For the use only of Registered Medical Practitioner

1. Generic Name & Brand Name

Mecobalamin, Alpha Lipoic Acid, Inositol, Folic Acid, Chromium Nicotinate, Selenious Acid and Benfotiamine Capsules

Surbex® Star[New]

2. Qualitative and Quantitative Composition:

Each soft gelatin capsule contains:
Mecobalamin I.P…….1500 mcg
Alpha Lipoic Acid I.P…….100 mg
Inositol I.P……...100 mg
Folic Acid I.P……...1.5 mg
Chromium Nicotinate.....200 mcg
Selenious Acid I.P…….....55 mcg
Benfotiamine……...150 mg
Excipients……q.s.
Colours: Ponceau 4R Supra, Ponceau 4R Lake, Sunset Yellow, Brilliant Blue and Titanium Dioxide I.P.

3. Dosage Form & strength

Refer section 1 & 2

4. Clinical particulars

4.1 Therapeutic indication

For Vitamins and Mineral deficiency states in adult patients.

4.2 Posology and method of administration

Posology

For oral administration in adults.
Usual dose is one capsule of Surbex Star [New] to be administered once daily. To be taken as prescribed by the physician.

Method of Administration:

4.3 Contraindications

Surbex Star [New] Soft Gelatin Capsules are contraindicated in the following:

- Hypersensitivity to Mecobalamin, alpha lipoic acid, pyridoxine, folic acid or to any component of the formulation.
- An existing hypervitaminosis.

4.4 Special warnings and precautions for use

Mecobalamin: Mecobalamin is susceptible to photolysis. Thus, Surbex Star [New] Soft Gelatin Capsules should not be exposed to light/moisture. The prolonged use of larger doses of Mecobalamin is not recommended for patients whose occupation requires the handling of mercury

or mercury compounds. Those with early Leber's disease (hereditary optic nerve atrophy) when treated with vitamin B12 can develop swift, and severe optic atrophy. Surbex Star [New] Soft Gelatin Capsules must be advocated with caution in such patients.

Alpha Lipoic Acid: Alpha lipoic acid should be avoided in alcoholics, thiamine deficiency, thyroid disease, and 2 weeks prior to surgery. Blood glucose must be regularly monitored while taking alpha lipoic acid due to its hypoglycemic action.

Pyridoxine: Pyridoxine may decrease the efficiency of levodopa. Pyridoxine should be advocated cautiously with drugs such as oral hypoglycemics, anticonvulsants, furosemide, isoniazid, penicillamine, hydralazine, and oral contraceptives.

Folic Acid: Taking folic acid might mask anemia caused by vitamin B12 deficiency and delay appropriate treatment. Taking folic acid supplements might make seizures worse in people with seizure disorders, particularly in high doses.

4.5 Drug Interactions

Mecobalamin:

- Oral Contraceptives: Serum concentrations of Mecobalamin may be decreased by use of oral contraceptives.
- Chloramphenicol: Chloramphenicol should not be used with Mecobalamin. Parenteral chloramphenicol may attenuate the effect of vitamin B12 in anemia.
- Other Drugs: Excessive alcohol consumption, anti-acne drugs, anti-retrovirals, anti-gout drugs (colchicine), antihypertensives, anti-tubercular drugs (aminosalicylic acid), anti-ulcer drugs, biguanides (metformin), H2 antagonists (cimetidine, ranitidine), proton pump inhibitors, sulfonamides, tetracyclines, aminoglycosides and antiepileptic drugs can reduce vitamin B12 levels. Thus, caution should be exercised while co-administering vitamin B12 with these drugs.
- Drug/Laboratory Test Interactions: Use of antibiotics, methotrexate, and pyrimethamine may invalidate vitamin B12 diagnostic blood assays.

Alpha Lipoic Acid:

There is some concern that antioxidants might decrease the effectiveness of some medications used for cancers. Alpha lipoic acid might decrease blood sugar levels. Taking alpha lipoic acid along with antidiabetic medications such as insulin, sulphonylureas, and glitazones might cause decrease in blood sugar level.

Pyridoxine:

Pyridoxine reduces the effects of levodopa and activity of altretamine. It also decreases serum concentrations of phenobarbital and phenytoin. Pyridoxine may decrease antibiotic activities of erythromycin, kanamycin, streptomycin, doxycycline, and lincomycin. Drugs such as hydralazine, isoniazid, penicillamine, and oral contraceptives may increase the requirements for pyridoxine.

Folic Acid:

- Phenytoin: Folic acid may increase phenytoin metabolism and lower the serum concentration of phenytoin resulting in increased seizure activity. Also, phenytoin may decrease serum folic acid concentrations.
- Methotrexate: Folic acid may decrease a patient's response to methotrexate therapy.
- Barbiturates: Folate reduces serum barbiturate concentrations.
- Other Drugs and Alcohol: Folate deficiency states may be produced by drugs such as antiepileptics, oral contraceptives, anti-tuberculosis drugs, alcohol, and folic acid antagonists such as methotrexate, pyrimethamine, triamterene, trimethoprim, and sulfonamides.
- Drug/Laboratory Test Interactions: Most of the antibiotics, methotrexate and pyrimethamine may invalidate folic acid diagnostic blood assays.

Benfotiamine:
5-fluorouracil inactivates thiamine. Certain medication categories can interact with Benalgis Tablet, including: Antacids (Aluminum hydroxide, calcium carbonate); Diuretics (Furosemide, hydrochlorothiazide); Antibiotics (Ciprofloxacin, levofloxacin); Antidepressants (Amitriptyline, sertraline); Anti-seizure medications (Carbamazepine, phenytoin)

4.6 Use in special populations (Pregnancy, Nursing woman, Pediatric use, Geriatric use)
Pregnant Women:
There are no adequate and well controlled studies of this combination therapy in pregnant women. Surbex Star [New] soft gelatin capsules should be used during pregnancy only if the potential benefit justifies the possible risk to the fetus.

Lactating Women:
It is not known whether components of Surbex Star [New] soft gelatin capsules are excreted in human milk. Caution should be exercised when this product is administered to a nursing woman. Nursing mothers should not use this preparation unless clearly needed and recommended by physician.

Paediatric Patients:
The safety and efficacy of this formulation has not been established in the paediatric population. Surbex Star [New] soft gelatin capsules are not intended for use in children.

Geriatric Patients:
Generally, dose adjustment is not required in the geriatric population. Elderly patients with normal renal and hepatic function may be given the same dose as recommended for adults.

Renal and Hepatic Impairment:
Patients This formulation has not been studied in patents with hepatic and renal impairment. Caution should be exercised when Surbex Star [New] soft gelatin capsules are administered to these patients and it is recommended to monitor renal and hepatic functions.

4.7 Effects on ability to drive and use machines
Surbex Star [New] Soft Gelatin Capsules are unlikely to cause any impact on the ability to drive and use machines.

4.8 Undesirable effects

Mecobalamin:

Rash, anorexia, nausea, vomiting, diarrhea, headache has been reported with Mecobalamin therapy. Other reported adverse reactions include anaphylactic reaction such as decrease in blood pressure or dyspnea, hot sensation, diaphoresis.

Alpha Lipoic Acid:

Side effects from using alpha lipoic acid appear to be rare and mild, such as nausea or skin rash.

Pyridoxine:

Pyridoxine usually has no side effects when used in recommended doses. Pyridoxine can cause side effects such as headache, nausea, drowsiness, numbness/tingling of arms/legs when taken in large doses and for a longer period of time.

Folic Acid:

Folic acid is generally well tolerated. Gastrointestinal disturbances and allergic reactions have been reported rarely with use of folic acid.

Benfotiamine:

Very rare: allergic reactions (urticaria, rash) can occur in isolated cases. Isolated cases of gastrointestinal disorders (i.e. nausea and other complaints) have been recorded in clinical trials.

4.9 Overdose

Mecobalamin:

Data regarding overdose with Mecobalamin is limited. Mecobalamin has excellent tolerability and no known toxicity. In the event of overdose, treatment should be symptomatic and supportive.

Alpha Lipoic Acid:

No overdose has been reported with alpha lipoic acid. Symptoms of overdose may include hypoglycemia, headache, feeling hungry, weakness, sweating, confusion, irritability, dizziness, tachycardia, or feeling jittery. If overdose occurs, treatment should be symptomatic and supportive.

Pyridoxine:

Pyridoxine can cause neurological disorders, such as loss of sensation in legs and lack of balance/coordination, when taken in high doses (200 mg or more per day) over a long period of time. Pyridoxine/vitamin B6 toxicity can damage sensory nerves, leading to numbness in the hands and feet as well as difficulty in walking. Symptoms of a pyridoxine overdose may include poor coordination, staggering, numbness, decreased sensation to touch, temperature, vibration and tiredness for up to 6 months.

Folic Acid:

Toxicity from excessive folic acid intake does not normally occur as folic acid is water soluble and regularly excreted by the body. High levels of folic acid can provoke seizures in patients taking anticonvulsant medications.

5. Pharmacological properties

5.1 Mechanism of action

Mecobalamin:

Mecobalamin regulates nerve function and reduces plasma homocysteine levels by following mechanisms:

1. Mecobalamin promotes myelination (phospholipid synthesis): Mecobalamin promotes the synthesis of lecithin, the main constituent of medullary sheath lipid and increases myelination of neurons in rat tissue culture more than cobamamide does.
2. Mecobalamin promotes axonal transport and axonal regeneration: Mecobalamin normalizes axonal skeletal protein transport in sciatic nerve cells from rat models with streptozotocin-induced diabetes mellitus. It exhibits neuropathologically and electrophysiologically inhibitory effects on nerve degeneration in neuropathies induced by drugs, such as adriamycin, acrylamide, and vincristine (in rats and rabbits), models of axonal degeneration in mice and neuropathies in rats with spontaneous diabetes mellitus.
3. Mecobalamin is a kind of endogenous coenzyme B12: Mecobalamin plays an important role in transmethylation as a coenzyme of methionine synthetase in the synthesis of methionine from homocysteine.
4. Mecobalamin is well transported to nerve cell organelles, and promotes nucleic acid and protein synthesis: Mecobalamin is better transported to nerve cell organelles than cyanocobalamin in rats. Also, Mecobalamin promotes nucleic acid and protein synthesis in rats more than cobamamide does.

Alpha Lipoic Acid:

As an antioxidant, alpha lipoic acid (ALA) directly terminates free radicals, chelates transition metal ions, increases cytosolic glutathione and vitamin C levels, and prevents toxicities associated with their loss. These diverse actions suggest that ALA acts by multiple mechanisms both physiologically and pharmacologically.

Pyridoxine:

Pyridoxine/vitamin B6 is a water-soluble vitamin required for amino acid, carbohydrate, and fat metabolism. Pyridoxine have role as a coenzyme in a wide variety of enzymes involved in cell growth and cell division. High homocysteine level in the blood (hyperhomocysteinemia) is a risk factor for cardiovascular disease, blood clotting abnormalities, myocardial infarction (heart attack), and ischemic stroke. Pyridoxine alone or in combination with folic acid has been shown to be effective for lowering homocysteine levels.

Folic Acid:

Folic acid is reduced in the body to tetrahydofolate, which is a coenzyme for various metabolic processes including the synthesis of purine and pyrimidine nucleotides, and hence the synthesis of DNA.

Benfotiamine:

Benfotiamine works by increasing the levels of thiamine (vitamin B1) in the body, which plays a crucial role in energy production and nerve function. It helps protect against oxidative stress and inflammation by inhibiting the pathways that lead to their formation.

5.2 Pharmacodynamic Properties

Mecobalamin:

Mecobalamin is the neurologically active form of vitamin B12. Mecobalamin is useful to treat or correct various neurological defects such as neuropathies. In many cases, liver does not convert cyanocobalamin, the commonly available form of vitamin B12, into adequate amounts of Mecobalamin. Nutritional inadequacies, enzyme defects, and pathological changes to tissues can all contribute to a reduced ability of the body to accomplish the synthesis of the active forms of vitamin B12 from cyanocobalamin. Surbex Star ^{New} Soft Gelatin Capsules provide readymade form of vitamin B12 i.e., Mecobalamin to treat various types of neuropathies including diabetic neuropathy.

Alpha Lipoic Acid:

Alpha-lipoic acid is an antioxidant also known as ALA, Acetate Replacing Factor, Biletan, Lipoicin or Thioctic Acid. Alpha lipoic acid is a naturally occurring fatty acid that can be found in many foods. Alpha lipoic acid seems to delay or reverse peripheral diabetic neuropathy through its multiple antioxidant properties. ALA improves glycemic control and polyneuropathies associated with diabetes mellitus. ALA enhances glucose uptake (thus, lowers plasma glucose levels) in type 2 diabetes patients. ALA has been useful to provide relief from symptoms caused by diabetic neuropathy such as the pain, burning, tingling, and numbing. Its biosynthesis decreases as people age and is reduced in people with compromised health, suggesting a supplementation of ALA in such cases.

Pyridoxine:

Pyridoxine is essential for cell growth and cell division. Pyridoxine also involves in carbohydrate, protein, and fat metabolism. Pyridoxine also reduces homocysteine levels in the blood.

Folic Acid:

Folic acid is used in the treatment and prevention of the folate deficiency state. It is also involved in some amino-acid conversions, and in the formation and utilization of formate.

Benfotiamine:

By improving thiamine levels, benfotiamine supports better heart health and reduces the risk of nerve damage associated with thiamine deficiency. It also aids in maintaining normal glucose metabolism and reducing complications related to high blood sugar levels. Additionally, benfotiamine may have antioxidant properties that help protect cells from damage caused by free radicals.

5.3 Pharmacokinetic properties

Mecobalamin:

Absorption: Vitamin B12 binds to intrinsic factor, a glycoprotein secreted by the gastric mucosa, and is then actively absorbed from the gastrointestinal tract. A small amount of vitamin B12 can

also be absorbed from the gastrointestinal tract by passive diffusion. When Mecobalamin was administered orally to healthy adult male volunteers at single doses of 120 mcg and 1500 mcg during fasting, the peak serum total vitamin B12 concentration was reached after 3 hours for both doses, and this was dose dependent.
Distribution: Vitamin B12 is extensively bound to specific plasma proteins called transcobalamins; transcobalamin II appears to be involved in the rapid transport of the cobalamins to tissues. Vitamin B12 diffuses across the placenta and also appears in breast milk.
Metabolism and Excretion: Vitamin B12 is stored in the liver and excreted in the bile. It undergoes extensive enterohepatic recycling; a part of the dose is excreted in the urine, most of it in the first 8 hours. About 40 to 80% of the cumulative amount of total B12 is excreted in the urine within 24 hours. Elimination half-life of Mecobalamin is 12.5 hours after a single-dose oral administration.

Alpha Lipoic Acid:
After oral administration, alpha lipoic acid is readily and nearly completely absorbed with a limited absolute bioavailability of about 30% caused by high hepatic extraction. The primary metabolic pathways of alpha lipoic acid are s-methylation and beta-oxidation. Major circulating metabolites were the s-methylated beta-oxidation products 4, 6-bismethylthio-hexanoic acid and 2,4-bismethylthio-butanoic acid, whereas its conjugated forms accounted for the major portion excreted in urine. Despite the prolonged half-lives of the major metabolites compared to the parent drug, no evidence of accumulation was found. Mean values of 12.4% of the administered dose were recovered in the urine after 24 hours as the sum of alpha lipoic acid and its metabolites.

Pyridoxine:
Absorption: Pyridoxine hydrochloride is absorbed rapidly from the upper intestine regardless of the size of the dose given. Absorption may also occur from ileum and to a small extent from the colon.
Metabolism and Distribution: Pyridoxine is rapidly converted in the liver to pyridoxine phosphate, pyridoxal phosphate and pyridoxamine phosphate via oxidation. This causes the release of pyridoxal and some pyridoxal phosphate to the general circulation where it reaches other organs chiefly as circulating pyridoxal. Pyridoxine and its metabolites are stored mainly in the liver where there is oxidation to 4-pyridoxic acid and other inactive metabolites. Pyridoxal crosses the placenta and is also distributed into breast milk.
Elimination: Pyridoxine is excreted primarily unchanged in the urine; with a small amount in the form of metabolite, most likely 4-pyridoxic acid. As the dose increases, proportionally greater amounts are excreted unchanged in the urine.

Folic Acid:
Absorption: Folic acid is rapidly absorbed from the gastrointestinal tract, mainly from the duodenum and jejunum.
Metabolism: The naturally occurring folate polyglutamates are largely deconjugated, and then reduced by dihydrofolate reductase in the intestines to form 5-methyltetrahydrofolate, which appears in the portal circulation, where it is extensively bound to plasma proteins. Folic acid given therapeutically enters the portal circulation largely unchanged, since it is a poor substrate for reduction by dihydrofolate reductase. Folic acid is converted to the metabolically active form 5-methyltetrahydrofolate in the plasma and liver.

Distribution and Elimination: Folate is distributed into breast milk. The principal storage site of folate is the liver; it is also actively concentrated in the cerebrospinal fluid (CSF). Folate undergoes enterohepatic circulation. Folate metabolites are eliminated in the urine and folate in excess of body requirements is excreted unchanged in the urine. Folic acid is removed by haemodialysis.
Benfotiamine:
After oral ingestion, Benfotiamine is digested in the gastrointestinal tract to a form which is more lipid-soluble than thiamine which permits this Benfotiamine-form to readily cross cell membranes in the intestines and enter the blood stream. A significant percentage of this lipid-soluble form is captured within red blood cells, where it is converted to free Thiamine as needed by the body. The bioavailability of Thiamine in blood plasma is reported to be approximately 3.6 times greater after a dose of Benfotiamine than after a dose of Thiamine.

6. Nonclinical properties

6.1 Animal Toxicology or Pharmacology

Mecobalamin:
Repeated dose toxicity: A total of 48 Sprague-Dawley rats were randomly assigned to receive, intravenously, 1, 5, 25 or 100 mg/kg of cyanocobalamin (6 males and 6 females in each group) three times per week until completion of the study at 182 days (26 weeks). The animals were weekly examined and blood samples were taken at days 1, 85 and 182 for cyanocobalamin determination. Finally, all animals survived throughout the study period and had similar growth rates. No evidence of toxicity was detected by a detailed weekly examination of animals during the study period. Therefore the no-observed-adverse-effect-level (NOAEL) can be established at least at 100 mg/kg under the test conditions.
Carcinogenicity: Vitamin B12 (as cyanocobalamin or hydroxocobalamin) has a long history of safe use even at high doses. A tumour promoting effect of vitamin B12 has been reported in one study in rats. Rats kept on a methionine deficient diet supplemented with 5 µg/100 g vitamin B12 and treated with the carcinogen p-dimethylaminobenzene (DAB) had a higher incidence of hepatomas compared to the group without supplemental vitamin B12. A control group receiving the supplemented diet without DAB showed no hepatic tumours. In another study, the effect of Mecobalamin and cyanocobalamin on the growth of Walker's carcinosarcoma and on the longevity of rats with implanted Zajdela ascites hepatoma cells has been studied. Study reported reduced survival of rats upon treatment with both compounds.

Alpha Lipoic Acid:
In acute toxicity studies, the cat was the most sensitive species, followed by the monkey, dog, mouse and rat. Route of administration impacts toxicity (intravenous was most toxic, followed by intraperitoneal, subcutaneous, and oral). The liver and kidney were targets of toxicity in short term studies in rats and cats. No toxicity was seen following chronic exposure in dogs. No data are available for reproductive/developmental toxicity. ALA is not mutagenic in the Ames and micronucleus genotoxicity assays. Carcinogenicity assessment was negative.
Ant-mutagenic effects: Study investigated both the mutagenicity and anti-mutagenicity of alpha lipoic acid (ALA) in the bone marrow cells of mice using a chromosomal aberration assay. Cyclophosphamide (CP) 40 mg/kg was used as a clastogen in the positive control, and a vehicletreated negative control group was also included. Multiple dose levels (15, 30, and 100 mg/kg of ALA) were given by intraperitoneal injection (IP) alone and in combination with CP (CP

was administered 1 h prior to ALA). Bone marrow samples were collected 12 and 24 h after drug administration. The results demonstrated a significant increase in the frequency of chromosomal aberrations (CA) in bone marrow cells with depressions in the mitotic index (MI) of the positive control group of mice. However, in the groups of mice treated with different doses of ALA in the presence of CP, the percentages of CA decreased significantly with increases in mitotic activity. The results also indicate that ALA given alone in different doses had no mutagenic effect on mouse bone marrow cells. ALA has a dose and time-dependent protective effect against the mutagenicity induced by CP.

Pyridoxine:

Acute oral toxicity: The acute oral toxicity of ten batches of pyridoxine hydrochloride was tested in mice. After 10 days observation period, the LD50 was found to be 6994 mg/kg body weight. Repeated dose toxicity - oral: Rats and mice were orally exposed to pyridoxine hydrochloride for 10 days. Only at the lowest test doses (2000 mg/kg/day (mice) and 500 mg/kg/day (rats)) no mortality was seen. Rats and mice dosed at the highest dose (16000 mg/ kg/day) died on the first day. At 8000 mg/kg/day, exposed mice and rats died after second dosing. Pyridoxine influenced body weight gain of mice and rats in a dose-dependent way.

Mutagenicity: An Ames test was performed with pyridoxine. All bacterial strains showed negative responses up to 5000 ug/plate, i.e. no significant dose-related increase in the number of revertants with or without metabolic activation was seen. No cytotoxicity and/or precipitation of the test substance was observed. The negative and strain-specific positive control values were within the laboratory historical control data ranges indicating that the test conditions were adequate and that the metabolic activation system functioned properly. Based on the results of this study it is concluded that pyridoxine is not mutagenic in the *Salmonella typhimurium* reverse mutation assay and in the *Escherichia coli* reverse mutation assay with or without metabolic activation.

Folic Acid:

Animal studies have shown that folic acid can be a neurotoxin, and can cause convulsions in laboratory animals. This evidence is in part based upon in vitro tissue and cell culture studies, and/or using very high dose levels (i.v. dosages 60-90 mg). There is however no clear evidence for a folic acid-induced neurotoxicity in humans. Toxicity in different strains of mice showed that toxic doses of folic acid may lead to convulsions, ataxia and weakness. Histopathological studies in some strains of mice showed acute renal tubular necrosis.

In another study with experimentally (diet) induced vitamin B12 deficiency in rhesus monkeys, three of the nine monkeys received 5 mg/week of supplemental folic acid intramuscularly, followed by 5 mg in the drinking water (5 days/week). Five animals developed visual impairment and optic atrophy, including the 3 monkeys that received supplemental folic acid. Apparently, the optical nerve lesions occurred earlier (by 10-11months) in the folic acid-treated animals. It should be noted that the visual lesions observed in these monkeys are only rarely noted in human disease. Spastic paralysis of hind legs and tail was found in 3 animals, including 2 animals receiving folic acid. Other lesions in cranial and peripheral nerves and in the white matter of the spinal cord were observed in some animals but were apparently not affected by supplemental folic acid.

7. Description

Surbex Star [New] is supplied for oral administration, as soft gelatin capsules of Mecobalamin, Alpha Lipoic Acid, Inositol, Folic Acid, Chromium Nicotinate, Selenious Acid I.P. & Benfotiamine. Each soft gelatin capsule of Surbex Star [New] contains Mecobalamin 1500 mcg, Alpha Lipoic Acid 100 mg, Inositol 100 mg, Folic Acid 1.5 mg, Chromium Nicotinate 200 mcg, Selenious Acid & Benfotiamine 150 mg.

8. Pharmaceutical particulars

8.1 Incompatibilities

NA

8.2 Shelf Life

Refer Pack

8.3 Packaging Information

Refer Pack

8.4 Storage condition & handling instructions

Refer Pack

9. Patient Counseling Information

Instructions to Patients:

- Instruct patients not to change their medication dose or schedule without consulting doctor. Do not exceed the recommended dose or duration of treatment.
- Pregnant women and nursing mothers can use this medicine only in consultation with their doctor. This medicine is not recommended for use in children.
- Instruct patients not to share their medication with others even though it has been prescribed for same disease/condition. Also, not to use medication prescribed for others.
- Surbex Star [New] Soft Gelatin Capsules should not be exposed to light/moisture because Mecobalamin is susceptible to degradation by photolysis.

10. Details of manufacturer

Refer Pack for manufacturer details.

Marketed by:
Abbott Healthcare Pvt. Ltd.
Angel Space, Bldg. D-4, Gala No. 1 to 3 &
11 to 13 Ground Floor, 101 to 106 &
111 to 116 First Floor, 201 to 206 & 211 to 216
2nd Floor, Pimplas, Dist. Thane, Bhiwandi - 421 302,
Maharashtra, India.

11. Details of permission or licence number

Refer Pack for Permission/License details

Version 2.0, dated 04-May-26
Replaces Version 1.0, dated 10-Apr-24

12. Date of revision
Version 2.0, dated 04-May-26

For Product Complaints/Adverse events or Queries please write to
webmasterindia@abbott.com