

What are the possible side effects of NSAIDs?

NSAIDs can cause serious side effects, including:

See “What is the most important information I should know about medicines called Nonsteroidal Anti-inflammatory Drugs (NSAIDs)?”

- new or worse high blood pressure
- heart failure
- liver problems including liver failure
- kidney problems including kidney failure
- low red blood cells (anemia)
- life-threatening skin reactions
- life-threatening allergic reactions
- **Other side effects of NSAIDs include:** stomach pain, constipation, diarrhea, gas, heartburn, nausea, vomiting, and dizziness.

Get emergency help right away if you have any of the following symptoms:

- shortness of breath or trouble breathing
- chest pain
- weakness in one part or side of your body
- slurred speech
- swelling of the face or throat

Stop taking your NSAID and call your healthcare provider right away if you get any of the following symptoms:

- nausea
- more tired or weaker than usual
- diarrhea
- itching
- your skin or eyes look yellow
- indigestion or stomach pain
- flu-like symptoms
- vomit blood
- there is blood in your bowel movement or it is black and sticky like tar
- unusual weight gain
- skin rash or blisters with fever
- swelling of the arms, legs, hands and feet

If you take too much of your NSAID, call your healthcare provider or get medical help right away.

These are not all the possible side effects of NSAIDs. For more information, ask your healthcare provider or pharmacist about NSAIDs.

Call your doctor for medical advice about side effects. You may report side effects to FDA at 1-800-FDA-1088.

Other information about NSAIDs

- Aspirin is an NSAID medicine but it does not increase the chance of a heart attack. Aspirin can cause bleeding in the brain, stomach, and intestines. Aspirin can also cause ulcers in the stomach and intestines.
- Some NSAIDs are sold in lower doses without a prescription (over-the-counter). Talk to your healthcare provider before using over-thecounter NSAIDs for more than 10 days.

General information about the safe and effective use of NSAIDs

Medicines are sometimes prescribed for purposes other than those listed in a Medication Guide. Do not use NSAIDs for a condition for which it was not prescribed. Do not give NSAIDs to other people, even if they have the same symptoms that you have. It may harm them.

If you would like more information about NSAIDs, talk with your healthcare provider. You can ask your pharmacist or healthcare provider for information about NSAIDs that is written for health professionals.

Manufactured and Distributed by:

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For more information, go to
www.carlsbadtech.com or call (760) 431-8284.

This Medication Guide has been approved by the U.S. Food and Drug Administration

Cardiovascular Thrombotic Events:

Advise patients to be alert for the symptoms of cardiovascular thrombotic events, including chest pain, shortness of breath, weakness, or slurring of speech, and to report any of these symptoms to their healthcare provider immediately (see WARNINGS: Cardiovascular Thrombotic Events).

Gastrointestinal Bleeding, Ulceration, and Perforation

Advise patients to report symptoms of ulcerations and bleeding, including epigastric pain, dyspepsia, melena, and hematemesis to their health care provider. In the setting of concomitant use of low-dose aspirin for cardiac prophylaxis, inform patients of the increased risk for the signs and symptoms of GI bleeding (see WARNING: Gastrointestinal Bleeding, Ulceration, and Perforation).

Hepatotoxicity

Inform patients of the warning signs and symptoms of hepatotoxicity (e.g., nausea, fatigue, lethargy, pruritus, diarrhea, jaundice, right upper quadrant tenderness, and “flu-like” symptoms). If these occur, instruct patients to stop diclofenac and seek immediate medical therapy (see WARNINGS; Hepatotoxicity).

Heart Failure and Edema:

Advise patients to be alert for the symptoms of congestive heart failure including shortness of breath, unexplained weight gain, or edema and to contact their healthcare provider if such symptoms occur (see WARNINGS; Heart Failure and Edema).

Anaphylactic Reactions

Inform patients of the signs of an anaphylactic reaction (eg, difficulty breathing, swelling of the face or throat). Instruct patients to seek immediate emergency help if these occur (see WARNINGS; Anaphylactic Reactions). Serious Skin Reactions, including DRESS Advise patients to stop taking Diclofenac Sodium immediately if they develop any type of rash or fever and to contact their healthcare provider as soon as possible [see *Warnings*].

Female Fertility

Advise females of reproductive potential who desire pregnancy that NSAIDs, including diclofenac, may be associated with a reversible delay in ovulation (see PRECAUTIONS; Carcinogenesis, Mutagenesis, Impairment of Fertility).

Fetal Toxicity

Inform pregnant women to avoid use of Diclofenac Sodium and other NSAIDs, starting at 30 weeks gestation because of the risk of the premature closure of the fetal ductus arteriosus. If treatment with Diclofenac Sodium is needed for a pregnant woman between about 20 to 30 weeks gestation, advise her that she may need to be monitored for oligohydramnios, if treatment continues for longer than 48 hours [see WARNINGS; Fetal Toxicity; PRECAUTIONS; Pregnancy].

Avoid Concomitant Use of NSAIDs

Inform patients that the concomitant use of diclofenac with other NSAIDs or salicylates (e.g., diflunisal, salsalate) is not recommended due to the increased risk of gastrointestinal toxicity, and little or no increase in efficacy (see WARNINGS; Gastrointestinal Bleeding, Ulceration, and Perforation and Drug Interactions). Alert patients that NSAIDs may be present in “over the counter” medications for treatment of colds, fever, or insomnia. Use of NSAIDs and Low-Dose Aspirin Inform patients not to use low-dose aspirin concomitantly with diclofenac until they talk to their healthcare provider (see PRECAUTIONS; Drug Interactions).

Masking of Inflammation and Fever

The pharmacological activity of diclofenac in reducing inflammation, and possibly fever, may diminish the utility of diagnostic signs in detecting infections.

Laboratory Monitoring

Because serious GI bleeding, hepatotoxicity, and renal injury can occur without warning symptoms or signs, consider monitoring patients on long-term NSAID treatment with a CBC and a chemistry profile periodically (see WARNINGS; Gastrointestinal Bleeding, Ulceration and Perforation, and Hepatotoxicity).

Drug Interactions

See Table 2 for clinically significant drug interactions with diclofenac.

Table 2: Clinically Significant Drug Interactions with Diclofenac

Drugs That Interfere with Hemostasis	
<i>Clinical Impact:</i>	• Diclofenac and anticoagulants such as warfarin have a synergistic effect on bleeding. The concomitant use of diclofenac and anticoagulants have an increased risk of serious bleeding compared to the use of either drug alone. • Serotonin release by platelets plays an important role in hemostasis. Case-control and cohort epidemiological studies showed that concomitant use of drugs that interfere with serotonin reuptake and an NSAID may potentiate the risk of bleeding more than an NSAID alone.
<i>Intervention:</i>	Monitor patients with concomitant use of diclofenac with anticoagulants (e.g., warfarin), antiplatelet agents (e.g., aspirin), selective serotonin reuptake inhibitors (SSRIs), and serotonin norepinephrine reuptake inhibitors (SNRIs) for signs of bleeding (see PRECAUTIONS; Hematological Toxicity).
Aspirin	
<i>Clinical Impact:</i>	Controlled clinical studies showed that the concomitant use of NSAIDs and analgesic doses of aspirin does not produce any greater therapeutic effect than the use of NSAIDs alone. In a clinical study, the concomitant use of an NSAID and aspirin was associated with a significantly increased incidence of GI adverse reactions as compared to use of the NSAID alone (see WARNINGS; Gastrointestinal Bleeding, Ulceration, and Perforation).
<i>Intervention:</i>	Concomitant use of diclofenac and analgesic doses of aspirin is not generally recommended because of the increased risk of bleeding (see PRECAUTIONS: Hematological Toxicity). Diclofenac is not a substitute for low dose aspirin for cardiovascular protection.
ACE Inhibitors, Angiotensin Receptor Blockers, and Beta-Blockers	
<i>Clinical Impact:</i>	• NSAIDs may diminish the antihypertensive effect of angiotensin converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), or beta-blockers (including propranolol). • In patients who are elderly, volume-depleted (including those on diuretic therapy), or have renal impairment, co-administration of an NSAID with ACE inhibitors or ARBs may result in deterioration of renal function, including possible acute renal failure. These effects are usually reversible.
<i>Intervention:</i>	• During concomitant use of diclofenac and ACE-inhibitors, ARBs, or betablockers, monitor blood pressure to ensure that the desired blood pressure is obtained. • During concomitant use of diclofenac and ACE-inhibitors or ARBs in patients who are elderly, volume-depleted, or have impaired renal function, monitor for signs of worsening renal function (see WARNINGS; Renal Toxicity and Hyperkalemia). • When these drugs are administered concomitantly, patients should be adequately hydrated. Assess renal function at the beginning of the concomitant treatment and periodically thereafter.

Diuretics	
<i>Clinical Impact:</i>	Clinical studies, as well as post-marketing observations, showed that NSAIDs reduced the natriuretic effect of loop diuretics (e.g., furosemide) and thiazide diuretics in some patients. This effect has been attributed to the NSAID inhibition of renal prostaglandin synthesis.
<i>Intervention:</i>	During concomitant use of diclofenac with diuretics, observe patients for signs of worsening renal function, in addition to assuring diuretic efficacy including antihypertensive effects (see WARNINGS; Renal Toxicity and Hyperkalemia).
Digoxin	
<i>Clinical Impact:</i>	The concomitant use of diclofenac with digoxin has been reported to increase the serum concentration and prolong the half-life of digoxin.
<i>Intervention:</i>	During concomitant use of diclofenac and digoxin, monitor serum digoxin levels.
Lithium	
<i>Clinical Impact:</i>	NSAIDs have produced elevations in plasma lithium levels and reductions in renal lithium clearance. The mean minimum lithium concentration increased 15%, and the renal clearance decreased by approximately 20%. This effect has been attributed to NSAID inhibition of renal prostaglandin synthesis.
<i>Intervention:</i>	During concomitant use of diclofenac and lithium, monitor patients for signs of lithium toxicity.
Methotrexate	
<i>Clinical Impact:</i>	Concomitant use of NSAIDs and methotrexate may increase the risk for methotrexate toxicity (e.g., neutropenia, thrombocytopenia, renal dysfunction).
<i>Intervention:</i>	During concomitant use of diclofenac and methotrexate, monitor patients for methotrexate toxicity.
Cyclosporine	
<i>Clinical Impact:</i>	Concomitant use of diclofenac and cyclosporine may increase cyclosporine’s nephrotoxicity.
<i>Intervention:</i>	During concomitant use of diclofenac and cyclosporine, monitor patients for signs of worsening renal function.
NSAIDs and Salicylates	
<i>Clinical Impact:</i>	Concomitant use of diclofenac with other NSAIDs or salicylates (e.g., diflunisal, salsalate) increases the risk of GI toxicity, with little or no increase in efficacy (see WARNINGS; Gastrointestinal Bleeding, Ulceration, and Perforation).
<i>Intervention:</i>	Concomitant use of diclofenac with other NSAIDs or salicylates is not recommended.
Pemetrexed	
<i>Clinical Impact:</i>	Concomitant use of diclofenac and pemetrexed may increase the risk of pemetrexedassociated myelosuppression, renal, and GI toxicity (see the pemetrexed prescribing information).
<i>Intervention:</i>	During concomitant use of diclofenac and pemetrexed, in patients with renal impairment whose creatinine clearance ranges from 45 to 79 mL/min, monitor for myelosuppression, renal and GI toxicity. NSAIDs with short elimination half-lives (e.g., diclofenac, indomethacin) should be avoided for a period of two days before, the day of, and two days following administration of pemetrexed. In the absence of data regarding potential interaction between pemetrexed and NSAIDs with longer half-lives (e.g., meloxicam, nabumetone), patients taking these NSAIDs should interrupt dosing for at least five days before, the day of, and two days following pemetrexed administration.
CYP2C9 Inhibitors or Inducers:	
<i>Clinical Impact:</i>	Diclofenac is metabolized by cytochrome P450 enzymes, predominantly by CYP2C9. Co-administration of diclofenac with CYP2C9 inhibitors (e.g. voriconazole) may enhance the exposure and toxicity of diclofenac whereas co-administration with CYP2C9 inducers (e.g. rifampin) may lead to compromised efficacy of diclofenac.
<i>Intervention:</i>	Dosage adjustment may be warranted when diclofenac is administered with CYP2C9 inhibitors or inducers (see CLINICAL PHARMACOLOGY; Pharmacokinetics).

Carcinogenesis, Mutagenesis, Impairment of Fertility

Carcinogenesis

Long-term carcinogenicity studies in rats given diclofenac sodium up to 2 mg/ kg/day (approximately 0.1 times maximum recommended human dose (MRHD) of diclofenac, 200 mg/day, based on body surface area (BSA) comparison) have revealed no significant increases in tumor incidence. A 2-year carcinogenicity study conducted in mice employing diclofenac sodium at doses up to 0.3 mg/kg/day (approximately 0.007 times the MRHD based on BSA comparison) in males and 1 mg/kg/day (approximately 0.02 times the MRHD based on BSA comparison) in females did not reveal any oncogenic potential.

Mutagenesis

Diclofenac sodium did not show mutagenic activity in in vitro point mutation assays in mammalian (mouse lymphoma) and microbial (yeast, Ames) test systems and was nonmutagenic in several mammalian in vitro and in vivo tests, including dominant lethal and male germinal epithelial chromosomal studies in mice, and nucleus anomaly and chromosomal aberration studies in Chinese hamsters.

Impairment of Fertility

Diclofenac sodium administered to male and female rats at 4 mg/kg/day (approximately 0.2 times the MRHD based on BSA comparison) did not affect fertility. Based on the mechanism of action, the use of prostaglandin-mediated NSAIDs, including diclofenac, may delay or prevent rupture of ovarian follicles, which has been associated with reversible infertility in some women. Published animal studies have shown that administration of prostaglandin synthesis inhibitors has the potential to disrupt prostaglandin-mediated follicular rupture required for ovulation. Small studies in women treated with NSAIDs have also shown a reversible delay in ovulation. Consider withdrawal of NSAIDs, including diclofenac, in women who have difficulties conceiving or who are undergoing investigation of infertility.

Pregnancy

Risk Summary

Use of NSAIDs, including Diclofenac Sodium, can cause premature closure of the fetal

ductus arteriosus and fetal renal dysfunction leading to oligohydramnios and, in some cases, neonatal renal impairment. Because of these risks, limit dose and duration of Diclofenac Sodium use between about 20 and 30 weeks of gestation, and avoid Diclofenac Sodium use at about 30 weeks of gestation and later in pregnancy.

Premature Closure of Fetal Ductus Arteriosus

Use of NSAIDs, including Diclofenac Sodium, at about 30 weeks gestation or later in pregnancy increases the risk of premature closure of the fetal ductus arteriosus.

Oligohydramnios/Neonatal Renal Impairment

Use of NSAIDs at about 20 weeks gestation or later in pregnancy has been associated with cases of fetal renal dysfunction leading to oligohydramnios, and in some cases, neonatal renal impairment.

Data from observational studies regarding other potential embryofetal risks of NSAID use in women in the first or second trimesters of pregnancy are inconclusive. In animal reproduction studies, no evidence of teratogenicity was observed in mice, rats or rabbits given diclofenac during the period of organogenesis at doses up to approximately 0.5 and 1 times, respectively, the maximum recommended human dose (MRHD) of diclofenac sodium delayed-release, 200mg/day, despite the presence of maternal and fetal toxicity at these doses (see Data).

The estimated background risk of major birth defects and miscarriage for the indicated population(s) is unknown. All pregnancies have a background risk of birth defect, loss, or other adverse outcomes. In the U.S. general population, the estimated background risk of major birth defects and miscarriage in clinically recognized pregnancies is 2-4% and 15-20%, respectively. [see WARNINGS; Fetal Toxicity].

Clinical Considerations

Fetal/Neonatal Adverse Reactions

Premature Closure of Fetal Ductus Arteriosus:

Avoid use of NSAIDs in women at about 30 weeks gestation and later in pregnancy, because NSAIDs, including Diclofenac Sodium, can cause premature closure of the fetal ductus arteriosus (see WARNINGS; Fetal Toxicity).

Oligohydramnios/Neonatal Renal Impairment

If an NSAID is necessary at about 20 weeks gestation or later in pregnancy, limit the use to the lowest effective dose and shortest duration possible. If Diclofenac Sodium treatment extends beyond 48 hours, consider monitoring with ultrasound for oligohydramnios. If oligohydramnios occurs, discontinue Diclofenac Sodium and follow up according to clinical practice (see WARNINGS; Fetal Toxicity).

Data

Human Data

Premature Closure of Fetal Ductus Arteriosus:

Oligohydramnios/Neonatal Renal Impairment

Published studies and postmarketing reports describe maternal NSAID use at about 20 weeks gestation or later in pregnancy associated with fetal renal dysfunction leading to oligohydramnios, and in some cases, neonatal renal impairment. These adverse outcomes are seen, on average, after days to weeks of treatment, although oligohydramnios has been infrequently reported as soon as 48 hours after NSAID initiation. In many cases, but not all, the decrease in amniotic fluid was transient and reversible with cessation of the drug. There have been a limited number of case reports of maternal NSAID use and neonatal renal dysfunction without oligohydramnios, some of which were irreversible. Some cases of neonatal renal dysfunction required treatment with invasive procedures, such as exchange transfusion or dialysis.

Methodological limitations of these postmarketing studies and reports include lack of a control group; limited information regarding dose, duration, and timing of drug exposure; and concomitant use of other medications. These limitations preclude establishing a reliable estimate of the risk of adverse fetal and neonatal outcomes with maternal NSAID use. Because the published safety data on neonatal outcomes involved mostly preterm infants, the generalizability of certain reported risks to the full-term infant exposed to NSAIDs through maternal use is uncertain.

Data

Animal Data

Reproductive and developmental studies in animals demonstrated that diclofenac sodium administration during organogenesis did not produce teratogenicity despite the induction of maternal toxicity and fetal toxicity in mice at oral doses up to 20 mg/kg/day (approximately 0.5 times the maximum recommended human dose [MRHD] of diclofenac, 200 mg/day, based on body surface area (BSA) comparison), and in rats and rabbits at oral doses up to 10 mg/kg/day (approximately 0.5 and 1 times, respectively, the MRHD based on BSA comparison). In a study in which pregnant rats were orally administered 2 or 4 mg/kg diclofenac (0.1 and 0.2 times the MRHD based on BSA) from Gestation Day 15 through Lactation Day 21, significant maternal toxicity (peritonitis, mortality) was noted. These maternally toxic doses were associated with dystocia, prolonged gestation, reduced fetal weights and growth, and reduced fetal survival. Diclofenac has been shown to cross the placental barrier in mice, rats, and humans.

Labor and Delivery

There are no studies on the effects of diclofenac during labor or delivery. In animal studies, NSAIDs, including diclofenac, inhibit prostaglandin synthesis, cause delayed parturition, and increase the incidence of stillbirth.

Nursing Mothers

Risk Summary

Based on available data, diclofenac may be present in human milk. The developmental and health benefits of breastfeeding should be considered along with the mother’s clinical need for diclofenac and any potential adverse effects on the breastfed infant from the diclofenac or from the underlying maternal condition.

Data

One woman treated orally with a diclofenac salt, 150 mg/day, had a milk diclofenac level of 100 mcg/L, equivalent to an infant dose of about 0.03 mg/kg/ day. Diclofenac was not detectable in breast milk in 12 women using diclofenac (after either 100 mg/day orally for 7 days or a single 50 mg intramuscular dose administered in the immediate postpartum period).

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

Elderly patients, compared to younger patients, are at greater risk for NSAIDassociated serious cardiovascular, gastrointestinal, and/or renal adverse reactions. If the anticipated benefit for the elderly patient outweighs these potential risks, start dosing at the low end of the dosing range, and monitor patients for adverse effects (see WARNINGS; Cardiovascular Thrombotic Events, Gastrointestinal Bleeding, Ulceration, and Perforation, Hepatotoxicity, Renal Toxicity and Hyperkalemia, PRECAUTIONS; Laboratory Monitoring).

Diclofenac is known to be substantially excreted by the kidney, and the risk of adverse reactions to this drug may be greater in patients with impaired renal function. Because elderly patients are more likely to have decreased renal function, care should be taken in dose selection, and it may be useful to monitor renal function (See CLINICAL PHARMACOLOGY, ADVERSE REACTIONS).

ADVERSE REACTIONS

The following adverse reactions are discussed in greater detail in other sections of the labeling:

- Cardiovascular Thrombotic Events (see WARNINGS)
- GI Bleeding, Ulceration and Perforation (see WARNINGS)
- Hepatotoxicity (see WARNINGS)
- Hypertension (see WARNINGS)
- Heart Failure and Edema (see WARNINGS)
- Renal Toxicity and Hyperkalemia (see WARNINGS)
- Anaphylactic Reactions (see WARNINGS)
- Serious Skin Reactions (see WARNINGS)
- Hematologic Toxicity (see WARNINGS)

Clinical Trials Experience

Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed

in practice.

In patients taking diclofenac sodium delayed-release tablets, or other NSAIDs, the most frequently reported adverse experiences occurring in approximately 1%-10% of patients are:

Gastrointestinal experiences including: abdominal pain, constipation, diarrhea, dyspepsia, flatulence, gross bleeding/perforation, heartburn, nausea, GI ulcers (gastric/duodenal) and vomiting.

Abnormal renal function, anemia, dizziness, edema, elevated liver enzymes, headaches, increased bleeding time, pruritus, rashes and tinnitus. Additional adverse experiences reported occasionally include:

Body as a Whole: fever, infection, sepsis

Cardiovascular System: congestive heart failure, hypertension, tachycardia, syncope

Digestive System: dry mouth, esophagitis, gastric/peptic ulcers, gastritis, gastrointestinal bleeding, glossitis, hematemesis, hepatitis, jaundice

Hemic and Lymphatic System: ecchymosis, eosinophilia, leukopenia, melena, purpura, rectal bleeding, stomatitis, thrombocytopenia

Metabolic and Nutritional: weight changes

Nervous System: anxiety, asthenia, confusion, depression, dream abnormalities, drowsiness, insomnia, malaise, nervousness, paresthesia, somnolence, tremors, vertigo

Respiratory System: asthma, dyspnea

Skin and Appendages: alopecia, photosensitivity, sweating increased

Special Senses: blurred vision

Urogenital System: cystitis, dysuria, hematuria, interstitial nephritis, oliguria/ polyuria, proteinuria, renal failure Other adverse reactions, which occur rarely are:

Body as a Whole: anaphylactic reactions, appetite changes, death Cardiovascular System: arrhythmia, hypotension, myocardial infarction, palpitations, vasculitis

Digestive System: colitis, eructation, fulminant hepatitis with and without jaundice, liver failure, liver necrosis, pancreatitis

Hemic and Lymphatic System: agranulocytosis, hemolytic anemia, aplastic anemia, lymphadenopathy, pancytopenia

Metabolic and Nutritional: hyperglycemia

Nervous System: convulsions, coma, hallucinations, meningitis

Respiratory System: respiratory depression, pneumonia

Skin and appendages: Angioedema, toxic epidermal necrolysis, erythema multiforme, exfoliative dermatitis, Stevens-Johnson syndrome, fixed drug eruption (FDE), urticaria

Special Senses: conjunctivitis, hearing impairment

OVERDOSAGE

Symptoms following acute NSAID overdosages have been typically limited to lethargy, drowsiness, nausea, vomiting, and epigastric pain, which have been generally reversible with supportive care. Gastrointestinal bleeding has occurred. Hypertension, acute renal failure, respiratory depression and coma have occurred, but were rare. (see WARNINGS; Cardiovascular Thrombotic Events, Gastrointestinal Bleeding, Ulceration, and Perforation, Hypertension, Renal Toxicity and Hyperkalemia) .

Manage patients with symptomatic and supportive care following an NSAID overdose. There are no specific antidotes. Consider emesis and/or activated charcoal (60 to 100 grams in adults, 1 to 2 grams per kg of body weight in pediatric patients) and/or osmotic cathartic in symptomatic patients seen within four hours of ingestion or in patients with a large overdose (5 to 10 times the recommended dosage). Forced diuresis, alkalization of urine, hemodialysis, or hemoperfusion may not be useful due to high protein binding.

For additional information about overdose treatment contact a poison control center (1-800-222-1222).

DOSAGE AND ADMINISTRATION

Carefully consider the potential benefits and risks of diclofenac sodium delayed-release tablets and other treatment options before deciding to use diclofenac. Use the lowest effective dose for the shortest duration consistent with individual patient treatment goals (see WARNINGS; Gastrointestinal Bleeding, Ulceration, and Perforation).

After observing the response to initial therapy with diclofenac, the dose and frequency should be adjusted to suit an individual patient’s needs.

For the relief of osteoarthritis, the recommended dosage is 100-150 mg/day in divided doses (50 mg twice a day or three times a day, or 75 mg twice a day).

For the relief of rheumatoid arthritis, the recommended dosage is 150-200 mg/day in divided doses (50 mg three times a day or four times a day, or 75 mg twice a day).

For the relief of ankylosing spondylitis, the recommended dosage is 100-125 mg/day, administered as 25 mg four times a day, with an extra 25-mg dose at bedtime if necessary.

HOW SUPPLIED

Diclofenac sodium delayed-release tablets

25 mg – white to off-white, biconvex, round-shaped, unscored (imprinted with “25” on one side and blank on the other side), supplied in bottles of 60, 100 and 1000.

Bottles of 60NDC 61442-101-60
Bottles of 100NDC 61442-101-01
Bottles of 1000NDC 61442-101-10

50 mg – white to off-white, biconvex, round-shaped, unscored with “5” engraved on one side and blank on the other side, supplied in bottles of 60, 100 and 1000

Bottles of 60NDC 61442-226-60
Bottles of 100NDC 61442-226-01
Bottles of 1000NDC 61442-226-10

75 mg – white to off-white, biconvex, round-shaped, unscored with “7” engraved on one side and blank on the other side, supplied in bottles of 60, 100, 500 and 1000.

Bottles of 60NDC 61442-227-60
Bottles of 100NDC 61442-227-01
Bottles of 500NDC 61442-227-05

Bottles of 1000NDC 61442-227-10
Store at room temperature 20°C to 25°C (68°F to 77°F); excursions permitted between 15°C to 30°C (59°F to 86°F) [see USP Controlled Room Temperature]. Protect from moisture.

Dispense in tight container (USP).

Manufactured and Distributed by:

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