PRESCRIBING INFORMATION

PrAPO-DEXAMETHASONE

Dexamethasone Tablets USP

0.5 mg and 4 mg

Corticosteroid

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Control #: ***237881***

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PrAPO-DEXAMETHASONE

Dexamethasone Tablets USP

0.5 mg and 4 mg

THERAPEUTIC CLASSIFICATION

Corticosteroid

ACTIONS AND CLINICAL PHARMACOLOGY

Comparative Bioavailability

A comparative bioavailability study was performed using healthy adult volunteers. The rate and extent of absorption of dexamethasone were measured and compared following administration of a 4 mg dose of either Apo-Dexamethasone 4 mg tablets or Decadron® 4 mg tablets under fasting conditions. The results from measured data are summarized as follows:

Summary Table of the Comparative Bioavailability Data
Dexamethasone (Dose: 1 x 4 mg) From Measured Data – Under Fasting Conditions

Parameter	Geometric Mean Arithmetic Mean (CV%)		Ratio of Geometric Means (%)**
	Apo-Dexamethasone	Decadron®†	
AUC_T (ng•hr/mL)	166 174 (32)	175 182 (31)	94.9
AUC_I (ng•hr/mL)	169 178 (32)	178 186 (33)	94.9
C_{max} (ng/mL)	27.2 27.9 (22)	29.7 30.4 (23)	91.4
T_{max} (hr)*	1.56 (41)	1.46 (38)	-
$t_{1/2}$ (hr)*	4.03 (17)	4.09 (15)	-

* Arithmetic means (CV%).
** Based on the least squares estimate.
† Decadron® is manufactured by Merck Frosst Canada & Co., and was purchased in Canada.

INDICATIONS AND CLINICAL USE

Allergic states: Control of severe or incapacitating allergic conditions not responsive to adequate trials of conventional treatment: seasonal or perennial allergic rhinitis, bronchial asthma, contact dermatitis, atopic dermatitis, serum sickness, drug hypersensitivity reactions.

Rheumatic disorders and collagen diseases: As adjunctive therapy for short-term administration (for acute episode or exacerbation) in: psoriatic and rheumatoid arthritis including juvenile rheumatoid arthritis (selected cases may require low dose maintenance therapy), ankylosing spondylitis, acute and subacute bursitis, acute non-specific tenosynovitis, acute gouty arthritis, post-traumatic osteoarthritis, synovitis of osteoarthritis, epicondylitis; for exacerbation or maintenance therapy in selected cases of systemic lupus erythematosus, acute rheumatic carditis, systemic dermatomyositis, polymyositis, polymyalgia rheumatica, giant cell arteritis.

Dermatologic diseases: Pemphigus, bullous dermatitis herpetiformis, severe erythema multiforme (Stevens-Johnson syndrome), exfoliative dermatitis, mycosis fungoids, severe psoriasis, severe seborrheic dermatitis.

Ophthalmic diseases: Severe acute and chronic allergic and inflammatory processes involving the eye and its adnexa such as allergic conjunctivitis, keratitis, allergic corneal marginal ulcers, herpes zoster ophthalmicus, iritis and iridocyclitis, chorioretinitis, anterior segment inflammation, diffuse posterior uveitis and choroiditis, optic neuritis, sympathetic ophthalmia.

Endocrine disorders: Primary or secondary adrenocortical insufficiency (hydrocortisone or cortisone is the first choice; synthetic analogs may be used in conjunction with mineralocorticoids where applicable; in infancy, mineralocorticoid supplementation is of particular importance); congenital adrenal hyperplasia; nonsuppurative thyroiditis; hypercalcemia associated with cancer.

Respiratory diseases: Symptomatic sarcoidosis, Loeffler's syndrome not manageable by other means, berylliosis, fulminating or disseminated pulmonary tuberculosis when concurrently accompanied by appropriate antituberculous chemotherapy, aspiration pneumonitis, pulmonary emphysema where bronchospasm or bronchial edema plays a significant role, diffuse interstitial pulmonary fibrosis (Hamman-Rich syndrome).

Hematologic disorders: Idiopathic thrombocytopenic purpura in adults, secondary thrombocytopenia in adults, acquired (autoimmune) hemolytic anemia, erythroblastopenia (RBC anemia), congenital (erythroid) hypoplastic anemia.

Neoplastic diseases: For palliative management of: leukemias and lymphomas in adults, acute leukemia of childhood.

Edematous states: To induce a diuresis or remission of proteinuria in the nephrotic syndrome without uremia, of the idiopathic type or that due to lupus erythematosus.

Nervous system: Apo-Dexamethasone (dexamethasone) may be used to treat patients with cerebral edema associated with primary or metastatic brain tumors, neurosurgery, head injury, pseudotumor cerebri and cerebral vascular accident (acute stroke) excluding intracerebral hemorrhage. Apo-Dexamethasone may also be used in the preoperative preparation of patients with increased intracranial pressure secondary to brain tumors or for palliation of patients with inoperable or recurrent brain neoplasms.

Gastrointestinal diseases: As adjunctive therapy in the treatment of ulcerative colitis and regional enteritis.

Miscellaneous: Tuberculous meningitis with subarachnoid block or impending block when concurrently accompanied by appropriate antituberculous chemotherapy; trichinosis with neurologic or myocardial involvement; postoperative dental inflammatory reactions. During an exacerbation or as maintenance therapy in selected cases of systemic lupus erythematosus, acute rheumatic carditis. In combination with ondansetron for the management of nausea and vomiting associated with cisplatin and non-cisplatin emetogenic chemotherapy.

Apo-Dexamethasone is also used in the diagnostic testing of adrenocortical hyperfunction and antenatal prophylaxis of neonatal respiratory distress.

CONTRAINDICATIONS

Hypersensitivity to the product and its components; systemic fungal infections; administration of live virus vaccines in patients receiving immunosuppressive corticosteroid doses (see **WARNINGS**).

WARNINGS

Infections: In patients on corticosteroid therapy subjected to unusual stress, increased dosage of rapidly acting corticosteroids before, during and after the stressful situation is indicated. Administration of live virus vaccines is contraindicated in individuals receiving immunosuppressive doses of corticosteroids. If inactivated viral or bacterial vaccines are administered to individuals receiving immunosuppressive doses of corticosteroids, the expected serum antibody response may not be obtained. However, immunization procedures may be undertaken in patients who are receiving corticosteroids as replacement therapy, e.g., for Addison's disease.

The use of dexamethasone in active tuberculosis should be restricted to those cases of fulminating or disseminated tuberculosis in which the corticosteroid is used for the management of the disease in conjunction with an appropriate antituberculous regimen. If corticosteroids are indicated in patients with latent tuberculosis or tuberculin reactivity, close observation is necessary as reactivation of the disease may occur. During prolonged corticosteroid therapy, these patients should receive chemoprophylaxis.

Corticosteroids may mask some signs of infection, and new infections may appear during their use. There may be decreased resistance and inability to localize infection when corticosteroids are used. If corticosteroids have to be used in the presence of bacterial infections, institute appropriate anti-infective therapy. Patients exposed to certain infections (e.g., measles, chickenpox) should seek medical advice. Corticosteroids may exacerbate systemic fungal infections and therefore should not be used in the presence of such infections unless they are needed to control drug reactions due to amphotericin B. Moreover, there have been cases reported in which concomitant use of amphotericin B and hydrocortisone was followed by cardiac enlargement and congestive failure.

In cerebral malaria, the use of corticosteroids is associated with prolongation of coma and a higher incidence of pneumonia and gastrointestinal bleeding.

Corticosteroids may activate latent amebiasis. Therefore, it is recommended that latent or active amebiasis be ruled out before initiating corticosteroid therapy in any patient who has spent time in

the tropics or in any patient with unexplained diarrhea.

Corticosteroids should be used cautiously in patients with ocular herpes simplex because of possible corneal perforation.

Prolonged use of corticosteroids may produce posterior subcapsular cataracts, glaucoma with possible damage to the optic nerves, and may enhance the establishment of secondary ocular infections due to fungi or viruses.

Literature reports suggest an apparent association between use of corticosteroids and left ventricular free wall rupture after a recent myocardial infarction; therefore, therapy with corticosteroids should be used with great caution in these patients.

Fluid and electrolyte balance:

Average and large doses of hydrocortisone or cortisone can cause elevation of blood pressure, salt and water retention and increased potassium excretion. These effects are less likely to occur with the synthetic derivatives except when used in large doses. Dietary salt restriction and potassium supplementation may be necessary. All corticosteroids increase calcium excretion.

Renal function impairment:

Edema may occur in the presence of renal disease with a fixed or decreased glomerular filtration rate. A degree of caution is advised when corticosteroids are used in patients with renal insufficiency, acute glomerulonephritis and chronic nephritis.

Gastrointestinal effects:

The association between peptic ulceration and corticosteroid therapy remains controversial. However, corticosteroid therapy may mask the symptoms of peptic ulcer. Perforation or hemorrhage may occur without significant pain.

Corticosteroids should be used with caution in patients with diverticulitis, fresh intestinal anastomoses, active or latent peptic ulcer and in nonspecific ulcerative colitis, if there is a probability of impending perforation, abscess or other pyogenic infection.

Immunosuppression:

Patients being treated with corticosteroids should not be vaccinated against smallpox. Other immunization procedures should generally not be undertaken in these patients, especially those on high doses, because of possible neurological complications and a lack of antibody response. Immunization procedures may be undertaken in patients who are receiving corticosteroids as replacement therapy. Corticosteroids may suppress reactions to skin tests.

Adrenal suppression:

Following prolonged therapy, abrupt discontinuation may result in withdrawal syndrome and secondary adrenocortical insufficiency. Symptoms of adrenal insufficiency resulting from rapid withdrawal include: nausea, fatigue, anorexia, dyspnea, hypotension, hypoglycemia, myalgia, fever, malaise, arthralgia, dizziness, desquamation of skin and fainting. This type of relative insufficiency may persist for months after discontinuation of therapy; therefore, in any stressful situation occurring during that period, reinstitute hormone therapy. If the patient is receiving corticosteroids already, the dosage may have to be increased. Since mineralocorticoid secretion may be impaired, salt and/or a mineralocorticoid may need to be used.

Hypersensitivity:

Rare instances of anaphylactoid reactions have occurred in patients receiving parenteral corticosteroid therapy. Appropriate precautionary measures should be taken prior to administration, especially when the patient has a history of allergy to any drug. Some corticosteroid products contain tartrazine and sodium bisulfite, both of which may cause severe allergic reactions in susceptible individuals.

Pregnancy: Some animal studies have shown that corticosteroids, when administered to the mother at high doses, may cause fetal malformations. Since adequate human reproduction studies have not been done with corticosteroids, the use of these drugs in pregnancy, nursing mothers or women of child-bearing potential requires that the benefits of the drug be carefully weighed against the potential risks to both mother and fetus. Corticosteroids cross the placenta. Infants born of

mothers who have received substantial doses of corticosteroids during pregnancy should be carefully observed for signs of hypoadrenalism.

Lactation: Corticosteroids appear in breast milk and could suppress growth, interfere with endogenous corticosteroid production or cause other unwanted effects. Mothers taking pharmacologic doses of corticosteroids should be advised not to nurse.

Children:
Prolonged therapy with corticosteroids in infants and children should be avoided if possible since corticosteroids may suppress growth. If corticosteroid therapy is deemed essential, institute alternate day therapy should be considered to minimize this side effect. Growth and development should be closely monitored.

PRECAUTIONS

Because complications of treatment with corticosteroids are dependent on the dosage regimen, a risk/benefit decision must be made in each individual case with respect to dose and duration of treatment and whether daily or intermittent therapy should be used. The lowest possible dose of corticosteroid should be used to control the condition under treatment, and when reduction in dosage is possible, the reduction should be gradual.

Drug-induced secondary adrenocortical insufficiency may result from too rapid withdrawal of corticosteroids and may be minimized by gradual reduction of dosage. This type of relative insufficiency may persist for months after discontinuation of therapy; therefore, in any situation of stress occurring during that period, corticosteroid therapy should be reinstituted or the current dosage may have to be increased. Since mineralocorticoid secretion may be impaired, salt and/or a mineralocorticoid should be administered concurrently.

Steroids should be used with caution in: nonspecific ulcerative colitis if there is a probability of impending perforation, abscess or other pyogenic infection; diverticulitis; fresh intestinal anastomoses; active or latent peptic ulcer, renal insufficiency; hypertension; osteoporosis; and myasthenia gravis. Signs of peritoneal irritation following gastrointestinal perforation in patients receiving large doses of corticosteroids may be minimal or absent. Fat embolism has been

reported as a possible complication of hypercortisonism.

There is an enhanced effect of corticosteroids in patients with hypothyroidism and in those with cirrhosis.

Psychic derangements may appear when corticosteroids are used, ranging from euphoria, insomnia, mood swings, personality changes and severe depression to frank psychotic manifestations; these reactions are sometimes seen following sharp decreases in corticosteroid dosage or during pulse therapy. Existing emotional instability or psychotic tendencies may be aggravated by corticosteroids.

Steroids may increase or decrease motility and number of spermatozoa in some patients.

Drug Interactions

ASA and nonsteroidal anti-inflammatory agents should be used cautiously in conjunction with corticosteroids in patients with hypoprothrombinemia.

Phenytoin, phenobarbital, ephedrine and rifampin may enhance the metabolic clearance of corticosteroids, resulting in decreased blood levels and lessened physiologic activity, thus requiring adjustment in corticosteroid dosage. These interactions may interfere with dexamethasone suppression tests which should be interpreted with caution during administration of these drugs.

False-negative results in the dexamethasone suppression test in patients being treated with indomethacin have been reported.

The prothrombin time should be checked frequently in patients who are receiving corticosteroids and coumarin anticoagulants at the same time because of reports that corticosteroids have altered the response to these anticoagulants. Studies have shown that the usual effect produced by adding corticosteroids is inhibition of response to coumarins, although there have been some conflicting reports of potentiation not substantiated by studies.

When corticosteroids are administered concomitantly with potassium-depleting diuretics, patients should be observed closely for development of hypokalemia.

Corticosteroids may affect the nitroblue tetrazolium test for bacterial infection and produce false-

negative results.

Patients who are on drugs which suppress the immune system are more susceptible to infections than healthy individuals. Chickenpox and measles, for example, can have a more serious or even fatal course in non-immune children or adults on corticosteroids. In such children or adults who have not had these diseases, particular care should be taken to avoid exposure. The risk of developing a disseminated infection varies among individuals and may be related to the dose, route and duration of corticosteroid administration as well as to the underlying disease. Exposed patients should be advised to seek medical advice without delay. If exposed to measles, prophylaxis with i.m. pooled immunoglobulin (IG) may be indicated. If exposed to chickenpox, prophylaxis with varicella zoster immune globulin (VZIG) may be indicated (see the respective Product Monographs for VZIG and IG for complete prescribing information). If chickenpox develops, treatment with antiviral agents should be considered.

When used in myasthenia gravis, hospitalization with careful observation is recommended because a transient worsening of symptoms possibly leading to respiratory distress may precede clinical improvement.

Avascular or aseptic necrosis of the femoral head has been associated with long-term corticosteroid treatment; however, it has also occurred in patients receiving high dose, short-term therapy. This adverse effect is more likely to occur in patients with a predisposing illness such as rheumatoid arthritis or systemic lupus erythematosis.

Drug-laboratory test interactions: Corticosteroids may decrease I^{131} uptake and produce false negative results in the nitroblue tetrazolium test for systemic bacterial infection.

ADVERSE REACTIONS

Fluid and electrolyte disturbances: sodium retention, fluid retention, congestive heart failure in susceptible patients, potassium loss, hypokalemic alkalosis, hypertension.

Musculoskeletal: muscle weakness, steroid myopathy, loss of muscle mass, osteoporosis, vertebral compression fractures, aseptic necrosis of femoral and humeral heads, pathologic fracture of long bones, tendon rupture.

Gastrointestinal: nausea, vomiting, anorexia which may result in weight loss; increased appetite which may result in weight gain; diarrhea or constipation, abdominal distention, pancreatitis, gastric irritation and ulcerative esophagitis; peptic ulcer with possible perforation and hemorrhage; perforation of the small and large bowel particularly in inflammatory bowel diseases..

Dermatologic: Impaired wound healing; thin fragile skin; petechiae and ecchymoses; facial erythema; striae; hirsutism; acneiform eruptions; suppressed reactions to skin tests; hypersensitivity reactions such as allergic dermatitis, urticaria, angioneurotic edema.

Neurologic: Seizures; increased intracranial pressure with papilledema (pseudotumor cerebri) in association with withdrawal of corticosteroid therapy; convulsions; vertigo; headache; psychic disturbances; neuritis; paresthesias.

Endocrine: Decreased carbohydrate tolerance; hyperglycemia; glycosuria; increased requirements for oral hypoglycemics or insulin in diabetes; manifestations of latent diabetes mellitus; menstrual irregularities; development of Cushingoid state; suppression of growth in children; secondary adrenocortical and pituitary unresponsiveness, particularly in times of stress, as in trauma, surgery or illness; hirsutism; increased sweating.

Ophthalmic: Increased intraocular pressure; glaucoma; exophthalmos; posterior subcapsular cataracts.

Metabolic: negative nitrogen balance due to protein catabolism.

Psychologic: Hallucinations; psychosis; euphoria; mood changes.

Cardiovascular: Thromboembolism; fat embolism; hypercholesterolemia; accelerated atherosclerosis; cardiac arrhythmias or ECG changes due to potassium deficiency; syncope; aggravation of hypertension; myocardial rupture following recent myocardial infarction; reports of cardiac arrhythmias, fatal arrest or circulatory collapse following rapid administration of intravenous methylprednisolone greater than 0.5 g given over a period of less than 10 minutes.

Hematologic: Leukocytosis, thrombocytopenia, lymphopenia.

Others: Hypersensitivity; thrombophlebitis; weight gain; increased appetite; nausea; malaise; hiccups; necrotizing angiitis; aggravation or masking of infections; insomnia; anaphylactoid reactions.

SYMPTOMS AND TREATMENT OF OVERDOSAGE

There is no known antidote but gastric lavage should be performed. Acute overdose even after ingestion of large doses is rarely a clinical problem. Continuous overdosage requires careful reduction of dosage in order to prevent the occurrence of acute adrenal insufficiency.

Anaphylactic and hypersensitivity reactions depending on their severity, may be treated with antihistamines with or without epinephrine. General supportive measures should also be employed.

For the management of suspected drug overdose, contact your regional Poison Control Centre.

DOSAGE AND ADMINISTRATION

Dosage requirements are variable and must be individualized according to the severity of the disease and the response of the patient. The usual initial dosage varies from 0.5 to 15 mg per day depending on the disease being treated. For infants and children, the recommended doses usually will have to be reduced, but dosage should be dictated by the severity of the condition rather than by age or body weight.

Corticosteroid therapy is an adjunct to, not a replacement of conventional therapy, which should be instituted as indicated.

Dosage must be decreased or therapy discontinued gradually when administration has been continued for more than a few days.

In acute conditions where prompt relief is urgent, large doses are permissible and may be mandatory for a short period. When symptoms have been suppressed adequately, dosage should be maintained at the minimum amount capable of providing sufficient relief without excessive hormonal effects.

Chronic conditions are subject to periods of spontaneous remission. When such periods occur, corticosteroids should be discontinued gradually.

Routine laboratory studies such as urinalysis, 2-hour post-prandial blood sugar, determinations of blood pressure and body weight, and a chest x-ray should be carried out at regular intervals during prolonged therapy. Periodic determinations of serum potassium are advisable if large doses are being used.

Patients may be transferred to dexamethasone from any other glucocorticoid with the proper adjustment in dosage.

The following mg equivalents facilitate changing to dexamethasone from other glucocorticoids (see Table I).

Table I Dexamethasone Tablets mg Equivalents

Dexamethasone	Methyl-prednisolone and Triamcinolone	Prednisolone and Prednisone	Hydrocortisone	Cortisone
0.75 mg	4 mg	5 mg	20 mg	25 mg

Milligram for milligram, dexamethasone is approximately equivalent to betamethasone, 4 to 6 times more potent than methylprednisolone and triamcinolone, 6 to 8 times more potent than prednisone and prednisolone, 25 to 30 times more potent than hydrocortisone, and about 35 times more potent than cortisone. At equipotent anti-inflammatory doses, dexamethasone almost completely lacks the sodium-retaining property of hydrocortisone and closely related derivatives of hydrocortisone.

Specific dosage recommendations: In chronic, usually nonfatal diseases including endocrine and chronic rheumatic disorders, edematous states, respiratory and gastrointestinal diseases, some dermatologic diseases and hematologic disorders, start with a low dose (0.5 to 1 mg a day) and gradually increase dosage to the smallest amount that gives the desired degree of symptomatic relief.

Dosage may be administered 2, 3 or 4 times a day.

In congenital adrenal hyperplasia, the usual daily dose is 0.5 to 1.5 mg.

In acute, nonfatal diseases, including allergic states, ophthalmic diseases, acute and subacute rheumatic disorders, dosage ranges between 2 and 3 mg a day, however, higher doses are necessary in some patients. Since the course of these conditions is self-limited, prolonged maintenance therapy is not usually necessary.

Antiemetic prophylaxis during emetogenic chemotherapy: dexamethasone administered concomitantly with ondansetron has been demonstrated to achieve enhanced efficacy for antiemetic prophylaxis during emetogenic chemotherapy. Various dosing schedules have been used in clinical studies; however, the following is suggested for this combination: 8 to 20 mg of dexamethasone infused over 5 to 15 minutes just prior to chemotherapy, followed by 4 mg of dexamethasone orally every 4 to 6 hours, or by 8 mg orally every 8 hours, and tapered in either strength or frequency of administration over 2 to 3 days. In general the total treatment duration for this indication should not exceed 5 days beyond chemotherapy. Alternatively, injectable dexamethasone can be infused intravenously in lieu of an oral formulation of dexamethasone using various schedules.

In acute, self-limited allergic disorders or acute exacerbations of chronic allergic disorders (e.g., acute allergic rhinitis, acute attacks of seasonal allergic bronchial asthma, urticaria medicamentosa, and contact dermatoses), the following dosage schedule, combining parenteral and oral therapy, is suggested (see Table II).

Table II: Dosage schedule

		Total daily dosage	Number of 0.5 mg tablets
1st day	1 or 2 mL, intramuscular dexamethasone phosphate injection (4 mg / mL)	4 or 8 mg	
2nd day	2 tablets dexamethasone (0.5 mg) twice a day	2 mg	4
3rd day	2 tablets dexamethasone (0.5 mg) twice a day	2 mg	4
4th day	1 tablet dexamethasone (0.5 mg) twice a day	1 mg	2
5th day	1 tablet dexamethasone (0.5 mg) twice a day	1 mg	2
6th day	1 tablet dexamethasone (0.5 mg) / day	0.5 mg	1
7th day	1 tablet dexamethasone (0.5 mg) / day	0.5 mg	1
8th day	Follow-up visit		

In chronic, potentially fatal diseases such as systemic lupus erythematosus, pemphigus, symptomatic sarcoidosis, the recommended initial dosage is 2 to 4.5 mg a day; higher doses may be necessary in some patients.

When the disease is acute and life-threatening (e.g., acute rheumatic carditis, crisis of systemic lupus erythematosus, severe allergic reactions, pemphigus, neoplastic diseases), the initial dosage is between 4 and 10 mg a day, administered in at least 4 divided doses.

Epinephrine is the drug of immediate choice in severe allergic reactions. Dexamethasone is useful either concurrently or as supplementary therapy.

In cerebral edema, when maintenance therapy is required. For palliative management of patients with recurrent or inoperable brain tumors, a dosage of 2 mg, 2 or 3 times a day may be effective. The smallest dosage necessary to control cerebral edema should be utilized.

In the adrenogenital syndrome, daily dosages of 0.5 to 1.5 mg may keep children in remission and prevent the recurrence of abnormal excretion of 17-ketosteroids.

As massive therapy in certain conditions, such as acute leukemia, the nephrotic syndrome, and pemphigus, the recommended dosage is from 10 to 15 mg a day. Patients receiving such a high dosage must be observed very closely for the appearance of severe reactions.

Dexamethasone Suppression Tests:

1. Test for Cushing's syndrome: Give 1 mg orally at 11:00 p.m. Blood is drawn for plasma cortisol determination at 8:00 a.m. the following morning. For greater accuracy, give 0.5 mg dexamethasone orally every 6 hours for 48 hours. Twenty-four hour urine collections are made for determination of 17-hydroxycorticosteroid excretion.

2. Test to distinguish Cushing's syndrome due to pituitary ACTH excess from Cushing's syndrome due to other causes. Give 2 mg of dexamethasone orally every 6 hours for 48 hours. Twenty-four hour urine collections are made for determination of 17-hydroxycorticosteroid excretion.

AVAILABILITY OF DOSAGE FORMS

APO-DEXAMETHASONE 0.5 mg tablets: Each yellow, round, flat faced, bevelled edge tablet, engraved "APO" on one side and scored and engraved “DEX” over "0.5” on the other contains 0.5 mg dexamethasone. Available in bottles of 100.

APO-DEXAMETHASONE 4 mg tablets: Each white, pentagonal, flat tablet, engraved "APO" over "4" on one side, and scored on the other, contains 4 mg dexamethasone. Available in bottles of 50 and 100.

Composition

In addition to dexamethasone, each tablet contains the non-medicinal ingredients: lactose, magnesium stearate and corn starch. APO-DEXAMETHASONE 0.5 mg tablets also contain the non-medicinal ingredient D & C Yellow #10 Aluminium Lake 14-18 %.

Stability and Storage Recommendations

Store at 15°C to 30°C.

Reporting Side Effects

You can report any suspected side effects associated with the use of health products to Health Canada by:

- Visiting the Web page on Adverse Reaction Reporting (https://www.canada.ca/en/health-canada/services/drugs-health-products/medeffect-canada/adverse-reaction-reporting.html) for information on how to report online, by mail or by fax; or
- Calling toll-free at 1-866-234-2345.

NOTE: Contact your health professional if you need information about how to manage your side effects. The Canada Vigilance Program does not provide medical advice

PHARMACEUTICAL INFORMATION

Drug Substance

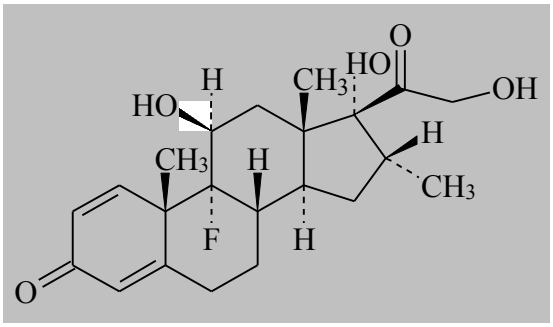
Proper Name: Dexamethasone

Chemical Names:

1) Pregna-1,4-diene-3,20-dione, 9-fluoro-11,17,21-trihydroxy-16-methyl-, (11β, 16α)-

2) 9-fluoro-11β,17,21-trihydroxy-16α-methylpregna-1,4-diene-3,20-dione

Structural Formula:



Molecular Formula: C22H29FO5

Molecular Weight: 392.5 g/mol

Description: White to practically white, odorless, crystalline powder. Is stable in air. Melts at about 250°, with some decomposition. Practically insoluble in water; sparingly soluble in acetone, in alcohol, in dioxane, and in methanol; slightly soluble in chloroform; very slightly soluble in ether.

REFERENCES

1. Product Monograph- ratio-DEXAMETHASONE (dexamethasone USP) 0.5 mg and 4 mg Tablets. Teva Canada Limited, Submission Control No: 162516, Date of Revision: February 26, 2013.