

FINASTERIDE SERVYCAL

FINASTERIDE 5 mg

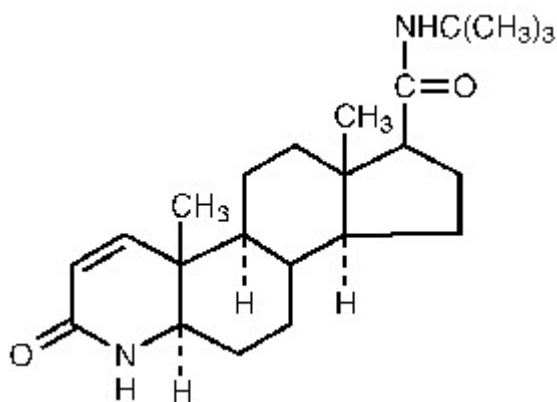
(Coated Tablets)

SALE UNDER PRESCRIPTION

MADE IN ARGENTINA

DRUG DESCRIPTION: Finasteride is a synthetic 4-azasteroid compound, and it is a specific inhibitor of steroid Type II 5 α -reductase, an intracellular enzyme that converts the androgen testosterone into 5 α -dihydrotestosterone (DHT).

Finasteride is 4-azadrost-1-ene-17-carboxamide, N-(1,1-dimethylethyl)-3oxo-, (5 α , 17 β). The empirical formula of finasteride is C₂₃ H₃₆ N₂ O₂, and its molecular weight is 372.55. Its structural formula is:



Finasteride is a white crystalline powder with a melting point near 250° C. This compound is freely soluble in chloroform and sparingly soluble in alcohol, but it is practically insoluble in water.

QUALI-QUANTITATIVE COMPOSITION:

Each coated tablet of Finasteride Servycal contains:

Finasteride	5.00 mg
Lactose	105.00 mg
Pregelatinized starch	15.00 mg
Sodium starch glycollate	7.00 mg
Docusate sodium	0.50 mg
Microcrystalline cellulose pH 200	15.00 mg
Povidone K-30	6.00 mg
Magnesium stearate	1.25 mg

Coating

Hydroxypropyl methylcellulose	7.20 mg
Titanium dioxide	1.70 mg
Brilliant blue aluminum lake	0.05 mg
Polyethylene glycol 6000	1.25 mg

DRUG CATEGORY AND ACTION: Finasteride is an inhibitor of the 5 α -reductase enzyme, which metabolizes testosterone to dihydrotestosterone (DHT) in the prostate gland. DHT accounts for the progressive enlargement of the prostate gland: benign prostatic hypertrophy (BPH), a pathology that increases with age.

This medicine decreases DHT levels; as a result the size of the prostate gland is decreased and obstructive and subjective symptoms improve.

This leads to the considerable improvement of bladder emptying, increase in urinary flow and strength of urinary stream, without affecting the plasma levels of testosterone and the role that said gland plays in male body (i.e. fertility, muscular strength, libido, etc.).

INDICATIONS: finasteride is indicated for the treatment of symptomatic benign prostatic hyperplasia (BPH).

This medicine has shown to:

- Improve symptoms.
- Reduce the risk of acute urinary retention.
- Reduce the risk of the need for surgery, including transurethral resection of the prostate and prostatectomy.

Before initiating finasteride therapy, patients should be tested in order to differentiate pathologies such as prostatic carcinoma, infections of the urinary tract, hypotonic bladder, and other neurogenic disorders simulating a BPH.

Finasteride administered in combination with the alpha-blocker doxazosin is indicated to reduce the risk of symptomatic progression of BPH.

CLINICAL PHARMACOLOGY:

The development and enlargement of the prostate gland is dependent on a potent androgen, the 5 α -dihydrotestosterone (DHT). Type II 5 α -reductase metabolizes testosterone to DHT in the prostate gland, liver and skin. DHT induces androgenic effects by binding to androgen receptors in the cell nuclei of these organs.

Finasteride is a competitive and specific inhibitor of Type II 5 α -reductase with which this drug slowly forms a stable enzyme complex. Turnover from this complex is extremely slow ($t_{1/2} \sim 30$ days). This has been demonstrated both in vivo and in vitro. Finasteride has no affinity for the androgen receptor. In men, steroidal metabolites of 5 α -reductase in blood and urine are decreased after finasteride administration.

In men, the administration of a single oral dose of finasteride produces a rapid reduction in serum DHT concentration, with the maximum effect observed after 8 hours after the first dose. The suppression of DHT is maintained throughout the 24-hour dosage interval when treatment is continued. Daily doses of finasteride at 5 mg/day for 4 years or more have shown to reduce the serum DHT concentration levels by about 70%. The mean circulating level of testosterone was increased by about 10-20% and remained within the physiological range.

Type II 5 α -reductase deficiency also decreases levels of DHT. Except for the associated urogenital defects at birth, no other clinical abnormalities related to Type II 5 α -reductase deficiency have been observed in this type of patients. These patients have a small prostate gland and do not develop BPH throughout their lives.

In patients with BPH treated with finasteride (1-100 mg/day) for 7-10 days prior to prostatectomy, an 80% lower DHT content was measured in the prostate tissue removed at

surgery, compared to placebo group in which testosterone tissue concentration was increased more than 10 times over pretreatment levels relative to placebo. Intraprostatic content of PSA (prostate specific antigen) was also decreased.

In healthy male volunteers treated with finasteride for 14 days, discontinuation of therapy lead to a return of DHT to pretreatment levels in approximately 2 weeks. In patients treated for 3 months, prostate volume, which decreased by about 20%, returned to baseline values after 3 months of therapy discontinuation.

Pharmacokinetics:

Absorption: In a study conducted in 15 healthy young volunteers, the mean bioavailability of 5-mg finasteride tablets was 63% (range 34-108%), based on the ratio of area under the curve (AUC) in connection with an intravenous (IV) reference dose. Average peak plasma concentration of finasteride was reached 1-2 hours after dosing. Bioavailability of finasteride was not affected by food.

Distribution: Mean steady-state volume of distribution was 76 L (range 44 - 96 L). About 90% of circulating finasteride is bound to plasma proteins. After multiple dosing, finasteride has a slow accumulation phase. After 5 mg/day of finasteride for 17 days, plasma concentrations were 47% and 54% higher than after the first dose in men of 45-60 years of age and ≥ 70 years of age, respectively. Mean concentrations after 17 days of dosing were 6.2 ng/mL (range 2.4 - 9.8 ng/mL) and 8.1 ng/mL (range 1.8 – 19.7 ng/mL), respectively, in both age groups.

Although steady state was not reached in this study, mean plasma concentrations in another study in patients with BPH (average age, 65 years) who received 5 mg/day were 9.4 ng/mL (range 7.1 – 13.3 ng/mL) after one year or more of dosing.

Finasteride crossed the blood brain barrier but does not seem to distribute preferentially to the CSF.

Two clinical studies conducted in healthy volunteers given 5 mg/day of finasteride for 6-24 weeks showed concentrations of this drug in semen that ranged from undetectable (<0.1 mg/mL) to 10.54 ng/mL. In a study using less sensitive assays, finasteride concentrations in 16 subjects receiving finasteride at 5 mg/day ranged from undetectable (<1.0 ng/mL) to 21 ng/mL. Based on an ejaculate volume of 5 mL, the amount of finasteride in semen was 50-100 fold less than the dose of finasteride (5 μ g) that had no effect on the circulating DHT levels in men.

Metabolism: Finasteride is extensively metabolized in the liver, primarily via the cytochrome P450 3 A4 enzyme subfamily. Two metabolites, the t-butyl side chain monohydroxylated and monocarboxylic acid metabolites, have been identified and they have more than 20% of the 5 α -reductase inhibitory activity of finasteride.

Excretion: In healthy young volunteers, mean plasma clearance of finasteride was 165 mL/min (range 70 – 279 mL/min); mean elimination half-life in plasma was 6 hs (range 3 – 16 hours). After an oral dose of ¹⁴C-finasteride in men, a mean of 39% (range 51 – 64%) of the dose was excreted in the urine in the form of metabolites and 57% (range 51 – 54%) was excreted in the feces.

The mean terminal half-life of finasteride in subjects ≥ 70 years old was approximately 8 hs (range 6 -15 hours), compared to 6 hours (range 4 – 12 hours) in subjects 45-60 years old. As a result, the mean AUC (0-24 hs) after 17 days of dosing was 15% higher in subjects ≥ 70 of age than in subjects 45-60 years of age ($p=0.02$).

Special Populations:

Pediatrics: The pharmacokinetic of finasteride has not been investigated in patients under 18 of age.

Gender: The pharmacokinetic of finasteride in women is not available.

Geriatrics: No dose adjustment is necessary in elderly patients. Although the elimination rate of finasteride is decreased in these patients, these findings have not been clinically significant.

Race: The pharmacokinetics of finasteride in the different races have not been studied.

Renal impairment: No dosage adjustment is required in patients with renal impairment. In patients with chronic renal impairment, with creatinine clearance values ranging from 9.0 to 55 mL/min, AUC, peak plasma concentration, half-life, and protein binding after a single dose of 14 C-finasteride were similar to those obtained in healthy volunteers. Urinary excretion of metabolites is decreased in patients with renal impairment. This decrease was associated with an increase in fecal excretion of metabolites.

However, finasteride was well tolerated in patients with normal renal function who received doses higher than 80 mg/day for 12 weeks, in which case exposure of these patients to the drug metabolites would presumably be much greater.

Hepatic impairment: The effect of hepatic impairment on the pharmacokinetics of finasteride has not been studied. Therefore, caution should be exercised when this drug is administered to patients with liver function abnormalities or disorders because finasteride is extensively metabolized in the liver.

Drug-drug interactions: No drug interactions of clinical significance have been determined. Finasteride does not appear to affect the metabolism enzyme system of cytochrome P450-linked drug. Compounds tested in men included antipyrine, digoxin, propranolol, theophylline, and warfarin, and no clinically significant drug interaction was found.

DOSAGE AND ADMINISTRATION: The recommended dose of finasteride is 5 mg once daily. Finasteride may be administered alone or in combination with the alpha-blocker doxazosin. This medicine may be taken with or without food.

No dose adjustment is required for elderly patients or for those with renal insufficiency.

CONTRAINDICATIONS: Finasteride is contraindicated in patients with hypersensitivity to any of its components.

This medicine is also contraindicated in patients who are or may potentially be pregnant. Because of the ability of Type II 5 α -reductase to inhibit the conversion of testosterone into

DHR, finasteride may cause abnormalities in the external genitals of male fetuses of pregnant women receiving finasteride. If this medicine is used during pregnancy or if the patient becomes pregnant while taking it, she should be advised about the potential hazard to the male fetus. In female rats, low doses of finasteride given during pregnancy resulted in abnormalities of the external genitals of male offspring.

WARNINGS: Finasteride is not indicated for use in pediatric patients or pregnant women since their exposure to this drug may harm male fetuses.

Women who are pregnant or may potentially be pregnant should not handle crushed or broken finasteride tablets due to the possibility of absorption and subsequent potential hazard to the male fetus.

PRECAUTIONS:

General: Before initiating finasteride therapy, an appropriate evaluation should be performed in order to identify other conditions such as infection, prostate cancer, hypotonic bladder, stricture disease, or other neurogenic disorders that may mimic BPH.

Patients with large residuary urinary volume and/or severely diminished urinary flow should be carefully monitored in order to early detect obstructive uropathy. These patients may not be eligible for finasteride therapy.

The administration of finasteride to patients with hepatic function abnormalities should be carried out cautiously since finasteride is extensively metabolized in the liver.

Effects on PSA and Prostate Cancer Detection: No clinical benefit has been demonstrated in patients with prostate cancer treated with finasteride. Patients with BPH and elevated PSA values were monitored in controlled clinical studies with serial PSA determinations and prostate biopsies. In these BPH studies, finasteride does not appear to alter the detection rate of prostate cancer, and the overall incidence of prostate cancer was not significantly different in patients treated with finasteride vs those treated with placebo.

Finasteride causes a decrease in serum PSA levels by about 50% in patients with BPH in the presence of prostate cancer. This decrease is predictable over the total range of PSA values, although they may vary in individual patients. Analysis of PSA data from 3000 patients treated with finasteride for 6 months or more, PSA values were doubled if compared to normal ranges of untreated men. These adjustments preserve the sensitivity and specificity of PSA assay and maintain its ability to detect prostate cancer.

Any sustained increases in PSA levels while patient is being treated with finasteride should be carefully evaluated, including non-compliance with finasteride therapy.

Percent free (free to total PSA ratio) may not be significantly decreased by finasteride. The ratio of free to total PSA remains constant even under the influence of finasteride. If physicians choose to use the ratio of free to total PSA as an aid in the detection of prostate cancer in men being treated with finasteride, no adjustment to these values seems necessary.

Patient information: Women who are pregnant or may potentially be pregnant should not handle crushed or broken finasteride tablets due to the possibility of absorption and subsequent potential hazard to the male fetus.

Physicians should inform patients the ejaculate volume may be decreased in some patients during treatment with finasteride. This decrease does not seem to interfere with normal sexual function.

Physicians should also instruct their patients to promptly report if any change in breasts appear, such as nodes, pain, nipple discharge. Breast changes such as breast enlargement, tenderness and neoplasm have also been reported.

Physicians should instruct their patients to read the patient package insert before initiating therapy with finasteride and to reread it every time prescription is renewed so as to be aware of information for patients concerning finasteride.

Drug/Laboratory tests interactions: In patients with BPH, finasteride had no effect on the circulating levels of prolactin, cortisol, estradiol, thyroid-stimulating hormone or thyroxine. No significant effect was observed on the plasma lipid profile (total cholesterol, low and high density lipoproteins and triglycerides), or on bone mineral density. An increase of about 10% was observed in luteinizing hormone (LH) and follicle-stimulating hormone values of patients receiving finasteride, but they remain within the normal range.

In healthy volunteers, treatment with finasteride does not alter the response of FSH and LH to the gonadotropin-releasing hormone, thus the hypothalamic-pituitary-testicular axis is not affected.

In treatment with finasteride for 24 months semen parameters in healthy male volunteers were evaluated, and no clinically significant effect on sperm concentration, morphology and pH was observed. Mean ejaculate volume decreased by 0.6 mL (22.1%) in treated patients, with concomitant reduction of total sperm per ejaculate.

These parameters remained within normal ranges and were reversible after therapy discontinuation, with an average time of 84 weeks to return to baseline values.

Drug-drug interactions:

No significant drug interactions have been observed. Finasteride does not appear to affect the metabolism enzyme system of cytochrome P450-linked drugs. Compounds tested in men included antipyrine, digoxin, propranolol, theophylline, and warfarin, and no clinically significant drug interaction was found.

Other concomitant therapies: Although no specific interaction studies have been conducted, finasteride was used concomitantly in clinical studies with acetylsalicylic acid, acetaminophen, α -blockers, inhibitors of the angiotensin-converting enzyme (ACE), analgesics, anticonvulsants, β -blockers, β -adrenergic blocking agents, diuretics, calcium channel blockers, nitrites, inhibitors of HMG-CoA reductase, non-steroidal anti-inflammatory drugs, benzodiazepines, H₂ antagonists, and quinolone antibiotics without any evidence of clinically significant adverse reactions.

Carcinogenesis, Mutagenesis and Impairment of Fertility: No evidence of a tumorigenic effect was observed in a 24-month study in Sprague-Dawley rats which were given doses of finasteride higher than 160 mg/kg/day in males and 320 mg/kg/day in females. These doses produced a systemic exposure in rats that was 111 to 274 times those given to humans based on the recommended human dose of 5 mg/day. All exposure calculation were based on a calculated AUC (0-24 hs) for animals and mean AUC (0-24 hs) for men (0.4 μ h/mL).

In a 19-month carcinogenicity study conducted in CD1 mice, a statistically significant ($p < 0.05$) increase in the development of testicular Leydig cell adenoma was observed when mice received 250 mg/kg/day (approximately 228 times the human exposure to the drug). In mice receiving 25 mg/kg/day (23 times the human exposure) and rats receiving ≥ 40

mg/kg/day (39 times the human exposure), an increase in the incidence of Leydig cell hyperplasia was observed. A positive correlation between the proliferative changes in Leydig cells and an increase in serum LH levels (2 to 3 times above control) has been demonstrated in both rodent species treated with high doses of finasteride. No drug-related changes in Leydig cells were observed in rats or dogs treated with 20 mg/kg/day and 45 mg/kg/day of finasteride (30 to 350 times the human exposure) for 1 year, or in mice treated with 2.5 mg/kg/day (2.3 times the estimated human exposure) for 19 months.

No evidence of mutagenicity was observed in an in vitro bacterial assay, a mammalian cell mutagenicity assay, or an in vitro alkaline elution assay. In an in vitro chromosome aberration assay using ovarian cells of Chinese hamster, a slight increase in chromosome aberrations was seen. These concentrations correspond to 4,000 to 5,000 times the peak plasma levels in men administered a total dose of 5 mg.

In an in vivo chromosome aberration assay in mice, no treatment-related increases in chromosome aberrations were observed at the maximum tolerated dose of finasteride of 250 mg/kg/day (228 times the human exposure) as determined in carcinogenicity studies.

In sexually mature rabbits treated with finasteride at 80 mg/kg/day (543 times the human exposure) for more than 12 weeks, no effect was observed on fertility, sperm count, or ejaculate volume. In sexually mature rats treated with finasteride at 80 mg/kg/day (61 times the human exposure) no significant effect on fertility was seen after 6 to 12 weeks of treatment; however when treatment was continued for more than 24 to 30 weeks, there was an apparent decrease in fertility, fecundity, and an associated significant decrease in the weight of seminal vesicles and prostate. All these effects were reversible within 6 weeks of treatment discontinuation. No drug-related adverse effects were seen on testes or mating performance in rats or rabbits. The decrease in fertility in rats treated with finasteride is secondary to its effect on accessory sex organs (prostate and seminal vesicles), resulting in failure to form seminal plug. This anatomic structure is essential for fertility in rats but is irrelevant in men.

Pregnancy: Pregnancy Category X.

The use of finasteride is contraindicated in women.

Administration of finasteride to pregnant rats at doses ranging from 100µg/kg/day to 100 mg/kg/day (1-1000 times the recommended human dose of 5 mg/day) resulted in dose-dependent development of hypospadias in 3.6 to 100% of male offspring. Pregnant rats produced male offspring with a decrease in prostatic and seminal vesicle weights, delayed preputial separation and transient nipple development when finasteride was administered at 30µg/kg/day ($\geq 3/10$ the recommended human dose of 5 mg/day), and decreased anogenital distance when administered at 3µg/kg/day ($\geq 3/100$ the recommended human dose of 5 mg/day). The critical period during which these effects may be induced in male rats has been defined to be between days 16-17 of the gestation period. The above described changes are expected pharmacological effects from drugs belonging to the class of Type II 5 α -reductase and are similar to those reported in male infants with a genetic deficiency of 5 α -reductase. No abnormalities were observed in female offspring exposed to any dose of finasteride in the uterus.

No abnormalities have been observed in the development of the first filial generation (F1) of male and female offspring resulting from mating male rats treated with finasteride (80 mg/kg/day, 61 times the human exposure) with untreated females.

Administration of 3 mg/kg/day of finasteride (30 times the recommended human dose of 5 mg/day) during the late gestation and lactancy period resulted in a slight decrease in fertility in F1 male offspring. This effect was not observed in female offspring. No evidence of malformations has been observed in rabbit fetuses exposed to finasteride from day 6 to 18 of the gestation period at doses higher than 100 mg/kg/day (1000 times the recommended human dose of 5 mg/kg/day). However, effects on male genitals would not be expected since rabbits were not exposed during the critical period of their genital system development.

The in utero effects of finasteride exposure during the period of embryonic and fetal development were studied and evaluated in Rhesus monkeys (days 20-100 of the gestation period), one of the most predictive species of human development, more than rats and rabbits. Intravenous administration of finasteride in pregnant monkeys at high daily doses of 800 ng/day (at least 60 to 120 times the highest estimated exposure of pregnant women to semen of men taking finasteride at 5 mg/day) resulted in no abnormalities in the development of male fetuses. In confirmation of the relevance of the rhesus monkeys model for human fetal development, oral administration of finasteride (2 mg/kg/day, 20 times the recommended human dose of 5 mg/day or approximately 1-2 million times the highest estimated exposure of pregnant women to semen of men taking finasteride at 5 mg/day) to pregnant monkeys resulted in male fetuses with abnormalities in external genitals. No other abnormalities were observed in male fetuses and no finasteride-related abnormalities were seen in female fetuses with at any other dose.

Breastfeeding mothers: Finasteride is not indicated to be used in women. It is unknown whether this drug is excreted in and present in human milk.

Pediatric use: This medicine is not indicated to be used in pediatric patients because its effectiveness and safety in this type of patients have not been established.

Geriatric use: According to a clinical study conducted in patients whose ages ranged from 65 to 75 years old or more, no overall differences in safety and effectiveness were observed in this type of patients and in younger patients; other reports from clinical studies did not identify differences in responses between elderly patients and younger patients. Thus, no dosage adjustments are required in the elderly.

ADVERSE REACTIONS: According to clinical studies with finasteride, the following adverse reactions have been observed.

Finasteride Year 1	%	Finasteride Years 2, 3 and 4	%
Impotence	8.1	Impotence	5.1
Decreased libido	6.4	Decreased libido	2.6
Decreased ejaculate volume	3.7	Decreased ejaculate volume	1.5
Ejaculation disorders	0.8	Ejaculation disorders	0.2
Breasts	0.5	Breasts	1.8
Breast enlargement	0.5	Breast enlargement	1.8
Breast tenderness	0.4	Breast tenderness	0.7
Rash	0.5	Rash	0.5

In a clinical study combining finasteride + doxazosin, the following adverse reactions were observed:

Adverse Reaction	Doxazosin %	Finasteride %	Combination %
Body			
Asthenia	15.7	5.3	16.8
Headache	4.1	2.0	2.3
Cardiovascular			
Hypotension	3.4	1.2	1.5
Postural hypotension	16.7	9.1	17.8
Metabolic and nutritional			
Peripheral edema	2.6	1.3	1.3
Nervous			
Dizziness	17.7	7.4	23.2
Decreased libido	7.0	10.0	11.6
Somnolence	3.7	1.7	3.1
Respiratory			
Dyspnea	2.1	0.7	1.9
Rhinitis	1.3	1.0	2.4
Urogenital			
Abnormal ejaculation	4.5	7.2	14.1
Gynecomastia	1.1	2.2	1.5
Impotence	14.4	18.5	22.6
Abnormal sexual function	2.0	2.5	3.1

Post-Marketing Experience: The following adverse reactions have been reported:

- Hypersensitivity reactions, including pruritus, urticaria, and swelling of the face and lips.
- Testicular pain.

OVERDOSAGE: Patients have received single doses of finasteride higher than 400 mg and multiple doses higher than 80 mg/day for 3 months without adverse events. Until further information is obtained, no specific treatment for overdose can be recommended. In male and female mice, significant lethality was observed at single oral doses of 1500 mg/m² (500 mg/kg/day); the same effect was seen in male and female rats at single oral doses of 2360 mg/m² (400 mg/kg) and 5900 mg/m² (1000 mg/kg), respectively.

In the case of acute poisoning, go to the nearest Hospital or contact Toxicology Centers:

Hospital de Niños Dr. Ricardo Gutiérrez: Ph: + 54 (11) 4962-6666 / 2247

Hospital Dr. Juan P. Garrahan: Ph: + 54 (11) 4941-6191 / 6012

Hospital Dr. Juan A. Fernández: Ph: + 54 (11) 4801-5555

Hospital A. Posadas: Ph: + 54 (11) 4654-6648 / 4658-7777

PRESENTATION: Finasteride SERVYCAL is supplied in cartons containing 30, 60 and 90 coated tablets.

STORAGE: Store at room temperature below 30° C, protected from light.

KEEP OUT OF THE REACH OF CHILDREN

PATIENT INFORMATION

If you are taking Finasteride Servycal, read this insert carefully. You should also read it each time prescription is renewed and if your dosage is changed.

Always remember that this insert does not replace explanations and/or discussions about this medicine with your doctor.

Besides, you and your doctor should discuss carefully when to start taking this medicine and the regular checkups that are required during treatment.

1) Why are you treated with Finasteride Servycal?

Your doctor has prescribed this medicine because you have a prostatic pathology called Benign Prostatic Hyperplasia (BPH).

What you need to know while taking Finasteride Servycal

- 1) **You must see your doctor regularly:** While you are taking this medicine, you must have regular checkups. Your doctor will determine when to make them.
- 2) **Adverse effects:** All prescription drugs may cause adverse effects. The adverse effects that may be caused by finasteride include:
Impotence (inability to have an erection) or less sexual desire: some men treated with this drug may have changes or problems with ejaculation, such as a decrease in the amount of semen released during sexual intercourse. This decrease does not seem to interfere with normal sexual function. In some cases these effects may continue while you are taking this medicine. Besides, some men may have breast enlargement and/or tenderness. You should promptly tell your doctor if you experience any of these symptoms, and also if you notice any changes in your breasts such as nodes or nipple discharge. Some men have also reported allergic reactions like rash, itching, hives, and swelling of the lips and face. Testicular pain has been rarely reported.
You should discuss adverse effects with your doctor before starting to take finasteride or whenever you think you are having an adverse effect.
- 3) **Checking for prostate cancer:** Your doctor has prescribed finasteride for symptomatic BPH and not for prostate cancer. But a man may have at the same time BPH and prostate cancer. Doctors usually recommend one yearly prostate cancer check when men come to 50, or to 40 if anyone in family has had prostate cancer. These checks should continue while you are taking finasteride since it is not a treatment for prostate cancer.
- 4) **PSA (prostate specific antigen):** Your doctor may tell you to have a blood test done called PSA. Finasteride may alter PSA values. For more information, consult your doctor.

- 5) **Warning about finasteride and pregnancy:** Finasteride must be used by men only. Women who are or may potentially be pregnant should not handle crushed or broken tablets of finasteride. If a pregnant woman with a male baby absorbs the active ingredient of this medicine, whether orally or through the skin, the baby may be born with alterations or abnormalities in the sex organs.

Finasteride is supplied in coated tablets to avoid or prevent contact with the active ingredient when they are handled. Crushing or breaking of tablets should be avoided.

If a pregnant woman comes into contact with the active ingredient of Finasteride Servycal, a doctor should be consulted.

Remember that these warnings only apply to women who are pregnant or may potentially become pregnant.

How should I take Finasteride Servycal?

You must carefully follow your doctor's directions on how to take this medicine. You must take it every day. You may take it with or between meals. To avoid forgetting to take finasteride, it may be helpful to take it each day at the same time.

Your doctor may prescribe this medicine along with other medicines such as an α -blocker called doxazosin, which may help you better manage BPH.

Do not share this medicine with anyone else because it has been prescribed only for you.

Keep this medicine out of the reach of children.

Medicinal Specialty Authorized by the Argentine Ministry of Health (A.N.M.A.T.)

Certificate No. 44.447

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