

NEW ZEALAND DATASHEET

Frusehexal

Furosemide, tablet, 40 mg

Presentation

White, 7 mm round, convex plain uncoated tablets, scored on one side. Each tablet contains furosemide 40 mg.

Uses

Actions

Pharmacotherapeutic group

C03CA01 - High-ceiling diuretics, sulfonamides, plain, furosemide.

Mechanism of action

Furosemide, an anthranilic acid derivative, is a potent diuretic. It inhibits sodium and chloride absorption in the ascending limb of the loop of Henle and in both the proximal and the distal tubules. The high degree of efficacy is due to this unique site of action. The action on the distal tubule is independent of any inhibitory effect on carbonic anhydrase or aldosterone.

Pharmacodynamic effects

Furosemide may promote diuresis in cases which have previously proved resistant to other diuretics. Furosemide has no significant pharmacological effects other than on renal function.

Onset and duration of action

The onset of diuresis following oral administration is within 1 hour. The peak effect occurs within the first or second hour. The duration of diuretic effect is 6 to 8 hours.

Pharmacokinetics

Absorption

Furosemide is rapidly absorbed from the gastrointestinal tract. Absorption rates in healthy subjects have been reported to be from 60 to 69% in healthy subjects and from 43 to 46% in patients with end stage renal failure. In fasted normal men, the mean bioavailability of furosemide from furosemide tablets is 64% of that from an intravenous injection of the medicine. Although furosemide is more rapidly absorbed from an oral solution (50 minutes) than from the tablet (87 minutes), peak plasma levels and area under the plasma concentration-time curves do not differ significantly. Peak plasma concentrations increase with increasing dose but times- to- peak do not differ among doses.

Distribution

Furosemide is extensively bound to plasma proteins, mainly to albumin. Plasma concentrations ranging from 1 to 400 mcg/ml are 91 to 99% bound in healthy individuals. The unbound fraction averages 2.3 to 4.1% at therapeutic concentrations.

Biotransformation

Recent evidence suggests that furosemide glucuronide is the only, or at least the major, biotransformation product of furosemide in humans.

Elimination

In patients with normal renal function, approximately 80% of an intravenous or intramuscular dose is excreted in the urine within 24 hours. Significantly more furosemide is excreted in urine following intravenous injection than after the tablet. Urinary excretion is accomplished both by glomerular filtration and proximal tubular secretion, which accounts for roughly 66% of the ingested dose, the remainder being excreted in the faeces. A small fraction is metabolised by cleavage of the side chain. Furosemide has a biphasic half-life in the plasma ranging up to 100 minutes but this is prolonged by renal and hepatic insufficiency and in new born infants.

Indications

Oedema

Furosemide is indicated in adults, infants and children for the treatment of oedema associated with congestive heart failure, cirrhosis of the liver and renal disease, including the nephrotic syndrome. Furosemide is particularly useful when an agent with greater diuretic potential than that of those commonly employed is desired.

Hypertension

Furosemide may be used in adults for the treatment of hypertension alone or in combination with other antihypertensive agents. Hypertensive patients who cannot be adequately controlled with thiazides will probably also not be adequately controlled with furosemide alone.

Dosage and administration

Oedema

Therapy should be individualised according to patient's response. This therapy should be titrated to gain maximal therapeutic response with the minimum dose possible to maintain that diuretic response.

Adults

The usual initial daily dose is 20 to 80 mg given as a single dose. If the diuretic response to a single dose of 20 to 80 mg is not satisfactory, increase this dose by increments of 20 to 40 mg, not sooner than 6 to 8 hours after the previous dose, until the desired diuretic effect is obtained. This individually determined dose should be given once or twice (e.g. at 8 am and 2 pm) daily. The dose of furosemide may be carefully titrated up to 400 mg/day (except in advanced renal failure) in those patients with severe clinical oedematous states. The mobilisation of oedema may be most efficiently and safely accomplished by giving furosemide on 2 to 4 consecutive days each week.

When doses exceeding 80 mg/day are given for prolonged periods, careful clinical laboratory observations are particularly advisable.

Infants and children:

The usual initial dose of oral furosemide for infants and children is 2 mg/kg body weight given as a single dose. If the diuretic response is not satisfactory, the dose may be increased by 1 to 2 mg/kg no sooner than 6 to 8 hours after the previous dose. Doses greater than 6 mg/kg body weight are not recommended.

For maintenance therapy in infants and children, the dose should be adjusted to the minimum effective level.

Hypertension

Therapy should be individualised according to the patient's response. This therapy should be titrated to gain maximal therapeutic response with the minimum dose possible to maintain that therapeutic response.

Adults

The usual initial daily dose of furosemide for hypertension is 80 mg, usually divided into 40 mg twice a day. Dosage should then be adjusted according to response. If response is not satisfactory, additional antihypertensive agents may be necessary.

Changes in blood pressure must be carefully monitored when furosemide is used with other antihypertensive medicines, especially during initial therapy.

To prevent an excessive drop in blood pressure, the dosage of other agents should be reduced by at least 50% when furosemide is added to the regimen. As the blood pressure falls under the potentiating effect of furosemide, a further reduction in dosage or even discontinuation of other antihypertensive medicines may be necessary.

Use in the elderly

No requirement exists for special dosage recommendations in the elderly.

Contraindications

Known hypersensitivity to furosemide, sulfonamides or to any of the inactive ingredients listed in [Further information](#). Patients allergic to sulfonamides (e.g. sulfonamide antibiotics or sulfonyleureas) may show cross sensitivity to furosemide.

Complete renal shutdown

If increasing azotaemia and oliguria occur during treatment of severe progressive renal disease, discontinue furosemide. Severe hypokalaemia, hyponatraemia, hypovolaemia or hypotension must be regarded as contraindications until serum electrolytes, fluid balance and blood pressure have been restored to normal levels.

In hepatic coma or pre-coma and conditions producing electrolyte depletion, furosemide therapy should not be instituted until the underlying conditions have been corrected or ameliorated.

In breast feeding women.

Do not administer furosemide to newborns presenting jaundice or to infants with conditions which might induce hyperbilirubinaemia or kernicterus (e.g. Rhesus incompatibility, familial non-haemolytic jaundice etc.) because of furosemide's *in vitro* potential to displace bilirubin from albumin.

Warnings and precautions

Excessive diuresis may result in dehydration and reduction in blood volume with circulatory collapse and with the possibility of vascular thrombosis and embolism, particularly in elderly patients.

Excessive loss of potassium in patients receiving cardiac glycosides may precipitate digitalis toxicity.

In patients with hepatic cirrhosis and ascites, initiation of therapy with furosemide is best carried out in hospital. Sudden alterations of fluid and electrolyte balance in patients with cirrhosis may precipitate hepatic coma, therefore, strict observation is necessary during the period of diuresis.

Cases of reversible or irreversible tinnitus or hearing impairment have been reported. Usually, reports indicate that furosemide ototoxicity is associated with severe renal impairment, hypoproteinaemia, doses exceeding several times the usual recommended dose, or concomitant therapy with aminoglycoside antibiotics, ethacrynic acid, or other ototoxic medicines. In patients with hypoproteinaemia, e.g. associated with nephrotic syndrome, the effect of furosemide may be weakened and its ototoxicity potentiated. Cautious dose titration is required.

Caution should be exercised when administering curare or its derivatives to patients undergoing furosemide therapy. It is also advisable to discontinue furosemide for one week prior to any elective

surgery.

Rigid sodium restriction is conducive to both hyponatraemia and hypokalaemia, thus strict restriction of sodium intake is not advisable in patients receiving furosemide.

Furosemide should be used with care, especially in the initial stages, in patients with impairment of micturition (e.g. prostatic hypertrophy). Urinary outflow must be secured. In patients with a partial obstruction of urinary outflow (e.g. in patients with bladder-emptying disorders, prostatic hyperplasia or narrowing of the urethra), increased production of urine may provoke or aggravate complaints. Thus, these patients require careful monitoring.

Particularly careful monitoring is required in patients with gout, patients with partial obstruction of urinary outflow, in patients at risk from hypotension (e.g. patients with coronary artery stenosis), in patients with latent or manifest diabetes mellitus, in patients with hepatorenal syndrome or in patients with hypoproteinaemia (e.g. associated with nephrotic syndrome). Dose titration, especially in this latter case, is required. In premature infants, there is the possible development of nephrocalcinosis/nephrolithiasis and therefore renal function must be monitored and renal ultrasonography performed. In premature infants, furosemide administered during the first weeks of life may increase the risk of persistence of Botallo's duct.

Caution should be exercised and the risks and benefits of combining risperidone with furosemide or other potent diuretics should be considered prior to the decision to treat. In the risperidone placebo-controlled trials in elderly patients with dementia, a higher incidence of mortality was observed in patients treated with furosemide plus risperidone (7.3% ; mean age 89 years, range 75 to 97) compared to treatment with risperidone alone (3.1% ; mean age 84 years, range 70 to 96) or furosemide alone (4.1% ; mean age 80 years, range 67 to 90). Concomitant use of risperidone with other diuretics (mainly thiazide diuretics used in low doses) was not associated with similar mortality findings. No pathophysiological mechanism has been identified to explain this finding and no consistent pattern for cause of death was observed. Nevertheless, caution is advised. Irrespective of treatment, dehydration was an overall risk factor for mortality and should, therefore, be carefully avoided in elderly patients with dementia.

As with any effective diuretic, electrolyte depletion may occur during therapy with furosemide, especially in patients receiving higher doses and a restricted salt intake. All patients receiving furosemide therapy should be observed for signs of fluid or electrolyte imbalance; namely hyponatraemia, hypochloraemic alkalosis, and hypokalaemia. Periodic determinations of serum electrolytes to detect a possible imbalance should be performed at appropriate intervals, as well as creatinine, blood urea and carbon dioxide content determinations. Serum and urine electrolyte determinations are particularly important when the patient is vomiting excessively or receiving parenteral fluids. Warning signs of an electrolyte imbalance irrespective of cause, are dryness of mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscular fatigue, hypotension, oliguria, tachycardia, arrhythmia, and gastrointestinal disturbances such as nausea and vomiting. Hypovolaemia or dehydration as well as any significant electrolyte and acid-base disturbances must be corrected. This may require temporary discontinuation of furosemide.

During long-term therapy, a high potassium diet is recommended (lean meat, potatoes, banana, tomatoes, cauliflower, spinach, dried fruit etc.). Potassium supplements may be required, especially when high doses are used for prolonged periods. Particular caution with potassium is necessary when the patient is on digitalis glycosides, potassium depleting steroids or in the case of infants and children. Potassium supplementation, diminution in dose, or discontinuation of furosemide therapy may be required.

Periodic checks on urine and blood glucose should be made in diabetic patients, and even in those suspected of having latent diabetes who are receiving furosemide. Increases in blood glucose and alterations in glucose tolerance tests with abnormalities of the fasting and 2 hour post prandial sugar have been observed, and rare cases of precipitation of diabetes mellitus have been reported.

Furosemide may lower calcium levels, and rare cases of tetany have been reported. Accordingly, periodic serum calcium levels should be obtained.

In children, urge to defecate, complaints of abdominal pain and cramping have been reported after intravenous furosemide. An association of these symptoms with a low serum calcium and/or a low calcium: protein ratio is possible.

Reversible elevations of blood urea may be seen. These have been observed in association with dehydration, which should be avoided, particularly in patients with renal insufficiency.

Furosemide increases cholesterol and triglycerides short-term. It is not clear whether this effect persists long-term; however, the current evidence does not indicate this.

As with many other medicines, patients should be observed regularly for the possible occurrence of blood dyscrasias, liver damage, or other idiosyncratic reactions.

The possibility exists of exacerbation or activation of systemic lupus erythematosus.

Asymptomatic hyperuricaemia can occur and rarely, gout may be precipitated.

Use in the elderly

No requirement exists for special dosage recommendations in the elderly. However, the smaller peak effect of a single dose together with a delay in its effect in conjunction with reduced renal function, as well as possible need for closer monitoring of water and electrolyte balances in the elderly, must be taken into consideration.

Lactose intolerance

Since this medicinal product contains lactose, patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine.

Pregnancy and lactation

Use in pregnancy

Assigned Category C by the Australian Drug Evaluation Committee. This category includes medicines which, owing to their pharmacological effects, have caused or may be suspected of causing, harmful effects on the human foetus or neonate without causing malformations. These effects may be reversible. Accompanying texts should be consulted for further details.

Furosemide must not be given during pregnancy unless there are compelling medical reasons. Treatment during pregnancy requires monitoring of foetal growth.

Diuretics may cause electrolyte disturbances in the foetus. Thiazides, related diuretics and loop diuretics enter the foetal circulation and may cause electrolyte disturbances. Neonatal thrombocytopenia has been reported with thiazides and related diuretics. Loop diuretics, like furosemide and bumetanide, are probably also associated with this risk. During the latter part of pregnancy products of this type should only be given on sound indications, and then in the lowest effective dose.

In pregnancy, furosemide must only be used in patients with a marked reduction in glomerular filtration.

Use in lactation

Furosemide passes into the breast milk and inhibits lactation. Women must not breast feed if being treated with furosemide.

Effects on ability to drive and use machines

This medicine is presumed to be safe or unlikely to produce an effect. Some adverse effects (e.g. an undesirable pronounced fall in blood pressure) may impair the patient's ability to concentrate and react and therefore constitute a risk in situations where these abilities are of special importance (e.g.

operating a vehicle or machinery).

Other

Preclinical safety data

The acute toxicity of furosemide has been determined in mice, rats and dogs. In all three, the oral LD50 exceeded 1,000 mg/kg body weight, while the intravenous LD50 ranged from 300 to 680 mg/kg bodyweight. The acute intragastric toxicity in neonatal rats is 7 to 10 times that of adult rats. The concentration of furosemide in biological fluids associated with toxicity or death is not known.

Adverse effects

As with other diuretics, electrolytes and water balance may be disturbed during therapy with furosemide, especially in patients receiving high doses for a prolonged period.

Excessive diuresis may give rise, especially in elderly patients and children, to circulatory disturbances such as headache, dizziness, dry mouth or visual impairment, as symptoms of hypovolaemia. In extreme cases, hypovolaemia and dehydration may lead to hypotension, circulatory collapse and, in elderly patients in particular, thrombophilia. However, with individualised dosage, acute haemodynamic reactions are generally not to be expected, although diuresis sets in rapidly.

All saluretics may cause hypokalaemia, mainly in cases of low potassium diet, vomiting or chronic diarrhoea.

Factors such as underlying diseases (liver cirrhosis, cardiac failure), concomitant medication (refer to Interactions) or nutritional inadequacies (excessive restriction of salt intake), may lead to sodium or other electrolyte or fluid deficiencies which may produce a fall in orthostatic blood pressure, calf muscle spasms, anorexia, weakness, dizziness, drowsiness, apathy, vomiting and confusion.

Furosemide may lower the serum calcium level which may trigger a state of increased neuromuscular irritability. In very rare cases, tetany has been observed. In premature infants, calcium salts may be deposited in the renal tissue (nephrocalcinosis).

Hypomagnesaemia and in rare cases, tetany or cardiac arrhythmias have been observed as a consequence of increased renal magnesium loss.

Treatment with furosemide may lead to transitory increases in blood creatinine and urea levels. Serum levels of uric acid may increase and attacks of gout may occur.

Pre-existing metabolic alkalosis (e.g. due to decompensated liver cirrhosis) may be aggravated during furosemide treatment.

Gastrointestinal

Reactions with normal doses are uncommon with furosemide. They include anorexia, oral and gastric irritation, nausea, vomiting, cramping, diarrhoea and constipation.

Hepatic

In isolated cases, acute pancreatitis and increases in liver transaminases have been observed. Additionally, intrahepatic cholestasis and jaundice have been reported. Furosemide may increase the bile flow and distend the biliary tree which is already obstructed.

Central nervous system

Reactions such as dizziness, vertigo, paraesthesia, headache and blurred vision occasionally accompany furosemide induced diuresis. Reversible hearing impairment and tinnitus and rarely, permanent tinnitus and impairment of hearing have been observed, especially in patients with markedly reduced renal function or hypoproteinaemia (e.g. in nephrotic syndrome). This occurs particularly in patients who are also receiving medicines known to be ototoxic.

Dermatological

Allergic reactions may occasionally occur in the form of dermatitis, including rash, urticaria and rare cases of exfoliative dermatitis, necrotising angitis, bullous eruptions, erythema multiforme and purpura and pruritus. Photosensitivity reactions have been reported.

Haematological

The following rare adverse reactions have been reported: eosinophilia, haemoconcentration, thrombophlebitis, haemolytic or aplastic anaemia, leukopenia, thrombocytopenia and agranulocytosis. Vasculitis may also occur.

Urinary tract

Excessive diuresis and dehydration could cause transient elevation of creatinine and BUN and reduction of glomerular filtration rate (GFR). In elderly men with prostatic hypertrophy, acute urinary retention with overflow incontinence may occur. Symptoms of existing conditions of obstructed micturition, such as uretostenosis or hydronephrosis, may be triggered or aggravated by pronounced diuresis. Interstitial nephritis has also been reported with furosemide use.

Cardiovascular

Orthostatic hypotension may occur and may be aggravated by alcohol, narcotics and barbiturates. Ischaemic complications have also been reported in elderly patients

Other

Restlessness, hyperuricaemia, fever, rise in serum cholesterol and triglyceride. In patients with hepatocellular insufficiency, hepatic encephalopathy may occur.

Treatment with furosemide has occasionally caused reduced glucose tolerance and deterioration in cases of manifest diabetes, or made latent diabetes manifest.

Rarely, fever or paraesthesiae and occasionally photosensitivity may occur.

In premature infants, furosemide may precipitate nephrocalcinosis/nephrolithiasis. If furosemide is administered to premature infants during the first weeks of life, it may increase the risk of persistence of patent ductus arteriosus.

Due to the possibility of side effects such as hypotension, patients' ability to drive or operate machinery may be impaired, especially at the commencement of therapy.

Anaphylactic shock is rare, but is acutely life-threatening if it does occur.

Whenever adverse reactions are moderate or severe, the furosemide dose should be reduced or the therapy withdrawn.

Interactions

Medicines and other pharmacologically active substances

ACE inhibitors and angiotensin II receptor antagonists

The action of other antihypertensive medicines may be potentiated by furosemide, especially in combination with angiotensin converting enzyme (ACE) inhibitors. The administration of ACE inhibitors to patients pre-treated with furosemide may lead to a deterioration in renal function including renal failure, or may result in severe hypotension, especially when an ACE inhibitor or angiotensin II receptor antagonist is given for the first time or for the first time in an increased dose. Therefore consideration must be given to interrupting the administration of furosemide temporarily or at least reducing the dose of furosemide for 3 days before starting treatment with an ACE inhibitor or increasing the dose of an ACE inhibitor or angiotensin II receptor antagonist.

Amphotericin

The combination of furosemide and amphotericin may result in an excessive loss of potassium.

Antibiotics

Furosemide may increase the ototoxic and nephrotoxic potential of certain antibiotics (e.g. aminoglycosides and some cephalosporins (e.g. cephaloridine), especially in the presence of impaired renal function. Therefore the simultaneous administration of these medicines is not advisable.

Anticonvulsants

Anticonvulsants may decrease the response to furosemide. A combination of furosemide and chloral hydrate may lead to diaphoresis, sensation of heat, flushes, nausea, tachycardia and elevation of blood pressure. As a result, this combination is not recommended.

Antihypertensives

If antihypertensive agents, diuretics or other medicines with blood pressure lowering potential are given concomitantly with furosemide, a more pronounced fall in blood pressure must be anticipated.

Cardiac glycosides

The effects of digitalis preparations and medicines inducing QT interval prolongation syndrome may be potentiated by changes in electrolyte concentrations (e.g. hypokalaemia, hypomagnesaemia) due to furosemide. When a cardiac glycoside is administered concurrently, it should be remembered that potassium or magnesium deficiency increases the sensitivity of the myocardium to digitalis and may increase the toxicity of drugs, which induce QT interval prolongation syndrome.

Carbenoxolone

Carbenoxolone may predispose a patient to hypokalaemia.

Cisplatin

Furosemide should not be used concomitantly with ethacrynic acid or cisplatin because of the possibility of ototoxicity. In addition, nephrotoxicity of cisplatin may be enhanced if furosemide is not given in low doses (e.g. 40 mg in patients with normal renal function) and with positive fluid balance when used to achieve forced diuresis during cisplatin treatment.

Corticosteroids

When a glucocorticoid is administered during diuretic treatment, the potassium-lowering effect of the steroid should be borne in mind (refer to Warnings and precautions).

Cyclosporin A

Concomitant use of cyclosporin A and furosemide is associated with increased risk of gouty arthritis secondary to furosemide-induced hyperuricemia and cyclosporin impairment of renal urate excretion.

Laxatives

Prolonged use of laxatives may predispose a patient to hypokalaemia

Lithium

Furosemide decreases the excretion of lithium salts and may cause increased serum lithium levels, resulting in increased risk of lithium toxicity, including increased risk of cardiotoxic and neurotoxic effects of lithium. Therefore, it is recommended that lithium levels are carefully monitored in patients receiving this combination.

Neuromuscular blocking agents

Interactions between furosemide and neuromuscular blocking agents have been reported. These appear to be dependent on the dose of furosemide and the neuromuscular blocking agent involved. Low doses of furosemide (0.1 to 10 mcg/kg) enhance the neuromuscular blockade of tubocurarine and succinylcholine. High doses (1 to 5 mg/kg) of furosemide have a tendency to antagonise the skeletal muscle relaxing effect of tubocurarine but may potentiate the action of succinylcholine. The

clinical relevance of these findings is uncertain.

Non-steroidal anti-inflammatory drugs (NSAIDs) and salicylates

NSAIDs including acetylsalicylic acid, indomethacin, aspirin may reduce the natriuretic and antihypertensive effects of furosemide in some patients by inhibiting prostaglandin synthesis. In patients with dehydration or pre-existing hypovolaemia, NSAIDs may cause acute renal failure.

Patients receiving high doses of salicylates, as in rheumatic disease, in conjunction with furosemide may experience salicylate toxicity at lower doses because of competitive renal excretory sites.

Radio contrast media

Patients who were at high risk for radio contrast nephropathy treated with furosemide experienced a higher incidence of deterioration in renal function after receiving radio contrast compared to high risk patients who received only intravenous hydration prior to receiving radio contrast.

Risperidone

Caution should be exercised and the risks and benefits of treating a patient on risperidone with furosemide or other potent diuretics should be considered prior to the decision to use. Refer to [Warnings and precautions](#) regarding increased mortality in elderly patients with dementia concomitantly receiving risperidone.

Sucralfate

Oral furosemide and sucralfate must not be taken within two hours of each other because sucralfate reduces the absorption of furosemide from the intestine and hence diminishes its effect.

Sympathomimetic agents

Furosemide may decrease arterial responsiveness to noradrenaline. This diminution is not sufficient to preclude effectiveness of the pressor agent for therapeutic use.

Others

Phenytoin, methotrexate, probenecid and other medicines which, like furosemide, undergo significant renal tubular secretion such as methotrexate and probenecid, may attenuate the effects of furosemide. Conversely, furosemide may decrease renal elimination of these medicines. In the case of high dose treatment (in particular of both furosemide and the other medicines), this may lead to an increased risk of adverse effects due to furosemide or the concomitant medication.

The effects of curare-type muscle relaxants or of theophylline may be enhanced.

It should be borne in mind that the effect of antidiabetics and pressor amines (eg adrenalin and noradrenaline) may be attenuated by furosemide (refer to [Warnings and precautions](#))

Food and alcohol

Whether and to what extent the absorption of furosemide is affected by taking it with food seems to depend on the pharmaceutical formulation of furosemide. It is recommended that furosemide tablets be taken on an empty stomach.

Ingestion of liquorice in large amounts may predispose a patient to hypokalaemia.

Overdosage

Signs and symptoms

The clinical picture in acute or chronic overdose depends primarily on the extent and consequences of electrolyte and fluid loss; e.g. dehydration, blood volume reduction, hypotension, electrolyte imbalance, cardiac arrhythmias (including AV block and ventricular fibrillation), hypokalaemia and hypochlorhaemic alkalosis, and extensions of its diuretic action. Symptoms of these disturbances

include severe hypotension (progressing to shock), acute renal failure, thrombosis, delirious states, flaccid paralysis, apathy and confusion.

Management

No specific antidote to furosemide is known. If ingestion has only just taken place, attempts may be made to limit further systemic absorption of the active ingredient by measures such as gastric lavage or those designed to reduce absorption (e.g. activated charcoal).

Treatment of overdosage is supportive and consists of replacement of excessive fluid and electrolyte losses. Serum electrolytes, carbon dioxide level and blood pressure should be determined frequently. Adequate drainage must be assured in patients with urinary bladder outlet obstruction (such as prostatic hypertrophy). Haemodialysis does not accelerate furosemide elimination.

Pharmaceutical precautions

Instructions for use/handling

Nil.

Incompatibilities

None known.

Special precautions for storage

Store at or below 25°C. Protect from light and moisture.

Medicine classification

Prescription Medicine.

Package quantities

Bottles of 100 tablets.

Further information

Furosemide is also known as frusemide.

List of excipients

Lactose, maize starch, sodium starch glycollate, cellulose, magnesium stearate.

Name and address

Novartis New Zealand Limited
Private Bag 65904 Mairangi Bay
AUCKLAND 0754

Telephone: (09) 361 8100

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