activity and less pronounced membrane stabilising activity than propranolol. The beta-blockade is nonselective. Labetalol has two centres of chirality which means that four isomers are possible, each of the isomers having different relative alpha- and beta-blocking activities.

Labetalol lowers blood pressure by blocking alpha-adrenoreceptors in peripheral arterioles and thereby reducing peripheral resistance. Labetalol also concurrently blocks beta-adrenoreceptors, notably in the heart, from the reflexly mediated drive caused by the peripheral vasodilatation. Therefore, the reduction in blood pressure is achieved without cardiac stimulation. Labetalol does not reduce cardiac output at rest, or after moderate exercise. Normally occurring increases in systolic pressure during exercise are, however, lessened by labetalol; corresponding changes in the diastolic pressure are not affected.

The precise relationship between alpha- and beta-blocking effects in contributing to the antihypertensive action is unknown. At therapeutic doses, labetalol is less active at alpha-adrenoreceptors than beta-adrenoreceptors, and adequate vasodilatation is achieved with incomplete blockade of the alpha-adrenoreceptors in the arterioles. The barostatic reflexes remain sufficiently active to prevent postural and exercise hypotension in most patients, but this phenomenon has been observed at all doses and becomes more common in patients with severe hypertension on large doses of the drug.

Clinical Trials

No data available.

5.2 PHARMACOKINETIC PROPERTIES

Absorption

Taken orally, PRESOLOL tablets are well absorbed.

The absolute bioavailability of labetalol is increased when administered with food.

Metabolism

The drug is extensively metabolised by the liver, and possibly in the gut wall, to O-phenyl-glucuronide, N-glucuronide and a glucuronide formed by conjugation at the secondary alcohol group. Peak plasma levels occur at 1 to 2 hours, associated with a reduction in blood pressure. On average, 35 to 40% of an administered dose reaches the circulation.

The plasma half-life is approximately 6 to 8 hours, but a hypotensive effect has been observed up to 11 hours after a given dose.

At therapeutic concentrations, the active drug binds to albumin (52%). Binding also occurs to melanin (0.05%) but the clinical significance is uncertain.

Excretion

Labetalol and its inactive metabolites are excreted by both the liver and the kidneys, so the drug is unlikely to accumulate in the body.

Pharmacokinetics in Special Populations

Whilst impairment of renal function does not require a change in dosage, impairment of hepatic function requires a reduction in dosage, due to the decrease in first-pass metabolism of the drug.

Labetalol does not adversely affect renal function and is a particularly suitable drug for use in hypertensive patients with renal disease.

5.3 PRECLINICAL SAFETY DATA

Genotoxicity

No data available.

Carcinogenicity

No data available.

6 PHARMACEUTICAL PARTICULARS

6.1 LIST OF EXCIPIENTS

The tablet contains the following excipients: microcrystalline cellulose, magnesium stearate, colloidal anhydrous silica, silicon dioxide, sodium starch glycollate, pregelatinised maize starch, sucrose and Opadry Orange OY-LS-23015 (ARTG PI No: 2693).

6.2 INCOMPATIBILITIES

Incompatibilities were either not assessed or not identified as part of the registration of this medicine.

6.3 SHELF LIFE

In Australia, information on the shelf life can be found on the public summary of the Australian Register of Therapeutic Goods (ARTG). The expiry date can be found on the packaging.

6.4 SPECIAL PRECAUTIONS FOR STORAGE

Store below 25°C.

6.5 NATURE AND CONTENTS OF CONTAINER

Container type: bottle (HDPE) with PP child-resistant cap

Pack sizes: 100

Some strengths, pack sizes and/or pack types may not be marketed.

Australian Register of Therapeutic Goods (ARTG)

AUST R 56475 – PRESOLOL 100 labetalol hydrochloride 100mg tablet bottle

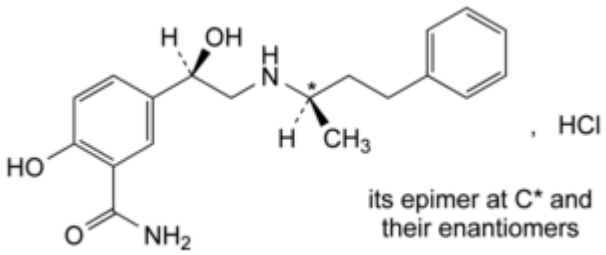
AUST R 56476 – PRESOLOL 200 labetalol hydrochloride 200mg tablet bottle

6.6 SPECIAL PRECAUTIONS FOR DISPOSAL

In Australia, any unused medicine or waste material should be disposed of by taking it to your local pharmacy.

6.7 PHYSICOCHEMICAL PROPERTIES

Chemical Structure



Labetalol hydrochloride is a white to off-white, practically odourless powder. Melting point is about 180°C.

Chemical name: 2-hydroxy-5-[1-hydroxy-2-(1-methyl-3-phenylpropylamino) ethyl]benzamide hydrochloride

Molecular formula: $C_{19}H_{24}N_2O_3HCl$

Molecular weight: 364.9

CAS Number

32780-64-6

7 MEDICINE SCHEDULE (POISONS STANDARD)

S4 (Prescription Only Medicine)

8 SPONSOR

Alphapharm Pty Ltd trading as Viatris

Level 1, 30 The Bond

30 – 34 Hickson Road

Millers Point NSW 2000

www.viatris.com.au

Phone: 1800 274 276

9 DATE OF FIRST APPROVAL

19/07/1996

10 DATE OF REVISION

21/06/2024

Summary Table of Changes

Section Changed	Summary of New Information
2, 3, 6.1	Minor editorial changes
6.4	Update storage condition
6.5	Include child-resistant cap. Insert AUST R numbers
8	Update sponsor’s details

PRESOLOL_pi\Jun24/00