

Overview of amytal: pharmacology, action and pharmacokinetics

[Health & Medicine](#)



**ASSIGN
BUSTER**

This barbiturate is a nonselective central nervous system (CNS) depressants that are primarily used as sedative hypnotics. In sub hypnotic doses, they are also used as an anticonvulsant.

Chemical Formulae: C₁₁H₁₈N₂O₃.

Pharmacology

Amobarbital (5-ethyl-5-isoamylbarbituric acid), like all barbiturates, is synthesized by reacting malonic acid derivatives with urea derivatives. In order to make amobarbital, α -ethyl- α -isoamylmalonic ester is reacted with urea (in the presence of sodium ethoxide). In an in vitro study in rat thalamic slices amobarbital worked by activating GABAA receptors, which decreased input resistance, depressed burst and tonic firing, especially in ventrobasal and intralaminar neurons, while at the same time increasing burst duration and mean conductance at individual chloride channels; this increased both the amplitude and decay time of inhibitory postsynaptic currents.

Amobarbital has been used in a study to inhibit mitochondrial electron transport in the rat heart in an attempt to preserve mitochondrial function following reperfusion.

Mechanism of Action

A 2010 Medscape study illustrates that “ Amytal depresses the cortex by interfering with transmission of impulses from thalamus. This results in decreased motor activity, altered cerebellar function, drowsiness, sedation and hypnosis. The onset of action is within 5 minutes and it is metabolized primarily hepatic via microsomal enzyme. ”

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.

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.