

AMYTAL SODIUM- amobarbital sodium injection, powder, lyophilized, for solution

Bausch Health US, LLC

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**AMYTAL[®] SODIUM CII
(amobarbital sodium)
FOR INJECTION, USP
Vials**

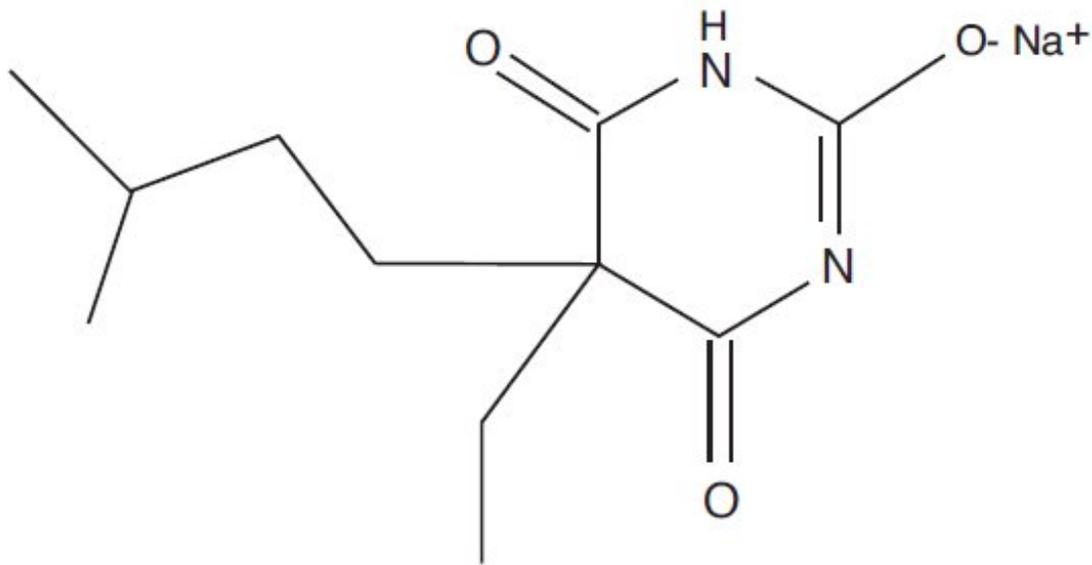
CAUTION: These products are to be used under the direction of a physician. The intravenous administration of AMYTAL[®] SODIUM (amobarbital sodium) FOR INJECTION, USP carries with it the potential dangers inherent in the intravenous use of any potent hypnotic.

DESCRIPTION

The barbiturates are nonselective central nervous system (CNS) depressants that are primarily used as sedative hypnotics. In subhypnotic doses, they are also used as anticonvulsants. The barbiturates and their sodium salts are subject to control under the Federal Controlled Substances Act.

Amobarbital sodium is a white, friable, granular powder that is odorless, has a bitter taste, and is hygroscopic. It is very soluble in water, soluble in alcohol, and practically insoluble in ether and chloroform. Amobarbital sodium is sodium 5-ethyl-5-isopentylbarbiturate and has the empirical formula $C_{11}H_{17}N_2NaO_3$. Its molecular weight is 248.26.

It has the following structural formula:



Amobarbital sodium is a substituted pyrimidine derivative in which the basic structure is barbituric acid, a substance that has no CNS activity.

Vials of amobarbital sodium are for parenteral administration. The vials contain 500 mg (2 mmol) amobarbital sodium as a sterile lyophilized powder.

CLINICAL PHARMACOLOGY

Barbiturates are capable of producing all levels of CNS mood alteration, from excitation to mild sedation, hypnosis, and deep coma. Overdosage can produce death. In high enough therapeutic doses, barbiturates induce anesthesia.

Barbiturates depress the sensory cortex, decrease motor activity, alter cerebellar function, and produce drowsiness, sedation, and hypnosis.

Barbiturate-induced sleep differs from physiologic sleep. Sleep laboratory studies have demonstrated that barbiturates reduce the amount of time spent in the rapid eye movement (REM) phase of sleep or the dreaming stage. Also, Stages III and IV sleep are decreased. Following abrupt cessation of barbiturates used regularly, patients may experience markedly increased dreaming, nightmares, and/or insomnia. Therefore, withdrawal of a single therapeutic dose over 5 or 6 days has been recommended to lessen the REM rebound and disturbed sleep that contribute to the drug withdrawal syndrome (for example, the dose should be decreased from 3 to 2 doses/day for 1 week).

In studies, secobarbital sodium and pentobarbital sodium have been found to lose most of their effectiveness for both inducing and maintaining sleep by the end of 2 weeks of continued drug administration, even with the use of multiple doses. As with secobarbital sodium and pentobarbital sodium, other barbiturates (including amobarbital) might be expected to lose their effectiveness for inducing and maintaining sleep after about 2 weeks. The short-, intermediate-, and to a lesser degree, long-acting barbiturates have been widely prescribed for treating insomnia. Although the clinical literature abounds with claims that the short-acting barbiturates are superior for producing sleep whereas the intermediate-acting compounds are more effective in maintaining sleep, controlled studies have failed to demonstrate these differential effects. Therefore, as sleep

medications, the barbiturates are of limited value beyond short-term use.

Barbiturates have little analgesic action at subanesthetic doses. Rather, in subanesthetic doses, these drugs may increase the reaction to painful stimuli. All barbiturates exhibit anticonvulsant activity in anesthetic doses. However, of the drugs in this class, only phenobarbital, mephobarbital, and metharbital are effective as oral anticonvulsants in subhypnotic doses.

Barbiturates are respiratory depressants, and the degree of respiratory depression is dependent upon the dose. With hypnotic doses, respiratory depression produced by barbiturates is similar to that which occurs during physiologic sleep and is accompanied by a slight decrease in blood pressure and heart rate.

Studies in laboratory animals have shown that barbiturates cause reduction in the tone and contractility of the uterus, ureters, and urinary bladder. However, concentrations of the drugs required to produce this effect in humans are not reached with sedative-hypnotic doses.

Barbiturates do not impair normal hepatic function but have been shown to induce liver microsomal enzymes, thus increasing and/or altering the metabolism of barbiturates and other drugs (see *PRECAUTIONS, Drug Interactions*).

Pharmacokinetics-

Barbiturates are absorbed in varying degrees following oral or parenteral administration. The salts are more rapidly absorbed than are the acids. The rate of absorption is increased if the sodium salt is ingested as a dilute solution or taken on an empty stomach.

The onset of action for oral administration of barbiturates varies from 20 to 60 minutes. For intramuscular (IM) administration, the onset of action is slightly faster. Following intravenous (IV) administration, the onset of action ranges from almost immediately for pentobarbital sodium to 5 minutes for phenobarbital sodium. Maximal CNS depression may not occur until 15 minutes or more after IV administration for phenobarbital sodium. Duration of action, which is related to the rate at which the barbiturates are redistributed throughout the body, varies among persons and in the same person from time to time. Amobarbital sodium, an intermediate-acting barbiturate, is a CNS depressant. For the oral form, the onset of sedative and hypnotic action is 3/4 to 1 hour, with a duration of action ranging from 6 to 8 hours. These values should serve as a guide but not be used to predict exact duration of effect. No studies have demonstrated that the different routes of administration are equivalent with respect to bioavailability.

Barbiturates are weak acids that are absorbed and rapidly distributed to all tissues and fluids, with high concentrations in the brain, liver, and kidneys. Lipid solubility of the barbiturates is the dominant factor in their distribution within the body. The more lipid soluble the barbiturate, the more rapidly it penetrates all tissues of the body. Barbiturates are bound to plasma and tissue proteins to a varying degree, with the degree of binding increasing directly as a function of lipid solubility.

Phenobarbital has the lowest lipid solubility, lowest plasma binding, lowest brain protein binding, the longest delay in onset of activity, and the longest duration of action. At the opposite extreme is secobarbital, which has the highest lipid solubility, highest plasma protein binding, highest brain protein binding, the shortest delay in onset of activity, and

the shortest duration of action. Amobarbital sodium is classified as an intermediate barbiturate. The plasma half-life for amobarbital sodium in adults ranges between 16 and 40 hours, with a mean of 25 hours.

Barbiturates are metabolized primarily by the hepatic microsomal enzyme system, and the metabolic products are excreted in the urine and, less commonly, in the feces. Only a negligible amount of amobarbital sodium is eliminated unchanged in the urine.

INDICATIONS AND USAGE

- A. Sedative
- B. Hypnotic, for the short-term treatment of insomnia, since it appears to lose its effectiveness for sleep induction and sleep maintenance after 2 weeks (see CLINICAL PHARMACOLOGY).
- C. Preanesthetic

CONTRAINDICATIONS

Amobarbital sodium is contraindicated in patients who are hypersensitive to barbiturates, in patients with a history of manifest or latent porphyria, and in patients with marked impairment of liver function or respiratory disease in which dyspnea or obstruction is evident.

WARNINGS

1. *Habit Forming* — Amobarbital sodium may be habit forming. Tolerance, psychological and physical dependence may occur with continued use (see CLINICAL PHARMACOLOGY, Pharmacokinetics and DRUG ABUSE AND DEPENDENCE). Patients who have psychological dependence on barbiturates may increase the dosage or decrease the dosage interval without consulting a physician and may subsequently develop a physical dependence on barbiturates. In order to minimize the possibility of overdosage or the development of dependence, the prescribing and dispensing of sedative-hypnotic barbiturates should be limited to the amount required for the interval until the next appointment. Abrupt cessation after prolonged use in a person who is dependent on the drug may result in withdrawal symptoms, including delirium, convulsions, and possibly death. Barbiturates should be withdrawn gradually from any patient known to be taking excessive doses over long periods of time (see DRUG ABUSE AND DEPENDENCE).
2. *Intravenous Administration* — Too rapid administration may cause respiratory depression, apnea, laryngospasm, or vasodilation with fall in blood pressure.
3. *Acute or Chronic Pain* — Caution should be exercised when barbiturates are administered to patients with acute or chronic pain because paradoxical excitement could be induced or important symptoms could be masked. However, the use of barbiturates as sedatives in the postoperative surgical period and as adjuncts to cancer chemotherapy is well established.
4. *Usage in Pregnancy* — Barbiturates can cause fetal damage when administered to a pregnant woman. Retrospective, case-controlled studies have suggested a connection between the maternal consumption of barbiturates and a higher than expected incidence of fetal abnormalities. Barbiturates readily cross the placental

barrier and are distributed throughout fetal tissues; the highest concentrations are found in the placenta, fetal liver, and brain. Fetal blood levels approach maternal blood levels following parenteral administration. Withdrawal symptoms occur in infants born to women who receive barbiturates throughout the last trimester of pregnancy (see DRUG ABUSE AND DEPENDENCE). If amobarbital sodium is used during pregnancy or if the patient becomes pregnant while taking this drug, the patient should be apprised of the potential hazard to the fetus.

5. *Synergistic Effects* — The concomitant use of alcohol or other CNS depressants may produce additive CNS depressant effects.

PRECAUTIONS

General-

Barbiturates may be habit forming. Tolerance and psychological and physical dependence may occur with continuing use (see DRUG ABUSE AND DEPENDENCE).

Barbiturates should be administered with caution, if at all, to patients who are mentally depressed, have suicidal tendencies, or have a history of drug abuse. Particular caution is also indicated before administering barbiturates to patients who have abused other classes of drugs (see WARNINGS).

Elderly or debilitated patients may react to barbiturates with marked excitement, depression, or confusion. In some persons, especially children, barbiturates repeatedly produce excitement rather than depression.

In patients with hepatic damage, barbiturates should be administered with caution and initially in reduced doses. Barbiturates should not be administered to patients showing the premonitory signs of hepatic coma.

Parenteral solutions of barbiturates are highly alkaline. Therefore, extreme care should be taken to avoid perivascular extravasation or intra-arterial injection. Extravascular injection may cause local tissue damage with subsequent necrosis; consequences of intra-arterial injection may vary from transient pain to gangrene of the limb. Any complaint of pain in the limb warrants stopping the injection.

The systemic effects of exogenous and endogenous corticosteroids may be diminished by amobarbital sodium. Thus, this product should be administered with caution to patients with borderline hypoadrenal function, regardless of whether it is of pituitary or of primary adrenal origin.

Information for Patients-

The following information should be given to patients receiving barbiturates:

1. The use of barbiturates carries with it an associated risk of psychological and/or physical dependence.
2. Barbiturates may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as driving a car or operating machinery. The patient should be cautioned accordingly.
3. Alcohol should not be consumed while taking barbiturates. The concurrent use of the barbiturates with other CNS depressants (e.g., alcohol, narcotics, tranquilizers, and antihistamines) may result in additional CNS depressant effects.

Laboratory Tests-

Prolonged therapy with barbiturates should be accompanied by periodic evaluation of organ systems, including hematopoietic, renal, and hepatic systems (see *PRECAUTIONS, General* and *ADVERSE REACTIONS*).

Drug Interactions-

Most reports of clinically significant drug interactions occurring with the barbiturates have involved phenobarbital. However, the application of these data to other barbiturates appears valid and warrants serial blood level determinations of the relevant drugs when there are multiple therapies.

1. *Anticoagulants* — Phenobarbital lowers the plasma levels of dicumarol and causes a decrease in anticoagulant activity as measured by the prothrombin time. Barbiturates can induce hepatic microsomal enzymes, resulting in increased metabolism and decreased anticoagulant response of oral anticoagulants (e.g., warfarin, acenocoumarol, dicumarol, and phenprocoumon). Patients stabilized on anticoagulant therapy may require dosage adjustments if barbiturates are added to or withdrawn from their dosage regimen.
2. *Corticosteroids* — Barbiturates appear to enhance the metabolism of exogenous corticosteroids, probably through the induction of hepatic microsomal enzymes. Patients stabilized on corticosteroid therapy may require dosage adjustments if barbiturates are added to or withdrawn from their dosage regimen.
3. *Griseofulvin* — Phenobarbital appears to interfere with the absorption of orally administered griseofulvin, thus decreasing its blood level. The effect of the resultant decreased blood levels of griseofulvin on therapeutic response has not been established. However, it would be preferable to avoid concomitant administration of these drugs.
4. *Doxycycline* — Phenobarbital has been shown to shorten the half-life of doxycycline for as long as 2 weeks after barbiturate therapy is discontinued. This mechanism is probably through the induction of hepatic microsomal enzymes that metabolize the antibiotic. If amobarbital sodium and doxycycline are administered concurrently, the clinical response to doxycycline should be monitored closely.
5. *Phenytoin, Sodium Valproate, Valproic Acid* — The effect of barbiturates on the metabolism of phenytoin appears to be variable. Some investigators report an accelerating effect, whereas others report no effect. Because the effect of barbiturates on the metabolism of phenytoin is not predictable, phenytoin and barbiturate blood levels should be monitored more frequently if these drugs are given concurrently. Sodium valproate and valproic acid appear to increase the amobarbital sodium serum levels; therefore, amobarbital sodium blood levels should be closely monitored and appropriate dosage adjustments made as clinically indicated.
6. *CNS Depressants* — The concomitant use of other CNS depressants, including other sedatives or hypnotics, antihistamines, tranquilizers, or alcohol, may produce additive depressant effects.
7. *Monoamine Oxidase Inhibitors (MAOIs)* — MAOIs prolong the effects of barbiturates, probably because metabolism of the barbiturate is inhibited.
8. *Estradiol, Estrone, Progesterone, and Other Steroidal Hormones* — Pretreatment with or concurrent administration of phenobarbital may decrease the effect of

estradiol by increasing its metabolism. There have been reports of patients treated with antiepileptic drugs (e.g., phenobarbital) who become pregnant while taking oral contraceptives. An alternate contraceptive method might be suggested to women taking barbiturates.

Carcinogenesis-

1. *Animal Data* - Phenobarbital sodium is carcinogenic in mice and rats after lifetime administration. In mice, it produced benign and malignant liver cell tumors. In rats, benign liver cell tumors were observed very late in life.
2. *Human Data* — In a 29-year epidemiologic study of 9,136 patients who were treated on an anticonvulsant protocol that included phenobarbital, results indicated a higher than normal incidence of hepatic carcinoma. Previously, some of these patients had been treated with thorotrast, a drug that is known to produce hepatic carcinomas. Thus, this study did not provide sufficient evidence that phenobarbital sodium is carcinogenic in humans.

A retrospective study of 84 children with brain tumors matched to 73 normal controls and 78 cancer controls (malignant disease other than brain tumors) suggested an association between exposure to barbiturates prenatally and an increased incidence of brain tumors.

Usage in Pregnancy-

1. *Teratogenic Effects* - (See *WARNINGS, Usage in Pregnancy.*)
2. *Nonteratogenic Effects* — Reports of infants suffering from long-term barbiturate exposure in utero included the acute withdrawal syndrome of seizures and hyperirritability from birth to a delayed onset of up to 14 days (see *DRUG ABUSE AND DEPENDENCE*).

Labor and Delivery-

Hypnotic doses of barbiturates do not appear to impair uterine activity significantly during labor. Full anesthetic doses of barbiturates decrease the force and frequency of uterine contractions. Administration of sedative-hypnotic barbiturates to the mother during labor may result in respiratory depression in the newborn. Premature infants are particularly susceptible to the depressant effects of barbiturates. If barbiturates are used during labor and delivery, resuscitation equipment should be available.

Data are not available to evaluate the effect of barbiturates when forceps delivery or other intervention is necessary or to determine the effect of barbiturates on the later growth, development, and functional maturation of the child.

Nursing Mothers-

Caution should be exercised when amobarbital sodium is administered to a nursing woman because small amounts of barbiturates are excreted in the milk.

Usage in Children-

Safety and effectiveness have not been established in children below the age of 6 years.

ADVERSE REACTIONS

The following adverse reactions and their incidence were compiled from surveillance of thousands of hospitalized patients who received barbiturates. Because such patients may be less aware of certain of the milder adverse effects of barbiturates, the incidence of these reactions may be somewhat higher in fully ambulatory patients.

More than 1 in 100 Patients

The most common adverse reaction, estimated to occur at a rate of 1 to 3 patients per 100, is the following:

Nervous System: Somnolence

Less than 1 in 100 Patients

Adverse reactions estimated to occur at a rate of less than 1 in 100 patients are listed below, grouped by organ system and by decreasing order of occurrence:

Nervous System: Agitation, confusion, hyperkinesia, ataxia, CNS depression, nightmares, nervousness, psychiatric disturbance, hallucinations, insomnia, anxiety, dizziness, abnormality in thinking

Respiratory System: Hypoventilation, apnea, postoperative atelectasis

Cardiovascular System: Bradycardia, hypotension, syncope

Digestive System: Nausea, vomiting, constipation

Other Reported Reactions: Headache, injection site reactions, hypersensitivity reactions (angioedema, skin rashes, exfoliative dermatitis), fever, liver damage, megaloblastic anemia following chronic phenobarbital use

To report SUSPECTED ADVERSE REACTIONS, contact Bausch Health US, LLC at 1-800-321-4576 or FDA at 1-800-FDA-1088 or www.fda.gov/medwatch.

DRUG ABUSE AND DEPENDENCE

Controlled Substance

Amobarbital sodium is a Schedule II drug.

Dependence-

Barbiturates may be habit-forming. Tolerance, psychological dependence, and physical dependence may occur, especially following prolonged use of high doses of barbiturates. Daily administration in excess of 400 mg of pentobarbital or secobarbital for approximately 90 days is likely to produce some degree of physical dependence. A dosage of 600 to 800 mg for at least 35 days is sufficient to produce withdrawal seizures. The average daily dose for the barbiturate addict is usually about 1.5 g. As tolerance to barbiturates develops, the amount needed to maintain the same level of intoxication increases; tolerance to a fatal dosage, however, does not increase more than twofold. As this occurs, the margin between intoxicating dosage and fatal dosage becomes smaller.

Symptoms of acute intoxication with barbiturates include unsteady gait, slurred speech, and sustained nystagmus. Mental signs of chronic intoxication include confusion, poor judgment, irritability, insomnia, and somatic complaints.

Symptoms of barbiturate dependence are similar to those of chronic alcoholism. If an

individual appears to be intoxicated with alcohol to a degree that is radically disproportionate to the amount of alcohol in his or her blood, the use of barbiturates should be suspected. The lethal dose of a barbiturate is far less if alcohol is also ingested.

The symptoms of barbiturate withdrawal can be severe and may cause death. Minor withdrawal symptoms may appear 8 to 12 hours after the last dose of a barbiturate. These symptoms usually appear in the following order: anxiety, muscle twitching, tremor of hands and fingers, progressive weakness, dizziness, distortion in visual perception, nausea, vomiting, insomnia, and orthostatic hypotension. Major withdrawal symptoms (i.e., convulsions and delirium) may occur within 16 hours and last up to 5 days after abrupt cessation of barbiturates. The intensity of withdrawal symptoms gradually declines over a period of approximately 15 days. Individuals susceptible to barbiturate abuse and dependence include alcoholics and opiate abusers, as well as other sedative-hypnotic and amphetamine abusers.

Drug dependence on barbiturates arises from repeated administration on a continuous basis, generally in amounts exceeding therapeutic dose levels. The characteristics of drug dependence on barbiturates include: (a) a strong desire or need to continue taking the drug; (b) a tendency to increase the dose; (c) a psychic dependence on the effects of the drug related to subjective and individual appreciation of those effects; and (d) a physical dependence on the effects of the drug, requiring its presence for maintenance of homeostasis and resulting in a definite, characteristic, and self-limited abstinence syndrome when the drug is withdrawn.

Treatment of barbiturate dependence consists of cautious and gradual withdrawal of the drug. Barbiturate-dependent patients can be withdrawn by using a number of different withdrawal regimens. In all cases, withdrawal requires an extended period of time. One method involves substituting a 30 mg dose of phenobarbital for each 100 to 200 mg dose of barbiturate that the patient has been taking. The total daily amount of phenobarbital is then administered in 3 or 4 divided doses, not to exceed 600 mg daily. If signs of withdrawal occur on the first day of treatment, a loading dose of 100 to 200 mg of phenobarbital may be administered intramuscularly in addition to the oral dose. After stabilization on phenobarbital, the total daily dose is decreased by 30 mg/day as long as withdrawal is proceeding smoothly. A modification of this regimen involves initiating treatment at the patient's regular dosage level and decreasing the daily dosage by 10% if tolerated by the patient.

Infants that are physically dependent on barbiturates may be given phenobarbital, 3 to 10 mg/kg/day. After withdrawal symptoms (e.g., hyperactivity, disturbed sleep, tremors, and hyperreflexia) are relieved, the dosage of phenobarbital should be gradually decreased and completely withdrawn over a 2-week period.

OVERDOSAGE

The toxic dose of barbiturates varies considerably. In general, an oral dose of 1 g of most barbiturates produces serious poisoning in an adult. Toxic effects and fatalities have occurred following overdoses of amobarbital sodium alone and in combination with other CNS depressants. Death commonly occurs after 2 to 10 g of ingested barbiturate. The sedated, therapeutic blood levels of amobarbital range between 2 to 10 mcg/mL; the usual lethal blood level ranges from 40 to 80 mcg/mL. Barbiturate intoxication may be confused with alcoholism, bromide intoxication, and various neurologic disorders.

Potential tolerance must be considered when evaluating significance of dose and plasma concentration.

Signs and Symptoms-

Symptoms of oral overdose may occur within 15 minutes beginning with CNS depression, absent or sluggish reflexes, underventilation, hypotension, and hypothermia and may progress to pulmonary edema and death. Hemorrhagic blisters may develop, especially at pressure points.

In extreme overdose, all electrical activity in the brain may cease, in which case a “flat” EEG normally equated with clinical death cannot be accepted. This effect is fully reversible unless hypoxic damage occurs. Consideration should be given to the possibility of barbiturate intoxication even in situations that appear to involve trauma.

Complications such as pneumonia, pulmonary edema, cardiac arrhythmias, congestive heart failure, and renal failure may occur. Uremia may increase CNS sensitivity to barbiturates if renal function is impaired. Differential diagnosis should include hypoglycemia, head trauma, cerebrovascular accidents, convulsive states, and diabetic coma.

Treatment-

To obtain up-to-date information about the treatment of overdose, a good resource is your certified Regional Poison Control Center. Telephone numbers of certified poison control centers are listed in the *Physicians' Desk Reference (PDR)**. In managing overdosage, consider the possibility of multiple drug overdoses, interaction among drugs, and unusual drug kinetics in your patient.

Protect the patient's airway and support ventilation and perfusion. Meticulously monitor and maintain, within acceptable limits, the patient's vital signs, blood gases, serum electrolytes, etc. Absorption of drugs from the gastrointestinal tract may be decreased by giving activated charcoal, which, in many cases, is more effective than emesis or lavage; consider charcoal instead of or in addition to gastric emptying. Repeated doses of charcoal over time may hasten elimination of some drugs that have been absorbed. Safeguard the patient's airway when employing gastric emptying or charcoal.

Diuresis and peritoneal dialysis are of little value; hemodialysis and hemoperfusion enhance drug clearance and should be considered in serious poisoning. If the patient has chronically abused sedatives, withdrawal reactions may be manifest following acute overdose.

PREPARATION OF SOLUTION

Solutions of amobarbital sodium should be made up aseptically with Sterile Water for Injection. The accompanying table will aid in preparing solutions of various concentrations. Ordinarily, a 10% solution is used. After Sterile Water for Injection is added, the vial should be rotated to facilitate solution of the powder. **Do not shake the vial.**

Several minutes may be required for the drug to dissolve completely, but under no circumstances should a solution be injected if it has not become absolutely clear within 5 minutes. Also, a solution that forms a precipitate after clearing should not be used.

Amobarbital sodium hydrolyzes in solution or on exposure to air. Not more than 30 minutes should elapse from the time the vial is opened until its contents are injected. Prior to administration, parenteral drug products should be inspected visually for particulate matter and discoloration whenever solution containers permit.

Quantity of Sterile Water for Injection Required to Dilute the Contents of a Given Vial of Amobarbital Sodium to Obtain the Percentages Listed. Solutions Derived Will Be in Weight/Volume.

AMOBARBITAL SODIUM					
Content in Weight	1%	2.5%	5%	10%	20%
0.5 g	50 mL	20 mL	10 mL	5 mL	2.5 mL

DOSAGE AND ADMINISTRATION

The dose of amobarbital sodium must be individualized with full knowledge of its particular characteristics and recommended rate of administration. Factors of consideration are the patient's age, weight, and condition. The maximum single dose for an adult is 1 g.

Intramuscular Use-

Intramuscular injection of the sodium salts of barbiturates should be made deeply into a large muscle. The average IM dose ranges from 65 mg to 0.5 g. A volume of 5 mL (irrespective of concentration) should not be exceeded at any one site because of possible tissue irritation. Twenty percent solutions may be used so that a small volume can contain a large dose. After IM injection of a hypnotic dose, the patient's vital signs should be monitored. Superficial IM or subcutaneous injections may be painful and may produce sterile abscesses or sloughs.

Intravenous Use-

Intravenous (IV) injection is restricted to conditions in which other routes are not feasible, either because the patient is unconscious (as in cerebral hemorrhage, eclampsia, or status epilepticus), because the patient resists (as in delirium), or because prompt action is imperative. Slow IV injection is essential, and patients should be carefully observed during administration. This requires that blood pressure, respiration, and cardiac function be maintained, vital signs be recorded and equipment for resuscitation and artificial ventilation be available. The rate of IV injection for adults should not exceed 50 mg/min to prevent sleep or sudden respiratory depression. The final dosage is determined to a great extent by the patient's reaction to the slow administration of the drug.

Adults:

- a. Sedative: 30 to 50 mg given 2 or 3 times daily.
- b. Hypnotic: 65 to 200 mg at bedtime.

Special Patient Population-

Dosage should be reduced in the elderly or debilitated because these patients may be

more sensitive to barbiturates. Dosage should be reduced for patients with impaired renal function or hepatic disease. Ordinarily, an IV dose of 65 mg to 0.5 g may be given to a child 6 to 12 years of age.

HOW SUPPLIED

AMYTAL® SODIUM Vials 0.5 g (dry powder) are available as follows:

NDC 0187-4303-05

Storage: Store at (15° to 30°C) (59° to 86°F)

Lyophilized

*Trademark of PDR Network, LLC

Distributed by:

Bausch Health US, LLC

Bridgewater, NJ 08807 USA

Manufactured by:

Alcami Carolinas Corporation

North Charleston, SC 29405 USA

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Revised: July 2020

9476203 PC3328F

PRINCIPAL DISPLAY PANEL

NDC 0187-4303-05

1 Vial

**AMYTAL® SODIUM CII
(amobarbital sodium)
FOR INJECTION, USP**

0.5 g/vial

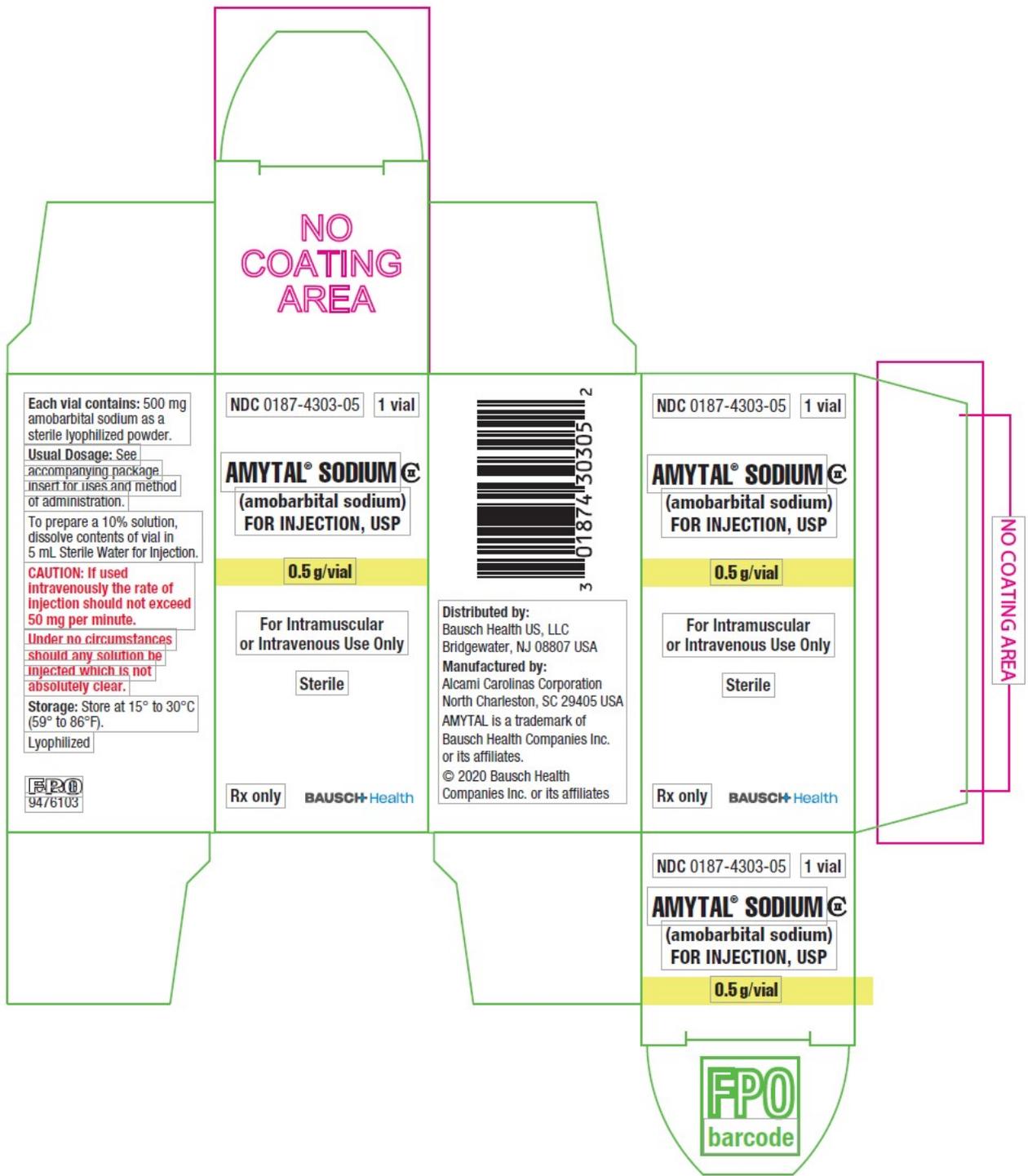
**For Intramuscular
or Intravenous Use Only**

Sterile

Rx only

Bausch Health

9476103



AMYTAL SODIUM

amobarbital sodium injection, powder, lyophilized, for solution

Product Information

Product Type

HUMAN PRESCRIPTION DRUG

Item Code (Source)

NDC:0187-4303

Route of Administration

INTRAMUSCULAR,
 INTRAVENOUS

DEA Schedule

CII

Active Ingredient/Active Moiety

Ingredient Name	Basis of Strength	Strength
AMOBARBITAL SODIUM (UNII: G0313KNC7D) (AMOBARBITAL - UNII:GWH6IJ239E)	AMOBARBITAL SODIUM	0.5 g in 5 mL

Packaging

#	Item Code	Package Description	Marketing Start Date	Marketing End Date
1	NDC:0187-4303-05	1 in 1 CARTON	09/25/2008	
1		5 mL in 1 VIAL, GLASS; Type 0: Not a Combination Product		

Marketing Information

Marketing Category	Application Number or Monograph Citation	Marketing Start Date	Marketing End Date
Unapproved drug other		09/25/2008	

Labeler - Bausch Health US, LLC (831922468)**Establishment**

Name	Address	ID/FEI	Business Operations
Alcami Carolinas Corporation		832394733	MANUFACTURE(0187-4303)

Revised: 7/2020

Bausch Health US, LLC