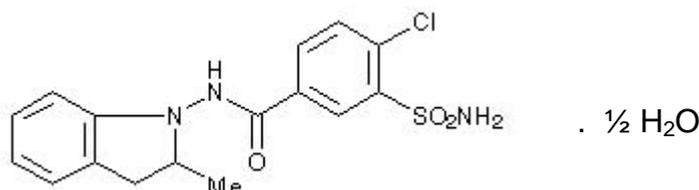


**CHEMMART INDAPAMIDE****NAME OF THE MEDICINE**

Indapamide hemihydrate.

Chemical Name: Indapamide hemihydrate is 4-chloro-N-(2-methyl-1-indolinyl)-3-sulfamoyl benzamide hemihydrate.

Chemical Structure:



Molecular Formula: C<sub>16</sub>H<sub>16</sub>ClN<sub>3</sub>O<sub>3</sub>S, ½ H<sub>2</sub>O

Molecular weight: 374.85.

CAS Registry Number: 26807-65-8.

**DESCRIPTION**

Indapamide hemihydrate is a nonthiazide indole derivative of chlorosulfonamide. It is a white crystalline lipophilic powder, soluble in methanol, ethanol, acetic acid and ethyl acetate, very slightly soluble in ether, chloroform and benzene and practically insoluble in water. Melting point: approximately 185°C.

Each white, biconvex, sugar coated tablet contains 2.5 mg of indapamide hemihydrate as the active ingredient.

In addition, each sugar coated tablet contains the following inactive ingredients: lactose monohydrate, povidone, maize starch, magnesium stearate, Opaseal clear P-2-0300G (ethyl acetate, stearic acid, polyvinyl acetate phthalate, industrial methylated spirit 74 OP), purified talc, calcium carbonate, acacia, titanium dioxide, sucrose, Opaglos 6000P off-white (shellac, industrial methylated spirit 74 OP, beeswax white, carnauba wax).

**PHARMACOLOGY****Pharmacological Actions**

Indapamide is an oral antihypertensive medicine. The mechanism whereby indapamide exerts its antihypertensive action has not been completely elucidated; both vascular and renal actions have been implicated.

At a dose of 2.5 mg the renal effects of indapamide are minimal and the antihypertensive effect of indapamide has been attributed to a reduction in vascular reactivity to pressor amines. The finding that indapamide retains its antihypertensive activity in patients who are functionally anephric lends support to this hypothesis.

The renal site of action of indapamide is the proximal segment of the distal tubule. Indapamide appears to have natriuretic properties (sodium and chloride being excreted in equivalent amounts) with less effect on kaliuresis or uric acid excretion. Only at doses greater than 2.5 mg/day is an appreciable increase in urinary volume observed in humans. No significant changes in plasma sodium levels have been observed in clinical studies. Significant hypokalaemia (plasma potassium < 3.2 mmol/L has been reported in some 10% of patients.

Indapamide (2.5 mg daily) does not adversely affect serum triglycerides, LDL cholesterol, the LDL-HDL cholesterol ratio or glucose tolerance.

## Pharmacokinetics

### Absorption

Possibly related to its high lipid solubility, absorption of indapamide from the gastrointestinal tract is rapid (within 0.5 to 1 hour after an oral dose) and complete. Bioavailability of the tablet formulation is 100% and is virtually unchanged with food or antacids.

### Distribution

Indapamide is widely distributed throughout the body, with extensive binding to specific sites. In blood, it is highly bound to red blood cells (80%) and, more specifically, to carbonic acid anhydrase (98%) without having any significant inhibiting activity on this enzyme. In plasma, it is relatively highly bound to plasma proteins (79%). It is also taken up to a significant degree in the vascular compartment, the drug has a relatively low apparent volume of distribution (approximately 60 L) and 40% of the dose is located in the blood one hour after administration.

### Metabolism

After a single dose of 2.5 mg, as well as after repeated administration of 2.5 mg daily for 15 days, plasma elimination half-life of unchanged indapamide is biphasic with half-lives of 14 and 25 hours, indicating that once daily dosing is possible and that no change in kinetics occurs after repeated dosing. Both single and multiple dose data indicate that indapamide's kinetics are linear. Steady state plasma levels are reached within three to four days after starting treatment, and the drug does not accumulate in hypertensive patients with various degrees of renal insufficiency.

Indapamide is extensively metabolised in the liver, mainly by CYP2C9 and CYP3A4 isozymes and by cytosolic hydrolysis enzymes. Care should be taken when administering indapamide in combination with drugs that alter the activity of these enzymes (see also **INTERACTIONS WITH OTHER MEDICINES**).

### Excretion

Following radioactivity studies using carbon-14, the main route of elimination is the urine, but only 5 to 7% of the dose is excreted into the urine as unchanged drug; 20 to 23% of total radioactivity is eliminated into the faeces. Renal clearance of indapamide (as unchanged drug) is approximately 5 mL/minute, representing less than 10% of systemic clearance.

The high lipid solubility of the indoline moiety confers to indapamide its highly localised binding to structures in the cardiovascular system.

## INDICATIONS

Management of essential hypertension. It may be tried as a sole therapeutic agent in the treatment of mild to moderate hypertension. Normally indapamide is used as the initial agent in multiple drug regimes.

## CONTRAINDICATIONS

- Severe renal failure
- Anuria
- Progressive and severe oliguria
- Hepatic encephalopathy
- Hepatic coma
- Severe hepatic impairment
- Hypokalaemia
- Concomitant administration with non-anti-arrhythmic agents causing torsades de pointes
- Known hypersensitivity to indapamide, other sulphonamide derivatives, or to any of the excipient ingredients in indapamide tablets.

## PRECAUTIONS

Electrolyte changes observed with indapamide become more pronounced at doses above 2.5 mg/day. The daily maximum recommended dose of indapamide is 2.5 mg administered as one tablet, since doses above 2.5 mg only increase the diuretic effect and electrolyte disturbances without any further appreciable antihypertensive effect.

### Hypokalaemia

Hypokalaemia may occur at all doses. Symptoms of hypokalaemia include weakness, cramps, and cardiac dysrhythmias. Hypokalaemia is a particular hazard in patients treated with digoxin as dangerous or fatal arrhythmias may be precipitated.

### Impaired renal function

Although indapamide 2.5 mg can be safely administered to hypertensive patients with renal impairment, caution should be observed when the drug is administered to patients with severe renal impairment. In this case the unchanged drug is excreted primarily by the renal route and plasma concentrations are elevated (see **Pharmacokinetics** and **PRECAUTIONS** sections).

### Uric Acid

Hyperuricaemia may occur during treatment with indapamide and gout has been reported rarely. Tendency to gout attacks may be increased in patients with hyperuricaemia.

### Lithium

Diuretics should not be given with lithium because they reduce its renal clearance and add a high risk of lithium toxicity (see **INTERACTIONS WITH OTHER MEDICINES**).

### Photosensitivity

Cases of photosensitivity reactions have been reported with thiazides and thiazide-related diuretics. It is recommended to stop treatment if a photosensitivity reaction occurs during treatment. If re-administration of the diuretic is deemed necessary, it is recommended that areas exposed to the sun or to artificial UVA are protected.

### Lactose intolerance

Indapamide tablets contain lactose. Patients with an intolerance to lactose, rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine.

### Water and electrolyte balance

Patients receiving indapamide should be monitored for signs and symptoms of fluid or electrolyte imbalance; namely hyponatraemia, hypochloraemia and hypokalaemia. Blood urea, nitrogen and uric acid should also be assessed during treatment.

The signs of electrolyte imbalance are dryness of the mouth, thirst, weakness, lethargy, drowsiness, restlessness, muscle pains or cramps, muscle fatigue, hypotension, oliguria, gastrointestinal disturbances such as nausea and vomiting, tachycardia and ECG changes.

### Plasma sodium

This must be measured before starting treatment, then at regular intervals as any treatment with diuretic may cause hyponatraemia, sometimes with very serious consequences. The decrease in plasma sodium may initially be asymptomatic. Regular monitoring is therefore essential, and should be even more frequent in the elderly and in patients with cirrhosis (see **ADVERSE EFFECTS** and **OVERDOSAGE** sections).

### Plasma potassium

Potassium depletion with hypokalaemia is the major risk of thiazide and related diuretics. The risk of onset of hypokalaemia (< 3.4 mmol/L) must be prevented in certain high risk populations, i.e. the elderly, malnourished and/or polymedicated, cirrhotic patients with oedema and ascites, and patients with coronary artery disease and/or heart failure. In these patients, hypokalaemia increases the cardiac toxicity of digitalis preparations and increases the risk of arrhythmias.

Hypokalaemia will be more common when combined with a steroid or adrenocorticotrophic (ACTH) treatment and when electrolyte intake is inadequate.

Individuals with a long QT interval, whether the origin is congenital or iatrogenic, are also at increased risk as hypokalaemia and bradycardia, are predisposing factors to the onset of severe arrhythmias, in particular, potentially fatal Torsades de pointes.

Plasma potassium should be measured in the first week of treatment. More frequent monitoring of plasma potassium is required in all the situations indicated above. Hypokalaemia, if detected, should be corrected.

### **Plasma calcium**

Diuretic treatment should be withdrawn before the investigation of parathyroid function. Thiazide and related diuretics may decrease urinary calcium excretion and cause a slight and transitory rise in calcium. Frank hypercalcaemia may be due to previously unrecognised hyperparathyroidism.

Caution should be used when treating patients with severe hepatic disease to avoid metabolic alkalosis in cases of potassium depletion which may precipitate episodes of hepatic encephalopathy. Treatment with the diuretic must be stopped immediately if this occurs.

When liver function is impaired, thiazide and thiazide-related diuretics may cause hepatic encephalopathy.

### **Orthostatic hypotension**

Orthostatic hypotension may occur and may be potentiated by alcohol, barbiturates, narcotics or concurrent therapy with other antihypertensives.

When indapamide is combined with other non-diuretic antihypertensive medicines, the effects on blood pressure are additive.

Sulphonamide derivatives have been reported to exacerbate or activate systemic lupus erythematosus.

Serious allergic skin reactions (such as Stevens-Johnson syndrome) have also occasionally been reported to be associated with sulphonamides. This should be considered when using indapamide.

Although an indapamide dose of one 2.5 mg tablet/ day can be used safely in patients with hypertension and renal impairment, treatment should be discontinued if there is an increase in azotaemia and oliguria.

Studies in functionally anephric patients on indapamide monotherapy for one month undergoing chronic haemodialysis have not shown evidence of drug accumulation, despite the fact that indapamide is not dialysable.

A study in patients with impaired renal function demonstrated that patients with severe renal impairment (creatinine clearance 11–35 mL/min) had impaired clearance of indapamide and elevated plasma levels of the drug.

### **Blood glucose**

Monitoring of blood glucose is important in patients with diabetes, in particular in the presence of hypokalaemia.

### **Athletes**

This medicinal product contains a drug substance which may give a positive reaction in doping tests.

### **Impaired Hepatic Function**

Special caution should be used in treating patients with severe hepatic disease to avoid metabolic alkalosis in cases of potassium depletion which may precipitate episodes of hepatic encephalopathy.

**Effects on fertility**

A reproduction study in rats showed no impairment of male or female fertility at oral indapamide doses up to 25 mg/kg/day, however, the number of implantation sites was reduced at the highest dose.

**Use in Pregnancy (Category C)**

Indapamide should be avoided in pregnant women and should not be used to treat oedema in pregnancy.

There are limited data with the use of indapamide in pregnant women. Prolonged exposure to thiazides during the third trimester of pregnancy can reduce maternal plasma volume as well as uteroplacental blood flow, which may cause foetal-placental ischaemia and growth retardation.

Animal studies do not indicate direct or indirect harmful effects with respect to reproductive toxicity.

Indapamide or its metabolites have been shown to cross the placenta and distribute in the foetus in pregnant animals. 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 frusemide and bumetanide are probably also associated with this risk. During the latter part of pregnancy products of this type should be used with caution and at the lowest effective dose.

**Use in Lactation**

Indapamide should not be used during breast-feeding. Indapamide is excreted in human breast milk and the possible effect on the newborn is unknown and cannot be excluded. Indapamide is closely related to thiazide diuretics which have associated with a decrease in, or even suppression of lactation. Hypersensitivity to sulphonamide-derived medicines and hypokalaemia might occur.

**Paediatric Use**

Safety and effectiveness have not been established.

**Genotoxicity**

Indapamide was negative in mutagenicity tests in bacteria and in a bone marrow micronucleus test in mice.

**Carcinogenicity**

Carcinogenicity studies in mice and rats showed no evidence of tumourigenicity when indapamide was administered in the diet at levels up to 100 mg/kg/day.

**Mutagenicity**

Indapamide was negative in mutagenicity tests in bacteria, and bone marrow micronucleus tests in mice. There was a decrease in weight gain of the F1 generation from rats treated orally at 2.5 mg/kg/day. Galactopoiesis was affected in the F1 generation from rats treated orally at 0.5 mg/kg/day and this led to increased mortality of the F2 generation during the first 48 hours of life. No embryo-fetal toxicity or teratogenic potential were seen in rats (up to 150 mg/kg/day) or rabbits (up to 180 mg/kg/day).

**Effect on laboratory tests**

Hyperuricaemia. Hyperglycaemia (see **ADVERSE EFFECTS**).

The following values represent the maximum variations from pre-treatment values in occasional patients at some stage during, but not necessarily throughout, treatment. Blood uric acid up 8.6%, blood glucose up 6%, BUN up 5.7%, blood creatinine up 3.6%.

**INTERACTIONS WITH OTHER MEDICINES**

No interactions have been reported between indapamide and anticoagulants or between Indapamide and uricosuric medicines.

It is recommended that the drug not be used in combination with a diuretic agent since the combination may cause hypokalaemia and hyperuricaemia.

### Combinations that are NOT RECOMMENDED

#### Lithium

Co-administration of indapamide and lithium may result in increased plasma lithium levels and produce symptoms of overdose (due to decreased urinary lithium excretion). However, if the use of diuretics is necessary, careful monitoring of plasma lithium and dose adjustment are required.

### Combinations use which requires special care

#### Torsades de Pointes-inducing drugs:

The combined use of indapamide and *Torsades de pointes*-inducing drugs, including the following, is not recommended due to the increased risk of ventricular arrhythmias, particularly *Torsades de pointes* (hypokalaemia is a risk factor):

- class Ia antiarrhythmics (e.g. disopyramide)
- class III antiarrhythmics (e.g. amiodarone, sotalol)
- some antipsychotics : phenothiazines (e.g. trifluoperazine), benzamides (e.g. amisulpride, sulpiride) and butyrophenones (e.g. droperidol, haloperidol)
- others: diphemanil, erythromycin IV, pentamidine, moxifloxacin.

Monitor (using plasma electrolytes and ECG) for hypokalaemia and correct, if required, before using indapamide and a *Torsades de pointes*-inducing drug in combination.

#### NSAID (Systemic Route) including COX-2 selective inhibitors, high dose salicylic acid ( $\geq 3$ g/day)

Due to the risk of acute renal failure in patients with dehydration as a result of decreased glomerular filtration, it is recommended that hydration and renal function be monitored at the start of treatment. Combined use with NSAIDs may also result in a reduction in the antihypertensive effect of indapamide.

#### Angiotensin converting enzyme (ACE) inhibitors

Combined use with ACE inhibitors in the presence of pre-existing sodium depletion (particularly in patients with renal artery stenosis) may increase the risk of sudden hypotension and/or acute renal failure.

In patients with hypertension when prior diuretic treatment may have caused sodium depletion, it is necessary to either:

- stop the diuretic three days before starting treatment with the ACE inhibitor, and restart a hypokalaemic diuretic if necessary; or
- give low initial doses of the ACE inhibitor and increase the dose gradually.

In patients with congestive heart failure, initiation with a very low dose of ACE inhibitor, possibly after a reduction in the dose of the hypokalaemic diuretic, is recommended.

The monitoring of renal function (plasma creatinine) during the first weeks of treatment with an ACE inhibitor is recommended in all patients.

#### Other compounds causing hypokalaemia: Amphotericin B (IV), glucocorticoids and mineralocorticoids (systemic route), stimulant laxatives.

Due to the increased risk of hypokalaemia (additive effect):

- monitoring, and correction if required, of plasma potassium (especially during treatment with digoxin) is recommended
- the use of non-stimulant laxatives is recommended.

#### Baclofen

Due to the increased risk of antihypertensive effects, it is recommended that hydration and renal function be monitored at the start of treatment.

### Digoxin

Monitoring of plasma potassium and ECG is recommended due to the increased risk of hypokalaemia following co-administration of indapamide and digoxin.

### Allopurinol

Combined use with indapamide may increase the incidence of hypersensitivity reactions to allopurinol.

### **Combinations to be taken into consideration.**

#### Potassium Sparing Diuretics (Amiloride, Spironolactone, Triamterene)

Due to the increased risk of either hyperkalaemia or hypokalaemia (particularly in patients with renal failure or diabetes), care should be taken when co-administering potassium-sparing diuretics. Plasma potassium and ECG should be monitored and, if necessary, treatment reviewed.

### Metformin

Do not co-administer with metformin when plasma creatinine exceeds 15 mg/L (135 µmol/L) in men and 12 mg/L (110 µmol/L) in women due to the increased risk of metformin induced lactic acidosis as a result of the possibility of functional renal failure associated with diuretics and more particularly with loop diuretics.

### Iodinated Contrast Media

Adequate hydration before administration of the iodinated compound is recommended due to an increased risk of acute renal failure resulting from dehydration, particularly when large doses of iodinated contrast media are used.

### Imipramine-like antidepressants, neuroleptics

Caution is recommended with these combinations due to an increased antihypertensive effect and increased risk of orthostatic hypotension.

### Calcium (Salts)

Caution is recommended with this combination due to the risk of hypercalcaemia resulting from decreased urinary elimination of calcium.

### Cyclosporin and Tacrolimus

Caution is recommended with this combination due to the risk of increased plasma creatinine without any change in circulating cyclosporin levels, even in the absence of water/sodium depletion.

### Corticosteroids, (systemic route)

Caution is recommended with this combination due to the risk of decreased antihypertensive effect (water/sodium retention due to corticosteroids).

### **Effects on the ability to drive or operate machinery**

Indapamide does not affect vigilance but different reactions related to a decrease in blood pressure may occur in individual cases, especially at the start of treatment or when another antihypertensive agent is added. Treatment with any blood pressure lowering agent may, therefore, affect the ability to drive, cross the road safely or operate machinery.

### **ADVERSE EFFECTS**

In general, most adverse effects are mild and transient. The most frequently reported are: hypersensitivity reactions, mainly dermatological (in subjects with a predisposition to allergic and asthmatic reactions and macropapular rashes), asthenia, dizziness, headache, fatigue, muscle cramps and gastrointestinal disturbances, usually occurring within the first month of treatment. The majority of adverse reactions concerning clinical or laboratory parameters are dose-dependent. Other adverse reactions have been non-specific. Cutaneous rash and impotence have been occasionally reported. Percentages shown below indicate the incidence in clinical trials.

The following undesirable effects have been observed with indapamide during treatment ranked according to the following frequencies: very common ( $\geq 1/10$ ); common ( $\geq 1/100$ ,  $< 1/10$ ); uncommon ( $\geq 1/1.000$ ,  $< 1/100$ ); rare ( $\geq 1/10.000$ ); very rare ( $< 1/10.000$ ); not known (cannot be estimated from the available data):

Blood and the lymphatic system disorders:

*Very rare:* thrombocytopaenia, leucopaenia, agranulocytosis, aplastic anaemia, haemolytic anaemia

Metabolism and nutrition disorders:

*Very rare:* hypercalcaemia

During clinical trials, hypokalaemia (plasma potassium  $< 3.4$  mmol/L) was seen in 25% of patients and  $< 3.2$  mmol/L in 10% of patients (Potassium supplementation may be required in up to 25% of cases), after 4 to 6 weeks treatment. After 12 weeks treatment, the mean fall in plasma potassium was 0.23 mmol/L. Hypochloraemia 9.4%; hyponatraemia 3.1%

Nervous system disorders:

*Common:* dizziness, headache, fatigue, asthenia, vertigo

*Uncommon:* drowsiness, sleepiness, insomnia, weakness, anxiety, visual disturbance

*Rare:* paresthesia

Cardiac disorders:

*Very rare:* arrhythmia, palpitations, chest pain

Gastrointestinal disorders:

*Uncommon:* vomiting, dyspepsia, abdominal pain, nausea, dry mouth constipation

*Very rare:* pancreatitis

Hepato-biliary disorders:

*Very rare:* abnormal hepatic function

Skin and subcutaneous tissue disorders:

*Common:* hypersensitivity reactions, mainly dermatological, in subjects with a predisposition to allergic and asthmatic reactions, maculopapular rashes

*Uncommon:* purpura, pruritus

*Very rare:* angioedema, urticaria, toxic epidermic necrolysis, Steven Johnson syndrome (see **PRECAUTIONS** section)

Musculoskeletal disorders:

*Common:* muscle cramps

Renal and urinary disorders:

*Uncommon:* cystitis

*Very rare:* renal failure

Vascular disorders

*Very rare:* hypotension

Other adverse reactions, reported in clinical studies with the immediate release formulation of indapamide include the following:

Central Nervous System: lethargy (Incidence  $< 1\%$ ).

Gastrointestinal: anorexia, gastralgia, diarrhoea (Incidence  $< 1\%$ ).

Musculoskeletal: joint pain, back pain, weakness of legs (Incidence  $< 1\%$ ).

Cardiac disorders: tachycardia, ECG changes (non specific ST-T changes, U waves, left ventricular strain) (Incidence  $< 1\%$ ).

Vascular disorders: orthostatic hypotension (Incidence < 1%).

Urogenital: modification of libido, polyuria (Incidence < 1%).

Endocrine: gout (Incidence < 1%).

Other: tinnitus, malaise/fainting, sweat (Incidence < 1%).

Laboratory abnormalities: BUN increase, blood creatinine increase.

**Post-Marketing experience, frequency unknown:**

Metabolism and nutrition disorder: Potassium depletion with hypokalaemia, particularly serious in certain high risk populations (see **CONTRAINDICATIONS** and **PRECAUTIONS** sections). Hyponatraemia with hypovolaemia responsible for dehydration and orthostatic hypotension. Concomitant loss of chloride ions may lead to secondary compensatory metabolic alkalosis: the incidence and degree of this effect are slight.

Nervous system disorders: Syncope

Eye disorders: Myopia, blurred vision, visual impairment

Cardiac disorders: Torsade de pointes (potentially fatal) (see **CONTRAINDICATIONS** and **INTERACTIONS WITH OTHER MEDICINES** sections)

Hepato-biliary disorders: Possibility of onset of hepatic encephalopathy in case of hepatic insufficiency (see **PRECAUTIONS** section), hepatitis

Skin and subcutaneous tissue disorders: Possible worsening of pre-existing acute disseminated lupus erythematosus. Cases of photosensitivity reactions have been reported (see **PRECAUTIONS** section).

Investigations: Electrocardiogram QT prolonged (see **PRECAUTIONS** and **INTERACTIONS WITH OTHER MEDICINES** sections). Elevated liver enzyme levels. Blood glucose increased and blood uric acid increased during treatment: appropriateness of these diuretics must be very carefully weighed in patients with gout or diabetes.

## **DOSAGE AND ADMINISTRATION**

### **Adults**

One tablet (indapamide 2.5 mg) daily to be taken in the morning. The action of indapamide is progressive and whilst the optimum reduction in blood pressure is usually seen after four weeks, a further small but useful reduction in blood pressure may be observed over the following four to six weeks. A larger dose than one tablet (2.5 mg) of indapamide daily is not recommended as there is little additional antihypertensive effect, whilst the diuretic effect becomes more pronounced.

A single tablet of indapamide may effectively be combined with the following antihypertensive agents:  $\beta$ -blockers, methyl dopa, clonidine, prazosin and angiotensin converting enzyme inhibitors.

Combination with a diuretic is not recommended as significant electrolyte disturbances may occur.

Indapamide has a slight but significant carry-over hypotensive effect lasting up to one to two weeks after the treatment is stopped.

## **OVERDOSAGE**

Signs of acute poisoning at higher doses take the form of water/electrolyte disturbances (hyponatraemia, hypokalaemia) and may include the possibility of nausea, vomiting, hypotension, cramps, vertigo, drowsiness, confusion, polyuria or oliguria possibly to the point of anuria (by hypovolaemia). In patients with cirrhosis, an overdose might precipitate hepatic coma.

**Treatment**

There is no specific antidote. Treatment is symptomatic and supportive. Discontinue drug; induce emesis or perform gastric lavage and/or administration of activated charcoal (activated charcoal may reduce absorption of the medicine if given within one or two hours after ingestion. In patients who are not fully conscious or have impaired gag reflex, consideration should be given to administering activated charcoal via a nasogastric tube, once the airway is protected), correct dehydration, electrolyte imbalance, hepatic coma and hypotension by established procedures.

**Contact the Poisons Information Centre on 13 11 26 (Australia) for advice on the management of an overdose.**

**PRESENTATIONS AND STORAGE CONDITIONS**

**Chemmart Indapamide** tablets are intended for oral administration. Each tablet contains 2.5 mg of indapamide hemihydrate

**Chemmart Indapamide 2.5 mg tablets**

White, biconvex, sugar coated tablets.  
PVC/PVDC/Al blister packs of 90 tablets (AUST R 167028).

**Storage**

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

**NAME AND ADDRESS OF THE SPONSOR**

Apotex Pty Ltd  
16 Giffnock Avenue  
Macquarie Park NSW 2113

**POISONS SCHEDULE OF THE MEDICINE**

S4: Prescription Only Medicine.

**DATE OF FIRST INCLUSION IN THE AUSTRALIAN REGISTER OF THERAPEUTIC GOODS (THE ARTG):** 11 January 2010

**DATE OF MOST RECENT AMENDMENT:** 11 August 2015