[thumb|](/wiki/File:Kwas_barbiturowy.svg" \o "File:Kwas barbiturowy.svg)[Barbituric acid](/wiki/Barbituric_acid), the basic structure of all barbiturates **Barbiturates** are [drugs](/wiki/Pharmaceutical_drug) that act as [central nervous system](/wiki/Central_nervous_system) [depressants](/wiki/Depressant), and can therefore produce a wide spectrum of effects, from mild [sedation](/wiki/Sedation) to total [anesthesia](/wiki/Anesthesia). They are also effective as [anxiolytics](/wiki/Anxiolytic), [hypnotics](/wiki/Hypnotic), and [anticonvulsants](/wiki/Anticonvulsant). Barbiturates also have [analgesic](/wiki/Analgesic) effects; however, these effects are somewhat weak, preventing barbiturates from being used in [surgery](/wiki/Surgery) in the presence of other analgesics ([opioids](/wiki/Opioid) or volatile anesthetics such as [halothane](/wiki/Halothane)).

Barbiturates have [addiction](/wiki/Addiction) potential, both physical and psychological. They have largely been replaced by [benzodiazepines](/wiki/Benzodiazepine) in routine medical practice – for example, in the treatment of anxiety and insomnia – mainly because benzodiazepines are significantly less dangerous in [overdose](/wiki/Drug_overdose) and there is no specific [antidote](/wiki/Antidote) for barbiturate overdose. However, barbiturates are still used in general anesthesia, for [epilepsy](/wiki/Epilepsy), for the treatment of acute migraines and [cluster headaches](/wiki/Cluster_headaches) (in the compound drugs [Fioricet](/wiki/Fioricet) and [Fiorinal](/wiki/Fiorinal)) (under stringent protocols with mandatory physician monitoring for addiction and abuse), and (where legal) assisted suicide, euthanasia, and capital punishment.[[1]](#cite_note-1) Barbiturates are derivatives of [barbituric acid](/wiki/Barbituric_acid).[[2]](#cite_note-2)

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## Medical uses[[edit](/index.php?title=(none)&action=edit&section=1)]

Barbiturates such as [phenobarbital](/wiki/Phenobarbital) were long used as [anxiolytics](/wiki/Anxiolytics) and [hypnotics](/wiki/Hypnotics), but today have been largely replaced by [benzodiazepines](/wiki/Benzodiazepines) for these purposes because of less potential for lethal [overdoses](/wiki/Drug_overdose).[[3]](#cite_note-3)[[4]](#cite_note-4)[[5]](#cite_note-5) However, barbiturates are still used as [anticonvulsants](/wiki/Anticonvulsants), as para-operative sedatives (ex. [sodium thiopental](/wiki/Sodium_thiopental)), and analgesics for cluster headaches/ migraines (ex. [Fioricet](/wiki/Fioricet)).

### Other uses related to their physiological properties[[edit](/index.php?title=(none)&action=edit&section=2)]

Barbiturates in high doses are used for [physician-assisted suicide](/wiki/Physician-assisted_suicide) (PAS), and in combination with a [muscle relaxant](/wiki/Muscle_relaxant) for [euthanasia](/wiki/Euthanasia) and for [capital punishment](/wiki/Capital_punishment) by [lethal injection](/wiki/Lethal_injection).[[6]](#cite_note-6)[[7]](#cite_note-7) Barbiturates are frequently employed as euthanizing agents in small animal [veterinary medicine](/wiki/Veterinary_medicine).

[Thiopental](/wiki/Thiopental) is an ultra-short acting barbiturate that is marketed under the name sodium pentothal. It is often mistaken for "truth serum" or sodium amytal, an intermediate-acting barbiturate that is used for sedation and to treat insomnia, but was also used in so-called sodium amytal "interviews" where the person being questioned would be much more likely to provide the truth whilst under the influence of this drug. When dissolved in water, sodium amytal can be swallowed, or it can be administered by intravenous injection. The drug does not itself force people to tell the truth, but is thought to decrease inhibitions and slow creative thinking, making subjects more likely to be caught off guard when questioned, and increasing the possibility of the subject revealing information through emotional outbursts.[[8]](#cite_note-8) The memory impairing effects and cognitive impairments induced by the drug are thought to reduce a subject's ability to invent and remember lies. This practice is no longer considered legally admissible in court due to findings that subjects undergoing such interrogations may form false memories, putting the reliability of all information obtained through such methods into question. Nonetheless, it is still employed in certain circumstances by defense and law enforcement agencies as a "humane" alternative to torture interrogation when the subject is believed to have information critical to the security of the state or agency employing the tactic.[[9]](#cite_note-9)

## Side effects[[edit](/index.php?title=(none)&action=edit&section=3)]

There are special risks to consider for older adults, women who are pregnant, and babies. When a person ages, the body becomes less able to rid itself of barbiturates. As a result, people over the age of sixty-five are at higher risk of experiencing the harmful effects of barbiturates, including drug dependence and accidental overdose.[[10]](#cite_note-10) When barbiturates are taken during pregnancy, the drug passes through the mother's bloodstream to her fetus. After the baby is born, it may experience withdrawal symptoms and have trouble breathing. In addition, nursing mothers who take barbiturates may transmit the drug to their babies through breast milk.[[11]](#cite_note-11) A rare adverse reaction to barbiturates is [Stevens-Johnson syndrome](/wiki/Stevens-Johnson_syndrome), which primarily affects the mucous membranes.

### Tolerance and dependence[[edit](/index.php?title=(none)&action=edit&section=4)]

[Template:Main](/wiki/Template:Main) With regular use, [tolerance](/wiki/Drug_tolerance) to the effects of barbiturates develops. As with all GABAergic drugs barbiturate withdrawal produces potentially fatal effects such as seizures in a manner reminiscent of [delerium tremens](/wiki/Delerium_tremens) and [benzodiazepine withdrawal](/wiki/Benzodiazepine_withdrawal) although its more direct mechanism of GABA agonism makes barbiturate withdrawal more severe than that of alcohol or benzodiazepines (subsequently making it one of the most dangerous withdrawals of any known addictive substance). Similar to benzodiazepines the longer acting barbiturates produce a less severe withdrawal syndrome than short acting and ultra short acting barbiturates. Withdrawal symptoms are dose-dependent with heavier users being affected worse than lower-dose addicts.

The pharmacological treatment of barbiturate withdrawal is an extended process often consisting of converting the patient to a long acting benzodiazepine (i.e. [Valium](/wiki/Valium)), followed by slowly tapering off the benzodiazepine. Mental cravings for barbiturates can last for months or years in some cases and counselling/support groups are highly encouraged by addiction specialists. Patients should never try to tackle the task of discontinuing barbiturates without consulting a doctor due to the high lethality and relatively sudden onset of the withdrawal, attempting to quit "cold turkey" may result in serious neurological damage, severe physical injuries received during convulsions, and even death via glutamatergic excitotoxicity.

### Overdose[[edit](/index.php?title=(none)&action=edit&section=5)]

[Template:Main](/wiki/Template:Main)

Some symptoms of an overdose typically include sluggishness, incoordination, difficulty in thinking, slowness of speech, faulty judgement, drowsiness, shallow breathing, staggering, and, in severe cases, coma or death. The lethal dosage of barbiturates varies greatly with tolerance and from one individual to another. The [lethal dose](/wiki/LD50) is highly variable among different members of the class with superpotent barbiturates such as pentobarbital being potentially fatal in considerably lower doses than the low-potency barbiturates such as butalbital. Even in inpatient settings, however, the development of tolerance is still a problem, as dangerous and unpleasant withdrawal symptoms can result when the drug is stopped after dependence has developed. Tolerance to the anxiolytic and sedative effects of barbiturates tends to develop faster than tolerance to their effects on smooth muscle, respiration, and heart rate, making them generally unsuitable for long time psychiatric use. Tolerance to the anticonvulsant effects tends to correlate more with tolerance to physiological effects, however, meaning that they are still a viable option for long-term epilepsy treatment.

Barbiturates in overdose with other CNS (central nervous system) depressants (e.g. alcohol, opiates, benzodiazepines) are even more dangerous due to additive CNS and respiratory depressant effects. In the case of benzodiazepines, not only do they have additive effects, barbiturates also increase the binding affinity of the benzodiazepine binding site, leading to exaggerated benzodiazepine effects. (ex. If a benzodiazepine increases the frequency of channel opening by 300%, and a barbiturate increases the duration of their opening by 300%, then the combined effects of the drugs increase the channels overall function by 900%, not 600%).

The longest-acting barbiturates have half-lives of a day or more, and subsequently result in [bioaccumulation](/wiki/Bioaccumulation) of the drug in the system. The therapeutic and recreational effects of long-acting barbiturates wear off significantly faster than the drug can be eliminated, allowing the drug to reach toxic concentrations in the blood following repeated administration (even when taken at the therapeutic/prescribed dose) despite the user feeling little or no effects from the plasma-bound concentrations of the drug. Users who consume alcohol or other sedatives after the drugs effects have worn but before it has cleared the system may experience a greatly exaggerated effect from the other sedatives which can be incapacitating or even fatal.

Barbiturates induce a number of hepatic [CYP](/wiki/Cytochrome_P450) enzymes (most notably [CYP2C9](/wiki/CYP2C9), [CYP2C19](/wiki/CYP2C19) and [CYP3A4](/wiki/CYP3A4)),[[12]](#cite_note-12) leading to exaggerated effects from many [prodrugs](/wiki/Prodrugs) and decreased effects from drugs which are metabolized by these enzymes to inactive metabolites. This can result in fatal overdoses from drugs such as [codeine](/wiki/Codeine), [tramadol](/wiki/Tramadol), and [carisoprodol](/wiki/Carisoprodol), which become considerably more potent after being metabolized by CYP enzymes. Although all known members of the class possess relevant enzyme induction capabilities the degree of inhibition overall as well as the impact on each specific enzyme span a broad range with phenobarbital and secobarbital being the most potent enzyme inducers and butalbital and talbutal being among the weakest enzyme inducers in the class.

Notably, [Judy Garland](/wiki/Judy_Garland), [Marilyn Monroe](/wiki/Marilyn_Monroe), [Dorothy Dandridge](/wiki/Dorothy_Dandridge), [Charles Boyer](/wiki/Charles_Boyer), [Ellen Wilkinson](/wiki/Ellen_Wilkinson), [Dalida](/wiki/Dalida), [Carole Landis](/wiki/Carole_Landis), [Dorothy Kilgallen](/wiki/Dorothy_Kilgallen), [Brian Epstein](/wiki/Brian_Epstein), [Jean Seberg](/wiki/Jean_Seberg), [Alan Wilson](/wiki/Alan_Wilson_(musician)), [Jimi Hendrix](/wiki/Jimi_Hendrix), [Edie Sedgwick](/wiki/Edie_Sedgwick), [Phyllis Hyman](/wiki/Phyllis_Hyman), [Inger Stevens](/wiki/Inger_Stevens), [Kenneth Williams](/wiki/Kenneth_Williams), [Felix Hausdorff](/wiki/Felix_Hausdorff) and [C. P. Ramanujam](/wiki/C._P._Ramanujam) each died from barbiturate overdose. [Ingeborg Bachmann](/wiki/Ingeborg_Bachmann) may have died of the consequences of barbiturate withdrawal.

## Recreational use[[edit](/index.php?title=(none)&action=edit&section=6)]

Recreational users report that a barbiturate high gives them feelings of relaxed contentment and [euphoria](/wiki/Euphoria). Physical and psychological dependence may also develop with repeated use.[[13]](#cite_note-13) Other effects of barbiturate [intoxication](/wiki/Drug_intoxication) include [drowsiness](/wiki/Drowsiness), [lateral](/wiki/Wikt:lateral) and [vertical](/wiki/Vertical_direction) [nystagmus](/wiki/Pathologic_nystagmus), [slurred speech](/wiki/Slurred_speech) and [ataxia](/wiki/Ataxia), decreased anxiety, a loss of inhibitions. Barbiturates are also used to alleviate the adverse or withdrawal effects of illicit drug use, in a manner similar to long-acting [benzodiazepines](/wiki/Benzodiazepines) such as [diazepam](/wiki/Diazepam) and [clonazepam](/wiki/Clonazepam).[[14]](#cite_note-14)[[15]](#cite_note-15) Drug users tend to prefer short-acting and intermediate-acting barbiturates.[[16]](#cite_note-16) The most commonly used are [amobarbital](/wiki/Amobarbital) (Amytal), [pentobarbital](/wiki/Pentobarbital) (Nembutal), and [secobarbital](/wiki/Secobarbital) (Seconal). A combination of amobarbital and secobarbital (called [Tuinal](/wiki/Tuinal)) is also highly used. Short-acting and intermediate-acting barbiturates are usually prescribed as sedatives and sleeping pills. These pills begin acting fifteen to forty minutes after they are swallowed, and their effects last from five to six hours.

Slang terms for barbiturates include barbs, bluebirds, dolls, wallbangers, yellows, downers, goofballs, sleepers, 'reds & blues' and tooties.[[17]](#cite_note-17)

## Mechanism of action[[edit](/index.php?title=(none)&action=edit&section=7)]

Barbiturates act as [positive allosteric modulators](/wiki/Positive_allosteric_modulator), and at higher doses, as [agonists](/wiki/Agonist) of [GABAA receptors](/wiki/GABAA).[[18]](#cite_note-18) [GABA](/wiki/GABA) is the principal inhibitory neurotransmitter in the [mammalian](/wiki/Mammal) [central nervous system](/wiki/Central_nervous_system) (CNS). Barbiturates bind to the GABAA receptor at multiple homologous transmembrane pockets located at subunit interfaces,[[19]](#cite_note-19) which are binding sites distinct from [GABA](/wiki/GABA) itself and also distinct from the [benzodiazepine](/wiki/Benzodiazepine) binding site. Like benzodiazepines, barbiturates potentiate the effect of GABA at this receptor. In addition to this GABAergic effect, barbiturates also block [AMPA](/wiki/AMPA_receptor) and [kainate receptors](/wiki/Kainate_receptor), subtypes of [ionotropic glutamate receptor](/wiki/Ionotropic_glutamate_receptor). Glutamate is the principal excitatory neurotransmitter in the mammalian CNS. Taken together, the findings that barbiturates potentiate inhibitory GABAA receptors and inhibit excitatory AMPA receptors can explain the superior CNS-depressant effects of these agents to alternative GABA potentiating agents such as benzodiazepines and [quinazolinones](/wiki/Quinazolinone). At higher concentration, they inhibit the [Ca2+](/wiki/Ca2+)-dependent release of neurotransmitters such as glutamate via an effect on [P](/wiki/P-type_calcium_channel)/[Q-type](/wiki/Q-type_calcium_channel) [voltage-dependent calcium channels](/wiki/Voltage-dependent_calcium_channel).[[20]](#cite_note-20)Barbiturates produce their pharmacological effects by increasing the duration of chloride ion channel opening at the GABAA receptor (pharmacodynamics: This increases the efficacy of GABA), whereas benzodiazepines increase the frequency of the chloride ion channel opening at the [GABAA](/wiki/GABAA) receptor (pharmacodynamics: This increases the potency of GABA). The direct gating or opening of the chloride ion channel is the reason for the increased toxicity of barbiturates compared to [benzodiazepines](/wiki/Benzodiazepines) in overdose.[[21]](#cite_note-21)[[22]](#cite_note-22) Further, barbiturates are relatively non-selective compounds that bind to an entire superfamily of ligand-gated ion channels, of which the GABAA receptor channel is only one of several representatives. This superfamily of ion channels includes the neuronal [nACh receptor](/wiki/NACh_receptor) channel, the [5-HT3 receptor](/wiki/5-HT3_receptor) channel, and the [glycine receptor](/wiki/Glycine_receptor) channel. However, while GABAA receptor currents are increased by barbiturates (and other general anaesthetics), ligand-gated ion channels that are predominantly permeable for cationic ions are blocked by these compounds. For example, neuronal nAChR channels are blocked by clinically relevant anaesthetic concentrations of both thiopental and pentobarbital.[[23]](#cite_note-23) Such findings implicate (non-GABA-ergic) ligand-gated ion channels, e.g. the neuronal nAChR channel, in mediating some of the (side) effects of barbiturates.[[24]](#cite_note-24) This is the mechanism responsible for the (mild to moderate) anesthetic effect of barbiturates in high doses when used in anesthetic concentration

## Legal status[[edit](/index.php?title=(none)&action=edit&section=8)]

During [World War II](/wiki/World_War_II), military personnel in the Pacific region were given "goofballs" to allow them to tolerate the heat and humidity of daily working conditions. Goofballs were distributed to reduce the demand on the respiratory system, as well as maintaining blood pressure, to combat the extreme conditions. Many soldiers returned with addictions that required several months of rehabilitation before discharge. This led to growing dependency problems, often exacerbated by indifferent doctors prescribing high doses to unknowing patients through the 1950s and 1960s. [Template:Citation needed](/wiki/Template:Citation_needed)

In the late 1950s and 1960s, increasing published reports of barbiturate [overdoses](/wiki/Drug_overdose) and dependence problems led physicians to cut back their prescription, particularly for spurious requests. This eventually led to the scheduling of barbiturates as controlled drugs.

In the United States, the [Controlled Substances Act](/wiki/Controlled_Substances_Act) of 1970 classified most barbiturates as controlled substances—and they remain so [Template:As of](/wiki/Template:As_of). [Barbital](/wiki/Barbital), [methylphenobarbital](/wiki/Methylphenobarbital) (also known as [mephobarbital](/wiki/Mephobarbital)), and [phenobarbital](/wiki/Phenobarbital) are designated [schedule IV](/wiki/Controlled_Substances_Act#Schedule_IV_drugs) drugs, and "Any substance which contains any quantity of a derivative of barbituric acid, or any salt of a derivative of barbituric acid"[[25]](#cite_note-25) (all other barbiturates) were designated as [schedule III](/wiki/Controlled_Substances_Act#Schedule_III_drugs). Under the original CSA, no barbiturates were placed in schedule I, II, or V,[[26]](#cite_note-26) however amobarbital, pentobarbital, and secobarbital are schedule II controlled substances unless they are in a suppository dosage form.[[27]](#cite_note-27) In 1971, the [Convention on Psychotropic Substances](/wiki/Convention_on_Psychotropic_Substances) was signed in [Vienna](/wiki/Vienna). Designed to regulate [amphetamines](/wiki/Amphetamine), barbiturates, and other synthetics, the 34th version of the [treaty](/wiki/Treaty), [Template:As of](/wiki/Template:As_of), regulates [secobarbital](/wiki/Secobarbital) as schedule II, [amobarbital](/wiki/Amobarbital), [butalbital](/wiki/Butalbital), [cyclobarbital](/wiki/Cyclobarbital), and [pentobarbital](/wiki/Pentobarbital) as schedule III, and [allobarbital](/wiki/Allobarbital), [barbital](/wiki/Barbital), [butobarbital](/wiki/Butobarbital), [mephobarbital](/wiki/Mephobarbital), [phenobarbital](/wiki/Phenobarbital), [butabarbital](/wiki/Butabarbital), and [vinylbital](/wiki/Vinylbital) as schedule IV on its "Green List".[[28]](#cite_note-28) The combination medication [Fioricet](/wiki/Fioricet), consisting of butalbital, caffeine, and [paracetamol](/wiki/Paracetamol) (acetaminophen), however, is specifically exempted from controlled substance status, while its sibling [Fiorinal](/wiki/Fiorinal), which contains aspirin instead of paracetamol and may contain [codeine](/wiki/Codeine) phosphate, remains a schedule III drug.

## Other uses in chemistry[[edit](/index.php?title=(none)&action=edit&section=9)]

In 1988, the synthesis and binding studies of an artificial receptor binding barbiturates by 6 complementary [hydrogen bonds](/wiki/Hydrogen_bonds) was published.[[29]](#cite_note-29) Since this first article, different kind of receptors were designed, as well as different barbiturates and [cyanurates](/wiki/Cyanuric_acid), not for their efficiencies as drugs but for applications in [supramolecular chemistry](/wiki/Supramolecular_chemistry), in the conception of materials and molecular devices.

Sodium barbital and barbital have also been used as pH buffers for biological research, e.g., in immunoelectrophoresis or in fixative solutions.[[30]](#cite_note-30)[[31]](#cite_note-31)

## Examples[[edit](/index.php?title=(none)&action=edit&section=10)]

[thumb|200px|Generic structure of a barbiturate, including numbering scheme](/wiki/File:Barbiturates_generic_numbered.svg)

|  |
| --- |
| + **Barbiturates** |
| **Short Name** | **R1** | **R2** | **IUPAC Name** |
| [allobarbital](/wiki/Allobarbital) | [CH2CHCH2](/wiki/Allyl) | [CH2CHCH2](/wiki/Allyl) | 5,5-diallylbarbiturate |
| [amobarbital](/wiki/Amobarbital)[[32]](#cite_note-32) | [CH2CH3](/wiki/Ethyl_group) | [(CH2)2CH(CH3)2](/wiki/Isopentyl) | 5-ethyl-5-isopentyl-barbiturate |
| [aprobarbital](/wiki/Aprobarbital) | [CH2CHCH2](/wiki/Allyl) | [CH(CH3)2](/wiki/Isopropyl) | 5-allyl-5-isopropyl-barbiturate |
| [alphenal](/wiki/Alphenal) | [CH2CHCH2](/wiki/Allyl) | [C6H5](/wiki/Phenyl) | 5-allyl-5-phenyl-barbiturate |
| [barbital](/wiki/Barbital) | [CH2CH3](/wiki/Ethyl_group) | [CH2CH3](/wiki/Ethyl_group) | 5,5-diethylbarbiturate |
| [brallobarbital](/wiki/Brallobarbital) | [CH2CHCH2](/wiki/Allyl) | [CH2CBrCH2](/wiki/Allyl) | 5-allyl-5-(2-bromo-allyl)-barbiturate |
| [pentobarbital](/wiki/Pentobarbital)[[32]](#cite_note-32) | [CH2CH3](/wiki/Ethyl_group) | [CH](/wiki/Methyl_group)[CH3(CH2)2CH3](/wiki/Butyl_group) | 5-ethyl-5-(1-methylbutyl)-barbiturate |
| [phenobarbital](/wiki/Phenobarbital)[[32]](#cite_note-32) | [CH2CH3](/wiki/Ethyl_group) | [C6H5](/wiki/Phenyl) | 5-ethyl-5-phenylbarbiturate |
| [secobarbital](/wiki/Secobarbital)[[32]](#cite_note-32) | [CH2CHCH2](/wiki/Allyl) | [CHCH3(CH2)2CH3](/wiki/Pentyl) | 5-[(2*R*)-pentan-2-yl]-5-prop-2-enyl-barbiturate; 5-allyl-5-[(2R)-pentan-2-yl]-barbiturate |

## History[[edit](/index.php?title=(none)&action=edit&section=11)]

[Barbituric acid](/wiki/Barbituric_acid) was first synthesized November 27, 1864, by [German](/wiki/Germany) chemist [Adolf von Baeyer](/wiki/Adolf_von_Baeyer). This was done by [condensing](/wiki/Condensation_(chemistry)) [urea](/wiki/Urea) (an animal waste product) with [diethyl malonate](/wiki/Diethyl_malonate) (an [ester](/wiki/Ester) derived from the [acid](/wiki/Acid) of [apples](/wiki/Apple)). There are several stories about how the substance got its name. The most likely story is that Baeyer and his colleagues went to celebrate their discovery in a [tavern](/wiki/Tavern) where the town's [artillery](/wiki/Artillery) [garrison](/wiki/Garrison) were also celebrating the feast of [Saint Barbara](/wiki/Saint_Barbara) – the patron saint of artillerymen. An artillery officer is said to have christened the new substance by amalgamating *Barbara* with *urea*.[[33]](#cite_note-33) Another story holds that Baeyer synthesized the substance from the collected urine of a Munich waitress named Barbara.[[34]](#cite_note-34) No substance of medical value was discovered, however, until 1903 when two German scientists working at [Bayer](/wiki/Bayer), [Emil Fischer](/wiki/Franz_Joseph_Emil_Fischer) and [Joseph von Mering](/wiki/Joseph_von_Mering), discovered that [barbital](/wiki/Barbital) was very effective in putting dogs to sleep. Barbital was then marketed by Bayer under the [trade name](/wiki/Trade_name) [Veronal](/wiki/Veronal). It is said that Mering proposed this name because the most peaceful place he knew was the [Italian](/wiki/Italy) city of [Verona](/wiki/Verona).[[33]](#cite_note-33) It was not until the 1950s that the behavioural disturbances and physical dependence potential of barbiturates became recognized.[[35]](#cite_note-35) Barbituric acid itself does not have any direct effect on the [central nervous system](/wiki/Central_nervous_system) and chemists have derived over 2,500 compounds from it that possess pharmacologically active qualities. The broad class of barbiturates is further broken down and classified according to speed of onset and duration of action. Ultrashort-acting barbiturates are commonly used for [anesthesia](/wiki/Anesthesia) because their extremely short duration of action allows for greater control. These properties allow doctors to rapidly put a patient "under" in emergency surgery situations. Doctors can also bring a patient out of anesthesia just as quickly, should complications arise during surgery. The middle two classes of barbiturates are often combined under the title "short/intermediate-acting." These barbiturates are also employed for anesthetic purposes, and are also sometimes prescribed for [anxiety](/wiki/Anxiety) or [insomnia](/wiki/Insomnia). This is not a common practice anymore, however, owing to the dangers of long-term use of barbiturates; they have been replaced by the [benzodiazepines](/wiki/Benzodiazepines) for these purposes. The final class of barbiturates are known as long-acting barbiturates (the most notable one being phenobarbital, which has a half-life of roughly 92 hours). This class of barbiturates is used almost exclusively as [anticonvulsants](/wiki/Anticonvulsants), although on rare occasions they are prescribed for daytime sedation. Barbiturates in this class are not used for insomnia, because, owing to their extremely long half-life, patients would awake with a residual "hang-over" effect and feel groggy.

Barbiturates can in most cases be used either as the free acid or as salts of sodium, calcium, potassium, magnesium, lithium, etc. [Codeine](/wiki/Codeine)- and [Dionine](/wiki/Ethylmorphine)-based salts of barbituric acid have been developed. In 1912, Bayer introduced another barbituric acid derivative, [phenobarbital](/wiki/Phenobarbital), under the trade name Luminal, as a [sedative](/wiki/Sedative)-[hypnotic](/wiki/Hypnotic).[[36]](#cite_note-36)

## See also[[edit](/index.php?title=(none)&action=edit&section=12)]

[Template:Portal](/wiki/Template:Portal)

* [Benzodiazepines](/wiki/Benzodiazepines)
* [Psycholeptic](/wiki/Psycholeptic)
* The [Dille–Koppanyi reagent](/wiki/Dille–Koppanyi_reagent), used as a spot test for barbiturates.
* The [Zwikker reagent](/wiki/Zwikker_reagent), also used as a spot test for barbiturates.

[Template:Clear](/wiki/Template:Clear)

## References[[edit](/index.php?title=(none)&action=edit&section=13)]

[Template:Reflist](/wiki/Template:Reflist)

## External links[[edit](/index.php?title=(none)&action=edit&section=14)]

[Template:Wiktionary](/wiki/Template:Wiktionary)

* [U.S. Drug Enforcement Administration](http://www.usdoj.gov/dea/concern/depressants.html) Source for some public domain text used on this page.
* [History of Barbiturates](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2424120/)
* [National Institute on Drug Abuse](/wiki/National_Institute_on_Drug_Abuse): "[NIDA for Teens: Prescription Depressant Medications](http://teens.drugabuse.gov/drug-facts/central-nervous-system-cns-depressants-facts)".

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