CARDIOPRIL

Tablets

Composition

Cardiopril 25 Tablets

Each tablet contains Captopril 25 mg.

Action

Captopril, the first of a class of drugs known as ACE inhibitors, is a highly specific competitive inhibitor of angiotensin I converting enzyme (ACE), the enzyme responsible for the conversion of angiotensin I to angiotensin II.

The mechanism of action of Captopril has not yet been fully elucidated. Its beneficial effects as an antihypertensive agent and its efficacy in the management of heart failure appear to result primarily from suppression of the renin-angiotensin-aldosterone system. However, there is no consistent correlation between renin levels and response to the drug. Renin, an enzyme synthesized by the kidneys, is released into the blood circulation where it acts on a plasma globulin substrate to produce angiotensin I, a relatively inactive decapeptide. Angiotensin I is then converted by angiotensin I converting enzyme to angiotensin II, a potent endogenous vasoconstrictor.

Angiotensin II also stimulates aldosterone secretion from the adrenal cortex, thereby contributing to sodium and fluid retention.

Captopril prevents the conversion of angiotensin I to angiotensin II by inhibition of ACE, resulting in a decrease in plasma angiotensin II. The loss of negative feedback on renin release caused by a reduction in angiotensin II results in increased plasma renin activity. The reduction in angiotensin II also leads to decreased aldosterone secretion that, in turn, may result in small increases in serum potassium along with sodium and fluid loss.

Captopril may also interfere with the degradation of the vasodepressor peptide, bradykinin. Increased concentrations of bradykinin or prostaglandin E2 may also have a role in the therapeutic effect of Captopril.

The antihypertensive effect of Captopril persists for a longer period than do demonstrable inhibition of circulating ACE. It is not known whether the ACE present in vascular endothelium is inhibited longer than the ACE in circulating blood.

Pharmacodynamics

Administration of Captopril results in a reduction of peripheral arterial resistance in hypertensive patients with either no change, or an increase in cardiac output. Renal blood flow is increased; glomerular filtration rate is usually unchanged.

Maximum blood pressure reduction occurs 60-90 minutes after the administration of an oral dose of Captopril, the duration of the effect being dose-related. Because the reduction in blood pressure may be progressive, several weeks of therapy may be required in order to achieve maximal therapeutic benefit. While the antihypertensive effects of Captopril and thiazide-type diuretics are additive, concomitant administration of Captopril and ß-blockers have a less than additive effect.

Blood pressure is lowered to about the same extent in both standing and supine positions. Orthostatic effects and tachycardia are infrequent but may occur in volume-depleted patients. Abrupt withdrawal of Captopril has not been associated with a rapid increase in blood pressure.

In patients with heart failure, significantly decreased peripheral (systemic vascular) resistance and blood pressure (after load), reduced pulmonary capillary wedge pressure (preload) and pulmonary vascular resistance, increased cardiac output, and increased exercise tolerance time have been demonstrated. These hemodynamic and clinical effects occur after the first dose and appear to persist for the duration of therapy.

Animal studies indicate that Captopril does not cross the blood-brain barrier to any significant extent.

Pharmacokinetics
Following oral administration, Captopril is rapidly absorbed with peak blood levels at about 1 hour, with the average minimal absorption being about 75%. Since the presence of food in the gastrointestinal tract reduces absorption by about 30-40%, Captopril should be taken 1 hour before meals. In a 24-hour period, over 95% of the absorbed drug is eliminated in the urine; 40-50% is unchanged drug, the remainder is the disulfide dimer of Captopril and Captopril-Cysteine disulfide.

Approximately 25-30% of the circulating drug is bound to plasma proteins. The apparent elimination half-life of total Captopril is probably less than 3 hours, while that of unchanged Captopril is probably less than 2 hours.

Indications

- All grades of hypertension.
- Congestive heart failure.

Contraindications

- History of previous hypersensitivity to Captopril or another ACE inhibitor (e.g. a patient who has experienced angioedema during therapy with another ACE inhibitor).
- Captopril should not be used in patients with aortic stenosis or outflow tract obstruction.
- As limited experience has been obtained in the treatment of acute hypertensive crisis, the use of Captopril should be avoided in these patients.
- Pregnancy and breastfeeding.

Warnings

Angioedema
Angioedema has been seen in patients treated with ACE inhibitors, including Captopril. Patients should be instructed to immediately report to their physician any signs or symptoms of angioedema (e.g. swelling of the face, eyes, lips, tongue, larynx, extremities, difficulty in swallowing or breathing, hoarseness) and to discontinue Captopril therapy immediately.

Where there is involvement of the tongue, glottis or larynx, airway obstruction may occur and be fatal. Emergency therapy, including but not necessarily limited to subcutaneous administration of a 1:1000 adrenaline solution should be promptly instituted.

Where swelling is confined to the face, lips, mucous membranes of the mouth and extremities, the condition will usually resolve with discontinuation of Captopril without further treatment, although antihistamines may be useful in relieving symptoms. These patients should be followed carefully until the swelling has resolved.

Anaphylactoid Reactions
Anaphylactoid reactions have been reported in patients receiving ACE inhibitors and undergoing hemodialysis with high-flux dialysis membranes, or low-density lipoprotein apheresis with dextran sulfate absorption.

Caution should be exercised in patients treated with ACE inhibitors undergoing desensitization procedures. Anaphylactoid reactions have been reported.

Neutropenia/ Agranulocytosis
Neutropenia (neutrophil count <1000/mm3) with myeloid hypoplasia has resulted from the use of Captopril. About half of the neutropenic patients developed systemic or oral cavity infections or other features of the syndrome of agranulocytosis.

The risk of neutropenia is dependent on the clinical status of the patient:

In hypertensive patients with normal renal function (serum creatinine <1.6 mg/dl) and no collagen disease, neutropenia occurs very rarely (in clinical trials, neutropenia was seen in 1 patient out of over 8,600 exposed).

In patients with some degree of renal failure (serum creatinine <1.6 mg/dl) but no collagen vascular disease, the risk of neutropenia in clinical trials was about 1 per 500. Daily doses were relatively high in these patients, particularly in view of their diminished renal function. Concomitant use of Captopril and allopurinol in patients with renal failure has been associated with neutropenia.

In patients with collagen vascular diseases (e.g. systemic lupus erythematosus, scleroderma) and impaired renal function, neutropenia occurred in 3.7% of patients in clinical trials.

While none of the over 750 patients in formal clinical trials of heart failure developed neutropenia, it has occurred during subsequent clinical experience. Of reported cases, about half had serum creatinine $\geq$1.6 mg/dl and more than 75% were also receiving procainamide.

It therefore appears that the same risk factors for neutropenia are present in patients treated with Captopril for heart failure.

Neutropenia has usually been detected within 3 months after starting Captopril therapy. Bone marrow examinations in patients with neutropenia consistently showed myeloid hypoplasia, frequently accompanied by erythroid hypoplasia and decreased numbers of megakaryocytes (e.g. hypoplastic bone marrow and pancytopenia). Anemia and thrombocytopenia were sometimes seen.

Neutrophils generally returned to normal in about 2 weeks after Captopril was discontinued, and serious infections were limited to clinically complex patients. About 13 % of the cases of neutropenia have ended fatally, but almost all fatalities were in patients with serious illness, having collagen vascular disease, renal failure, heart failure or immunosuppressant therapy or a combination of these complicating factors.

Evaluation of the hypertensive or heart failure patient should always include assessment of renal function.

If Captopril is used in patients with impaired renal function, white blood cell and differential counts should be evaluated prior to starting treatment and at approximately 2-week intervals for about 3 months, then periodically.

In patients with collagen vascular disease or patients who are exposed to other drugs known to affect the white cells or immune response, particularly when there is impaired renal function, Captopril should be used only after an assessment of benefit and risk, and then with caution.

All patients treated with Captopril should be instructed to report any signs of infection (e.g. sore throat, fever). If infection is suspected, a differential white blood cell count should be performed immediately. Captopril and other concomitant medication should be withdrawn if neutropenia (neutrophil count <1000/mm3) is detected or suspected. In most patients, neutrophil counts rapidly return to normal upon discontinuing Captopril.

Proteinuria

Proteinuria in patients with prior normal renal function is rare.

Total urinary proteins >l gram/day were seen in about 0.7 % of patients receiving Captopril in clinical trials. About 90% of affected patients had evidence of prior renal disease or received high doses of the drug (>150 mg/day), or both. The nephrotic syndrome occurred in about one fifth of proteinuric patients. In most cases, proteinuria subsided or cleared within 6 months whether or not Captopril therapy was continued. Parameters of renal function, such as BUN and creatinine, were seldom altered in the patients with proteinuria.

Since most cases of proteinuria occurred by the 8th month of therapy with Captopril, patients with prior renal disease or those receiving Captopril at doses >150 mg/day should have urinary protein estimations (dip-stick on first morning urine) before therapy, and periodically thereafter. If repeated determinations show increasing amounts of urinary protein, a 24-hour quantitative determination should be obtained, and if this exceeds 1 gram/day, the benefits and risks of continuing Captopril should be evaluated.

Although membranous glomerulopathy was found in biopsies taken from some proteinuric patients, a causal relationship to Captopril has not been established.

Hypotension
With the first one or two doses of Captopril, some patients treated for hypertension may experience symptomatic hypotension. While excessive hypotension has rarely been seen in hypertensive patients being treated with Captopril, it is a possibility in patients with acute volume depletion (e.g. because of vomiting, diarrhea, or vigorous treatment with diuretics) or in patients undergoing renal dialysis.

In heart failure patients, where blood pressure was either normal or low, transient decreases in mean blood pressure >20% have been recorded in about half of the patients. This transient hypotension is more likely to occur after any of the first several doses and is usually well tolerated, resulting in either no symptoms or brief mild light headedness, although in rare incidences it has been associated with arrhythmia or conduction defects.

Because of the potential fall in blood pressure in these patients, therapy should be initiated under very close medical supervision. A starting dose of 6.25 mg or 12.5 mg 3 times daily may minimize the hypotensive effect. Patients should be monitored closely for the first 2 weeks of treatment and whenever the dose of Captopril and/or diuretic is increased.

Hypotension per itself is not a reason to discontinue Captopril. Some decrease of systemic blood pressure is a common and desirable observation upon initiation of Captopril treatment in heart failure. The magnitude of the decrease is greatest early in the course of treatment; this effect stabilizes in a week or two, and generally returns to pre-treatment levels, without a decrease in therapeutic efficacy, within two months.

Precipitous reduction of blood pressure may occasionally occur within the first hour following administration of the initial Captopril dose in patients on diuretics, especially those in whom diuretics were recently instituted, and in patients on severe dietary salt restriction or dialysis.

Patients with severe and renin-dependent hypertension (e.g. renovascular hypertension) or severe congestive heart failure, who are receiving large doses of a diuretic, are more likely to experience such exaggerated hypotensive responses. This possible hypotensive effect can be minimized by discontinuing diuretic therapy (or significantly reducing diuretic dosage) or increasing salt intake, about 1 week prior to initiation of Captopril therapy, or by initiating therapy with small doses (6.25 mg or 12.5 mg). Alternatively, medical supervision should be provided for at least 1 hour after the initial dose.

In most instances, simply the patient lying down relieves symptoms of hypotension. Some patients may benefit from an infusion of saline.

Impairment of fertility
Studies in rats have revealed no impairment of fertility.

Pregnancy
Category D
Captopril is contraindicated in pregnancy and should not be used in women of childbearing potential unless protected by effective contraception.
When pregnancy is detected, Captopril should be discontinued as soon as possible.

Exposure of the mother in the second and third trimesters of pregnancy to ACE inhibitor therapy has been associated with fetal and neonatal injury including hypotension, neonatal skull hypoplasia, anuria, renal failure and death. Oligohydramnios has also been reported, presumably resulting from decreased fetal renal function. Its presence has been associated with fetal limb contractures, craniofacial deformation, and hypoplastic lung development.

Female patients of childbearing age should be informed by their physician of the possible consequences of second and third trimester exposure to ACE inhibitors, and urged to report a pregnancy as soon as possible.

These adverse effects do not appear to have occurred as a result of intrauterine ACE inhibitor exposure limited to the first trimester.

Infants with histories of in utero exposure to ACE inhibition should be closely observed for hypotension, oliguria, and hyperkalemia.

Nursing Mothers
Concentrations of Captopril in human milk are approximately 1% of those in maternal blood. Because of the potential for serious adverse reactions in nursing infants from Captopril, a decision should be made whether to continue breastfeeding or to discontinue the drug, taking into account the importance of Captopril therapy to the mother.

Adverse Reactions
The incidence of adverse reactions to Captopril is principally associated with renal function since primarily the kidney excretes the drug. The dosage of Captopril should not exceed that necessary for adequate control and should be reduced in patients with impaired renal function.

Renal
About 1% of patients developed proteinuria.

Elevated blood urea and creatinine, elevated serum potassium and acidosis.

Renal insufficiency, renal failure, nephrotic syndrome, polyuria, oliguria and urinary frequency have been reported in 0.1 -0.2% of patients.

Hematological
Neutropenia/agranulocytosis has occurred.

Cases of anemia, thrombocytopenia and pancytopenia have been reported.

Dermatological
Rash (usually maculopapular, rarely urticarial), often with pruritus, and sometimes with fever, arthralgia and eosinophilia have occurred in about 4-7% of patients (depending on renal status and dose), mostly during the first 4 weeks of therapy. The rash is usually mild and disappears within a few days of dosage reduction, short-term antihistamine treatment, and/or discontinuation of Captopril treatment. Remission may occur even if Captopril treatment is continued.

Pruritus, without rash, occurs in about 2% of patients. Between 7-10% of patients with skin rash have shown an eosinophilia and/or positive ANA titers. A reversible associated pemphigoid-like lesion and photosensitivity have also been reported.

Flushing or pallor has been reported in 0.2-0.5% of patients.

Cardiovascular
Hypotension may occur.

Tachycardia, chest pain and palpitations have each been observed in about 1% of patients.

Angina pectoris, myocardial infarction, Raynaud's syndrome, and congestive heart failure have each occurred in 0.2-0.3% of patients.

Dysgeusia
About 2-4% of patients (depending on renal status and dose) developed a diminution or loss of taste perception. Taste impairment is reversible and usually self-limited (2-3 months) even with continued use of Captopril. Weight loss may be associated with loss of taste.

Gastrointestinal
Stomtatitis, resembling aphthous ulcers, has been reported.

Rare cases of hepatocellular injury and cholestatic jaundice have been reported. Gastric irritation and abdominal pain may occur.

Angioedema
Angioedema has been reported in approximately 0. 1 % of patients.

Cough
Cough has been reported in 0.5-2% of patients treated with Captopril in clinical trials. Characteristically, the cough is non-productive, persistent and resolves after discontinuation of therapy. Cough induced by ACE inhibitors should be considered as part of the differential diagnosis of cough.

Other
Gastric irritation, abdominal pain, nausea, vomiting, diarrhea, anorexia, constipation, aphthous ulcers, peptic ulcer, dizziness, headache, malaise, fatigue, insomnia, dry mouth, dyspnea, alopecia and paresthesias have been reported in about 0.5-2% of patients, but did not appear at increased frequency compared to placebo or other treatments used in controlled trials.

There have been reports of other clinical adverse effects since Captopril was marketed, for which a causal relationship or an estimation of incidence cannot be determined. These effects include: asthenia, gynecomastia, lymphadenopathy, serum sickness; cardiac arrest, cerebrovascular accident/insufficiency, rhythm disturbances, orthostatic hypotension, syncope; bullous pemphigus, erythema multiforme (including Stevens-Johnson syndrome), exfohative dermatitis; pancreatitis, glossitis, dyspepsia; aplastic and hemolytic anemia; jaundice, hepatitis, including rare cases of necrosis, cholestitis; symptomatic hyponatremia; myalgia, myasthenia; ataxia, confusion, depression, nervousness, somnolence; bronchospasm, eosinophilic pneumonitis, rhinitis; blurred vision; impotence.

As with other ACE inhibitors, a syndrome has been reported which may include fever, myalgia, arthralgia, interstitial nephritis, vasculitis, rash or other dermatological manifestations, eosinophilia and an elevated ESR.

Precautions
Evaluation of the hypertensive or heart failure patient should always include assessment of renal function prior to initiation of Captopril therapy and at appropriate intervals thereafter.

Patients should be cautioned that excessive perspiration and dehydration might lead to a severe drop in blood pressure because of reduction in fluid volume. Other causes of volume depletion such as vomiting or diarrhea may also lead to a drop in blood pressure.

Heart failure patients receiving Captopril should be cautioned against rapid increases in physical activity.

Impaired Renal Function
Some hypertensive patients with renal disease, particularly those with bilateral renal artery stenosis or unilateral renal artery stenosis in a single functioning kidney, have developed increases in BUN and serum creatinine after reduction of blood pressure with Captopril. It may be necessary to reduce Captopril dosage and/or discontinue concomitant diuretic therapy. For some of these patients, normalization of blood pressure and maintenance of adequate renal perfusion may not be possible.

About 20% of heart failure patients develop stable elevations of BUN and serum creatinine >20% above normal or baseline upon long-term treatment with Captopril. Very few (less than 5% of patients), and generally only those with severe pre-existing renal disease, require discontinuation of treatment due to progressively increasing creatinine.

Hyperkalemia
Elevations in serum potassium have been observed in some patients being treated with ACE inhibitors, including Captopril. Patients at risk include those with renal insufficiency, diabetes mellitus, or those taking drugs associated with increases in serum potassium. Potassium sparing diuretics, potassium supplements or potassium containing salt substitutes should not be used routinely, and the patient should be apprised of this precaution.

Major Surgery, Anaesthesia
In patients undergoing major surgery or during anaesthesia with agents that produce hypotension, Captopril will block angiotensin II formation secondary to compensatory renin release. If hypotension occurs and is considered to be due to this mechanism, it can be corrected by volume expansion.

Drug Interactions
Captopril/ Diuretics
Diuretics potentiate the antihypertensive efficacy of Captopril.
A sharp reduction of blood pressure may occasionally occur, usually within the first hour following administration of the initial Captopril dose in patients receiving diuretics.

Captopril/ Vasodilators
Data on the effect of concomitant use of other vasodilators in patients receiving Captopril for heart failure are not available; therefore, nitroglycerin or other nitrates (as used for the management of angina) or other drugs having vasodilator activity should, if possible, be discontinued before starting Captopril. If resumed during Captopril therapy, such agents should be administered cautiously and perhaps at lower dosage.

Captopril/ Agents causing Renin Release
Antihypertensive agents that cause renin release, e.g. thiazide diuretics that activate the renin-angiotensin-aldosterone system, will augment the effect of Captopril.

Captopril/ Agents affecting Sympathetic Activity
The sympathetic nervous system may be especially important in supporting blood pressure in patients receiving Captopril alone or with diuretics. Therefore, use agents affecting sympathetic activity (e.g. ganglion-blocking agents or adrenergic neuron-blocking agents) with caution. ß-Adrenergic blocking drugs add some further antihypertensive effect to Captopril, but the overall response is less than the additive.

Captopril/ Agents increasing Serum Potassium
Since Captopril decreases aldosterone production, elevation of serum potassium may occur.

Potassium-sparing diuretics such as spironolactone, triamterene or amiloride, or potassium supplements should be given only for documented hypokalemia, and then with caution, since they may lead to a significant increase of serum potassium. Salt substitutes containing potassium should also be used with caution .

Captopril/ Indomethacin/ Other Non-steroidal Anti-inflammatory Drugs
It has been reported that indomethacin may reduce the antihypertensive effect of Captopril, especially in cases of low renin hypertension. Other non-steroidal anti-inflammatory drugs (e.g. aspirin) may have a similar effect.

Captopril/ Clonidine
It has been suggested that the antihypertensive effect of Captopril can be delayed when patients treated with clonidine are changed to Captopril.

Captopril/ Allopurinol/ Procainamide
There have been reports of neutropenia and/or Stevens-Johnson syndrome in patients receiving Captopril concomitantly with either allopurinol or procainamide. Although a causal relationship has not been established, these combinations should be used with caution, especially in patients with impaired renal function.

Captopril/ Immunosuppressants
Azathioprine and cyclophosphamide have been associated with blood dyscrasia in patients with renal failure who were also taking Captopril.

Captopril/ Probenecid
The renal clearance of Captopril is reduced in the presence of probenecid.

Captopril/ Lithium
Concomitant use of lithium and ACE inhibitors may result in increased lithium levels and symptoms of lithium toxicity, especially if a diuretic is also being used. Frequent monitoring of serum lithium levels is recommended.

Diagnostic Interference
Captopril may cause a false-positive urine test for acetone.
Small increases in serum potassium concentration frequently occur, especially in patients with renal impairment .

Hyponatremia, particularly in patients receiving a low sodium diet or concomitant diuretics, has been reported.

Transient elevations of BUN and serum creatinine may occur, especially in volume-depleted or salt-depleted patients or those with renovascular hypertension. Rapid reduction of longstanding or markedly elevated blood pressure can result in decreases in the glomerular filtration rate and, in turn, lead to increases in serum creatinine and BUN.
A positive ANA has been reported.

Elevations of liver transaminases, alkaline phosphatase, and serum bilirubin have occurred.

Dosage and Administration
Cardiopril should be taken 1 hour before meals. Patients should be warned against interruption or discontinuation of treatment unless instructed by the physician.

Hypertension
Initiation of therapy requires consideration of recent antihypertensive drug treatment (if any), the extent of blood pressure elevation, salt restriction, and other clinical circumstances. If possible, discontinue the patient's previous drug regimen for 1 week prior to the initiation of Cardiopril therapy.

The possibility of a hypotensive reaction occurring, especially if Cardiopril is about to be initiated in a patient currently receiving diuretic treatment, should be born in mind.

Treatment with Cardiopril should be at the lowest effective dose that should be titrated according to the needs of the patient.

In mild to moderate hypertension, the starting dosage is 12.5 mg twice daily. The usual maintenance dosage is 25 mg twice daily that can be increased incrementally, at 2-4 week intervals, to a maximum of 50 mg twice daily, until a satisfactory response is achieved.

Concomitant sodium restriction may be beneficial when Cardiopril is used alone.
A modest dose of thiazide diuretic (e.g. hydrochlorothiazide 25 mg daily) may be added to the Cardiopril regimen if a satisfactory response has not been achieved. The dose of the diuretic may be increased at 1-2 week intervals to the level of optimum response, or until its maximum recommended dosage for hypertension is reached.

In severe hypertension where standard therapy is ineffective or inappropriate because of adverse effects, the starting dosage is 12.5 mg twice daily. The dosage may be increased incrementally to a maximum of 50 mg three times a day. Cardiopril may be used together with other antihypertensive agents, but the dose of these should be individually titrated. A daily dosage of 150 mg of Cardiopril should not normally be exceeded.

Congestive Heart Failure
Cardiopril therapy must be started under close medical supervision. Cardiopril should be introduced when diuretic therapy (such as furosemide 40-80 mg or equivalent) is insufficient to control symptoms.

A starting dose of 6.25 mg or 12.5 mg may minimize a transient hypotensive effect. The possibility of this occurring can be reduced by discontinuing or reducing diuretic therapy if possible, prior to initiating Cardiopril.
The usual maintenance dosage is 25 mg 2 or 3 times a day that can be increased incrementally, with intervals of at least 2 weeks, until a satisfactory response is achieved. The usual maximum dosage is 150 mg daily.

Elderly Patients
The dosage should be titrated against the blood pressure response and kept as low as possible to achieve adequate control. Since elderly patients may have reduced renal function and other organ dysfunctions, it is suggested that a low dose of Cardiopril be used initially.

Children
Safety and efficacy in children have not been established in (large) well-controlled trials.
Cardiopril is not recommended for the treatment of mild to moderate hypertension in children.

Experience in neonates, particularly premature infants, is limited. Because renal function in infants is less developed than in children and adults, lower doses of Cardiopril should be used, under close medical supervision.

The starting dose should be 0.3 mg/kg body weight up to a maximum of 6 mg/kg body weight daily in divided doses. The dose should be individualized according to the response and may be given 2 or 3 times daily.

Patients with Renal Impairment
Because primarily the kidneys excrete Captopril, patients with impaired renal function will take longer to reach steady-state Captopril levels and will eventually reach higher levels for a given daily dosage than patients with normal renal function. Therefore, these patients may respond to smaller or less frequent doses.

Accordingly, for patients with significant renal impairment, the initial daily dosage of Cardiopril should be reduced, and smaller increments utilized for titration, which should be spaced at 1-2 week

intervals. Once the desired therapeutic effect has been achieved, the dosage should be back titrated to determine the minimal effective dosage.

When concomitant diuretic therapy is required, a loop diuretic such as furosemide, rather than a thiazide diuretic, is preferred in patients with severe renal impairment.

Cardiopril is readily eliminated by hemodialysis.

Over Dosage

The primary concern is correction of possible hypotension because of an overdose. Volume expansion with an intravenous infusion of normal saline is the treatment of choice for restoration of blood pressure. In adults, Captopril may be removed from the general circulation by hemodialysis; there are inadequate data on the efficacy of hemodialysis in neonates and children. Peritoneal dialysis is not effective.

Presentation

Cardiopril 25 Tablets

Box of 20 tablets.