For the management of narcolepsy in adults, sodium oxybate is given in initial oral doses of 4.5 g daily, as two equally-divided doses. The first dose should be taken at bedtime while in bed and at least 2 to 3 hours after food; the second dose should be taken 2.5 to 4 hours later also while sitting in bed. Both doses should be prepared before going to bed: each dose should be diluted with 60 mL of water. The initial dose may be increased in steps of 1.5 g (0.75 g per dose) every 1 to 2 weeks to a maximum dose of 9 g daily. If therapy has been stopped for more than 14 consecutive days, titration should be restarted at the lowest dose. Reduced doses are recommended in patients with hepatic impairment (see below).

Administration in hepatic impairment. The recommended initial oral dose of sodium oxybate (see above) should be halved in patients with hepatic impairment. Subsequent increases should be monitored against effect.

Alcohol withdrawal syndrome. Gamma-hydroxybutyric acid has been reported[1] to be effective in reducing symptoms of alcohol withdrawal (p.1626) and to be of use as an aid in the maintenance of abstinence.[2,3] However, following reports of CNS toxicity associated with abuse of gamma-hydroxybutyric acid its role in the treatment of substance abuse disorders appears questionable.[4]

1. Gallimberti L, *et al.* Gamma-hydroxybutyric acid for treatment of alcohol withdrawal syndrome. *Lancet* 1989; **ii:** 787–9.
2. Gallimberti L, *et al.* Gamma-hydroxybutyric acid in the treatment of alcohol dependence: a double blind study. *Alcohol Clin Exp Res* 1992; **16:** 673–6.
3. Addolorato G, *et al.* Maintaining abstinence from alcohol with γ-hydroxybutyric acid. *Lancet* 1998; **351:** 38.
4. Quinn DI, *et al.* Pharmacokinetic and pharmacodynamic principles of illicit drug use and treatment of illicit drug users. *Clin Pharmacokinet* 1997; **33:** 344–400.

Narcoleptic syndrome. Sodium oxybate[1-7] given at night is used to improve cataplexy and excessive daytime sleepiness in patients with narcoleptic syndrome (p.2148).

For a reference to the pharmacokinetics of sodium oxybate in narcoleptic patients, see above.

1. Scharf MB, *et al.* The effects and effectiveness of γ-hydroxybutyrate in patients with narcolepsy. *J Clin Psychiatry* 1985; **46:** 222–5.
2. Mamelak M, *et al.* Treatment of narcolepsy with γ-hydroxybutyrate: a review of clinical and sleep laboratory findings. *Sleep* 1986; **9:** 285–9.
3. Scrima L, *et al.* Efficacy of gamma-hydroxybutyrate versus placebo in treating narcolepsy-cataplexy: double-blind subjective measures. *Biol Psychiatry* 1989; **26:** 331–43.
4. US Xyrem Multicenter Study Group. A randomized, double blind, placebo-controlled multicenter trial comparing the effects of three doses of orally administered sodium oxybate with placebo for the treatment of narcolepsy. *Sleep* 2002; **25:** 42–9.
5. US Xyrem Multicenter Study Group. A 12-month, open-label, multicenter extension trial of orally administered sodium oxybate for the treatment of narcolepsy. *Sleep* 2003; **26:** 31–5.
6. Xyrem International Study Group. Further evidence supporting the use of sodium oxybate for the treatment of cataplexy: a double-blind, placebo-controlled study in 228 patients. *Sleep Med* 2005; **6:** 415–21.
7. Lemon MD, *et al.* Sodium oxybate for cataplexy. *Ann Pharmacother* 2006; **40:** 433–40.

Preparations

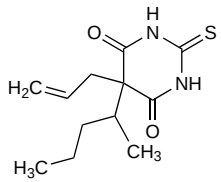
Proprietary Preparations (details are given in Part 3)
Austria: Alcover; ***Cz.:*** Xyrem; ***Fr.:*** Gamma-OH; ***Ger.:*** Somsanit; ***Hung.:*** Alcover†; ***Ital.:*** Alcover; ***Neth.:*** Xyrem; ***Port.:*** Xyrem; ***UK:*** Xyrem; ***USA:*** Xyrem.

Thiamylal Sodium

Tiamilal sódico. Sodium 5-allyl-5-(1-methylbutyl)-2-thiobarbiturate.

$C_{12}H_{17}N_2NaO_2S$ = 276.3.

CAS — 77-27-0 (thiamylal); 337-47-3 (thiamylal sodium).



(thiamylal)

Pharmacopoeias. In *Jpn.*

Profile

Thiamylal sodium is a short-acting intravenous barbiturate anaesthetic. It is possibly slightly more potent than thiopental sodium (p.1796) and has similar actions and uses. It has been used for the production of complete anaesthesia of short duration, for the induction of general anaesthesia, or for inducing a hypnotic state.

Thiopental Sodium *(BANM, rINN)*

Natrium Isopentylaethylthiobarbituricum (cum Natrio Carbonico); Penthiobarbital Sodique; Sodyum Tiopental; Sodyum Tiopenton; Soluble Thiopentone; Thiomebumalnatrium cum Natrii Carbonate; Thiopental et carbonate sodiques; Thiopental Sodique; Thiopental Sodium and Sodium Carbonate; Thiopental sodná sůl a uhličitan sodný; Thiopentalum Natricum; Thiopentalum natricum et natrii carbonas; Thiopentobarbitalum Solubile; Thiopentone Sodium; Tiopentaalinatrium; Tiopentaalinatrium ja natriumkarbonaatti; Tiopental sódico; Tiopentalio natrio druska ir natrio karbonatas; Tiopentalnatrium; Tiopentál-nátrium és nátrium-karbonát; Tiopentalnatrium och natriumkarbonat. Sodium 5-ethyl-5-(1-methylbutyl)-2-thiobarbiturate.

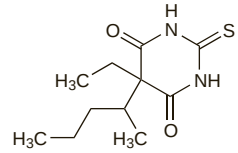
Тиопентал Натрий

$C_{11}H_{17}N_2NaO_2S$ = 264.3.

CAS — 76-75-5 (thiopental); 71-73-8 (thiopental sodium).

ATC — N01AF03; N05CA19.

ATC Vet — QN01AF03; QN05CA19.



(thiopental)

NOTE. The name thiobarbital has been applied to thiopental and has also been used to describe a barbiturate of different composition.

Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), *Int.*, *Jpn*, *US*, and *Viet.* Some include thiopental sodium with, some without, anhydrous sodium carbonate; some only include a sterile mixture for injection.

Ph. Eur. 6.2 (Thiopental Sodium and Sodium Carbonate; Thiopental Sodium BP 2008). A yellowish-white hygroscopic powder. It contains 84 to 87% thiopental and 10.2 to 11.2% sodium. Freely soluble in water; partly soluble in dehydrated alcohol. Store in airtight containers. Protect from light.

USP 31 (Thiopental Sodium). A white to off-white crystalline powder, or yellowish-white to pale greenish-yellow hygroscopic powder. May have a disagreeable odour. Its solutions are alkaline to litmus, decompose on standing, and on boiling, precipitation occurs. Soluble in water and in alcohol; insoluble in ether, in petroleum spirit, and in benzene. Store in airtight containers.

Incompatibility. Solutions of thiopental sodium are incompatible with acidic and oxidising substances including some antibacterials, neuromuscular blockers and analgesics. Compounds commonly listed as incompatible include amikacin sulfate, benzylpenicillin salts, cefapirin sodium, codeine phosphate, ephedrine sulfate, fentanyl citrate, glycopyrronium bromide, morphine sulfate, pentazocine lactate, prochlorperazine edisilate, suxamethonium salts, and tubocurarine chloride. Solutions decompose on standing and precipitation occurs on boiling.

Stability. Loss of thiopental in PVC and cellulose propionate delivery systems has been reported,[1,2] but in another study,[3] no loss of potency was noted. Adsorption has been reported[4] not to occur in polyolefin infusion bags.

1. Kowaluk EA, *et al.* Interactions between drugs and polyvinyl chloride infusion bags. *Am J Hosp Pharm* 1981; **38:** 1308–14.
2. Kowaluk EA, *et al.* Interactions between drugs and intravenous delivery systems. *Am J Hosp Pharm* 1982; **39:** 460–7.
3. Martens HJ, *et al.* Sorption of various drugs in polyvinyl chloride, glass, and polyethylene-lined infusion containers. *Am J Hosp Pharm* 1990; **47:** 369–73.
4. Trissel LA, *et al.* Drug compatibility with new polyolefin infusion solution containers. *Am J Health-Syst Pharm* 2006; **63:** 2379–82.

Adverse Effects and Treatment

As for Phenobarbital, p.492.

Excitatory phenomena such as coughing, hiccuping, sneezing, and muscle twitching or jerking may occur with any of the barbiturate anaesthetics, particularly during induction, but they occur more frequently with methohexital than with thiopental. Cough, sneezing, and laryngeal spasm or bronchospasm may also occur during induction. The intravenous injection of concentrated solutions of thiopental sodium such as 5% may result in thrombophlebitis. Extravasation of barbiturate anaesthetics may cause tissue necrosis. Intra-arterial injection causes severe arterial spasm with burning pain and may cause prolonged blanching of the forearm and hand and gangrene of digits. Hypersensitivity reactions have been reported. Barbiturate anaesthetics can cause respiratory depression. They depress cardiac output and often cause an initial fall in blood pressure, and overdosage may result in circulatory failure. Arrhythmias may occur. Postoperative vomiting is infrequent but shivering may occur and there may be persistent drowsiness, confusion, and amnesia. Headache has also been reported.

See also under Adverse Effects of General Anaesthetics, p.1779.

Hypersensitivity. Anaphylactic reactions to thiopental have been reported[1,2] although such reactions are rare. There has also been a report of haemolytic anaemia and renal failure in association with the development of an anti-thiopental antibody in a patient who had undergone general anaesthesia induced by thiopental.[3]

1. Westacott P, *et al.* Anaphylactic reaction to thiopentone: a case report. *Can Anaesth Soc J* 1984; **31:** 434–8.
2. Moneret-Vautrin DA, *et al.* Simultaneous anaphylaxis to thiopentone and a neuromuscular blocker: a study of two cases. *Br J Anaesth* 1990; **64:** 743–5.
3. Habibi B, *et al.* Thiopental-related immune hemolytic anemia and renal failure: specific involvement of red-cell antigen I. *N Engl J Med* 1985; **312:** 353–5. Correction. *ibid.*; 1136.

Intra-arterial injection. Accidental intra-arterial injection of thiopental sodium produces severe arterial spasm with intense burning pain. Anaesthesia, paresis, paralysis, and gangrene may occur. Therapy has concentrated on dilution of injected thiopental, prevention and treatment of arterial spasm, prophylaxis of thrombosis, thrombectomy and other measures to sustain good blood flow. There has been a report[1] of the successful use of urokinase intra-arterially in the management of one patient accidentally given thiopental intra-arterially.

1. Vangerven M, *et al.* A new therapeutic approach to accidental intra-arterial injection of thiopentone. *Br J Anaesth* 1989; **62:** 98–100.

Precautions

Barbiturate anaesthetics are contra-indicated when there is dyspnoea or respiratory obstruction such as in acute severe asthma or when maintenance of an airway cannot be guaranteed.

Barbiturate anaesthetics should be used with caution in shock and dehydration, hypovolaemia, severe anaemia, hyperkalaemia, toxaemia, myasthenia gravis, myxoedema and other metabolic disorders, or in severe renal disease. Caution is also required in patients with cardiovascular disease, muscular dystrophies, adrenocortical insufficiency, or with increased intracranial pressure. Reduced doses are required in the elderly and in severe hepatic disease.

See also Precautions for General Anaesthetics, p.1779.

Breast feeding. No adverse effects have been seen in breast-fed infants whose mothers received thiopental, and the American Academy of Pediatrics[1] considers that it is therefore usually compatible with breast feeding.

In two groups of 8 women undergoing induction with thiopental, the milk-to-plasma ratio was less than 1 in both groups and it was considered that the effects of thiopental on breast-fed infants would be negligible.[2]

1. American Academy of Pediatrics. The transfer of drugs and other chemicals into human milk. *Pediatrics* 2001; **108:** 776–89. Correction. *ibid.*; 1029. Also available at: http://aappolicy.aappublications.org/cgi/content/full/pediatrics%3b108/3/776 (accessed 26/05/04)
2. Andersen LW, *et al.* Concentrations of thiopentone in mature breast milk and colostrum following an induction dose. *Acta Anaesthesiol Scand* 1987; **31:** 30–2.

Porphyria. Barbiturates including thiopental sodium have been associated with acute attacks of porphyria and are considered unsafe in porphyric patients.

Interactions

Difficulty may be experienced in producing anaesthesia with the usual dose of barbiturate anaesthetics in patients accustomed to taking alcohol or other CNS depressants; additional anaesthetics may be necessary. Patients being treated with phenothiazine antipsychotics may experience increased hypotension. Some phenothiazines, especially promethazine, may also increase the incidence of excitatory phenomena produced by barbiturate anaesthetics; cyclizine may possibly have a similar effect. Opioid analgesics can potentiate the respiratory depressant effect of barbiturate anaesthetics and the dose of the anaesthetic may need to be reduced. Use with nitrous oxide greatly reduces the dose of barbiturate anaesthetics required for

anaesthesia. Reduced doses of thiopental may be required in patients receiving sulfafurazole.

See also Interactions of General Anaesthetics, p.1779.

Antidepressants. Potentiation of barbiturate anaesthesia may be expected in patients receiving *tricyclic antidepressants* or *MAOIs* (see Anaesthesia under Precautions of Amitriptyline, p.378 and for Phenelzine, p.417, respectively).

Antipsychotics. For mention of the effect of *droperidol* on thiopental see Gastrointestinal Drugs, below.

Aspirin. Pretreatment with aspirin, a highly protein-bound drug, has been shown to potentiate thiopental anaesthesia.[1]

1. Dundee JW, *et al.* Aspirin and probenecid pretreatment influences the potency of thiopentone and the onset of action of midazolam. *Eur J Anaesthesiol* 1986; **3:** 247–51.

Gastrointestinal drugs. *Metoclopramide* profoundly reduced the dose of thiopental required to produce hypnosis in female patients; *droperidol* had a similar effect.[1]

1. Mehta D, *et al.* Metoclopramide decreases thiopental hypnotic requirement. *Anesth Analg* 1993; **77:** 784–7.

Probenecid. Pretreatment with probenecid, a highly protein-bound drug, has been shown to potentiate thiopental anaesthesia.[1]

1. Dundee JW, *et al.* Aspirin and probenecid pretreatment influences the potency of thiopentone and the onset of action of midazolam. *Eur J Anaesthesiol* 1986; **3:** 247–51.

Pharmacokinetics

Thiopental is highly lipid soluble and when it is given intravenously as the sodium salt, concentrations sufficient to produce unconsciousness are achieved in the brain within 30 seconds. Onset of action occurs within 8 to 10 minutes when thiopental sodium is given rectally but absorption may be unpredictable if a suspension rather than a solution is used. Recovery from anaesthesia is also rapid due to redistribution to other tissues, particularly fat. About 80% of thiopental may be bound to plasma proteins, although reports show a wide range of figures. Thiopental is metabolised almost entirely in the liver, but as it is only released slowly from lipid stores this occurs at a very slow rate.

It is mostly metabolised to inactive metabolites but a small amount is desulfurated to pentobarbital. Repeated or continuous use can lead to accumulation of thiopental in fatty tissue and this can result in prolonged anaesthesia and respiratory and cardiovascular depression. Elimination of thiopental after bolus injection can be described by a triexponential curve. The terminal elimination half-life has been reported to be 10 to 12 hours in adults and about 6 hours in children. However, values of 26 to 28 hours have been reported in obese patients and pregnant patients at term. Thiopental readily diffuses across the placenta and is distributed into breast milk.

◊ References.

1. Gaspari F, *et al.* Elimination kinetics of thiopentone in mothers and their newborn infants. *Eur J Clin Pharmacol* 1985; **28:** 321–5.
2. Swerdlow BN, Holley FO. Intravenous anaesthetic agents: pharmacokinetic-pharmacodynamic relationships. *Clin Pharmacokinet* 1987; **12:** 79–110.
3. Esener Z, *et al.* Thiopentone and etomidate concentrations in maternal and umbilical plasma, and in colostrum. *Br J Anaesth* 1992; **69:** 586–8.
4. Gedney JA, Ghosh S. Pharmacokinetics of analgesics, sedatives and anaesthetic agents during cardiopulmonary bypass. *Br J Anaesth* 1995; **75:** 344–51.

Uses and Administration

Thiopental is a short-acting barbiturate anaesthetic. It is given intravenously, usually for the induction of general anaesthesia (p.1780), but may be used as the sole anaesthetic to maintain anaesthesia for short procedures with minimal painful stimuli. It is also used in anaesthesia as a supplement to other anaesthetics and as a hypnotic in balanced anaesthesia. Thiopental sodium may also be used intravenously in the control of refractory tonic-clonic status epilepticus and in neurosurgical patients to reduce increased intracranial pressure. It has also been given rectally for basal anaesthesia or basal narcosis.

Thiopental does not usually produce excitation and induction of anaesthesia is usually smooth. It has poor muscle relaxant properties and a muscle relaxant must be given before intubation is attempted. Thiopental also has poor analgesic properties and small doses may even lower the pain threshold. Recovery from moderate doses usually occurs within 10 to 30 minutes, but the patient may remain sleepy or confused for several hours. Large doses, repeated smaller doses, or continuous use may markedly delay recovery.

In **anaesthesia**, the dosage of thiopental varies greatly according to the state of the patient and the nature of other drugs being used concurrently (see under Precautions above and Interactions above for further details). Thiopental is usually given intravenously as the sodium salt as a 2.5% solution but a 5% solution is occasionally used. UK licensed product information states that a typical dose for inducing anaesthesia is 100 to 150 mg injected over 10 to 15 seconds, repeated after 30 to 60 seconds according to response. It also recommends that the total dosage used should not exceed 500 mg; in pregnant patients the total maximum dose is 250 mg. In some other countries, it is recommended that induction begin with a test dose of 25 to 75 mg; thereafter, a dose of 50 to 75 mg may be given at intervals of 20 to 40 seconds according to response. Once anaesthesia has been established, additional doses of 25 to 50 mg may be given as necessary. When thiopental is used as the sole anaesthetic, anaesthesia can be maintained by repeat doses as needed or by continuous intravenous infusion of a 0.2 or 0.4% solution.

To **reduce elevations of intracranial pressure** in neurological patients, thiopental sodium is licensed for use as intermittent bolus injections of 1.5 to 3 mg/kg if adequate ventilation is provided (but see also Cerebrovascular Disorders, below). Higher doses have been tried.

For suggested doses in refractory tonic-clonic **status epilepticus** see Status Epilepticus, below.

For dosage in *children* and the *elderly*, also see below.

◊ References.

1. Russo H, Bressolle F. Pharmacodynamics and pharmacokinetics of thiopental. *Clin Pharmacokinet* 1998; **35:** 95–134.

Administration in children. For the *induction of anaesthesia* in children, UK licensed product information recommends that thiopental sodium is given by slow intravenous injection (over 10 to 15 seconds) in a dose of 2 to 7 mg/kg; the dose may be repeated after 1 minute.

In the treatment of prolonged *status epilepticus* (see below), the *BNFC* recommends an initial dose of up to 2 mg/kg in neonates or up to 4 mg/kg in children aged 1 month and over, given as a slow intravenous injection; for all patients, this should then be followed by a continuous intravenous infusion of up to 8 mg/kg per hour, adjusted according to response.

Intravenous injections are normally given as a 2.5% solution; the *BNFC* recommends that intravenous infusions are given as a 0.25% solution.

Administration in the elderly. It is usually recommended that the dosage of barbiturate anaesthetics is reduced in the elderly. A study[1] in elderly patients demonstrated that although reducing the rate of intravenous injection reduced the speed of induction, the dosage required was also reduced. Giving thiopental sodium 2.5% solution at a rate of 125 mg/minute induced anaesthesia in a mean of 90.8 seconds and required a mean dose of 2.8 mg/kg. Corresponding values for a rate of 500 mg/minute were 40.8 seconds and 5 mg/kg, respectively.

1. Berthoud MC, *et al.* Comparison of infusion rates of three i.v. anaesthetic agents for induction in elderly patients. *Br J Anaesth* 1993; **70:** 423–7.

Anaesthesia. Some of the adverse effects of the neuromuscular blocker suxamethonium may be reduced when thiopental is used as part of the anaesthetic regimen. For a suggestion that thiopental may help to counteract the rise in intra-ocular pressure associated with the use of suxamethonium for intubation, see under Anaesthesia, p.1900.

Cerebrovascular disorders. Barbiturates are considered to be suitable anaesthetics for use in patients with or at risk of raised intracranial pressure. Barbiturate-induced coma (commonly with pentobarbital or thiopental) has been used, both therapeutically and prophylactically, to protect the brain from ischaemia resulting from neurological insults including head injury, stroke, Reye's syndrome, and hepatic encephalopathy.[1-3] Rationale includes the ability of barbiturates to reduce intracranial pressure and to reduce metabolic demands of cerebral tissues. Although thiopental protected patients against the neuropsychiatric complications of cardiopulmonary bypass,[4] the Brain Resuscitation Clinical Trial I Study Group[5] found no cerebral benefit from thiopental in comatose survivors of cardiac arrest. Nor did others[6] observe any benefit from thiopental-induced coma in infants with severe birth asphyxia. A review in 1989 considered that there was no convincing evidence of improvement in neurological outcome to justify the risks of the procedure in conditions causing global ischaemia, although use of barbiturates without necessarily inducing coma might have a limited role in reduction of raised intracranial pressure refractory to other therapy. Use of barbiturates in the setting of regional cerebral ischaemia, including use during cardiopulmonary bypass to prevent focal neurological complications, remained controversial.[1] A systematic review[7] came to similar conclusions, pointing out that although the barbiturates may reduce intracranial pressure their hypotensive effects are likely to offset any beneficial action on cerebral perfusion, perhaps accounting for the lack of evidence for any clinical benefit.

For a discussion of the treatment of raised intracranial pressure, including a mention of the use of barbiturates, see p.1181.

1. Rogers MC, Kirsch JR. Current concepts in brain resuscitation. *JAMA* 1989; **261:** 3143–7.
2. Lyons MK, Meyer FB. Cerebrospinal fluid physiology and the management of increased intracranial pressure. *Mayo Clin Proc* 1990; **65:** 684–707.
3. Woster PS, LeBlanc KL. Management of elevated intracranial pressure. *Clin Pharm* 1990; **9:** 762–72.
4. Nussmeier NA, *et al.* Neuropsychiatric complications after cardiopulmonary bypass: cerebral protection by a barbiturate. *Anesthesiology* 1986; **64:** 165–70.
5. Abramson NS, *et al.* Randomized clinical study of thiopental loading in comatose survivors of cardiac arrest. *N Engl J Med* 1986; **314:** 397–403.
6. Eyre JA, Wilkinson AR. Thiopentone induced coma after severe birth asphyxia. *Arch Dis Child* 1986; **61:** 1084–9.
7. Roberts I. Barbiturates for acute traumatic brain injury. Available in The Cochrane Database of Systematic Reviews; Issue 3. Chichester: John Wiley; 1999 (accessed 16/06/05).

Status epilepticus. General anaesthesia may be used to control refractory tonic-clonic status epilepticus (p.469). A short-acting barbiturate such as thiopental is usually used.

Doses of thiopental sodium in the treatment of status epilepticus appear to vary widely and may be defined by local clinical protocols. Licensed product information and the *BNF* recommend a dose of 75 to 125 mg intravenously as a 2.5% solution. Other regimens advocate an initial loading dose followed by further intermittent doses or a continuous infusion. One high dose regimen suggests an intravenous loading dose of 5 mg/kg followed after 30 minutes by an infusion given at a rate of 1 to 3 mg/kg per hour and adjusted to maintain a maximum blood concentration of 60 to 100 micrograms/mL.[1] It has been recommended that dosage should be continued for at least 12 hours after seizure activity has ceased and then slowly stopped.[2] Recovery may be prolonged.[3]

For the dose of thiopental in children see Administration in Children, above.

1. O'Brien MD. Management of major status epilepticus in adults. *BMJ* 1990; **301:** 918.
2. Bauer J, Elger CE. Management of status epilepticus in adults. *CNS Drugs* 1994; **1:** 26–44.
3. Parviainen I, *et al.* High-dose thiopental in the treatment of refractory status epilepticus in intensive care unit. *Neurology* 2002; **59:** 1249–51.

Preparations

BP 2008: Thiopental Injection;
USP 31: Thiopental Sodium for Injection.

Proprietary Preparations (details are given in Part 3)
Arg.: Bensulf; Hipnopento; Pentothal†; ***Austral.:*** Pentothal; ***Belg.:*** Pentothal; ***Braz.:*** Thionembutal; Thiopentax; ***Canad.:*** Pentothal; ***Chile:*** Pentothal†; ***Denm.:*** Pentothal; ***Fin.:*** Pentothal; ***Ger.:*** Trapanal; ***Gr.:*** Pentothal; ***Hong Kong:*** Pentothal; ***Hung.:*** Trapanal†; ***India:*** Anesthal; ***Indon.:*** Pentothal; ***Irl.:*** Intraval Sodium†; ***Israel:*** Pentothal; ***Ital.:*** Farmotal; Pentothal; ***Malaysia:*** Pentotex; ***Mex.:*** Pentarim; Pentothal; Sodipental; ***Neth.:*** Pentothal; ***Norw.:*** Pentothal; ***NZ:*** Intraval; ***Philipp.:*** Pentobrim; Pentothal; ***Singapore:*** Pentothal†; ***Spain:*** Pentothal; Tiobarbital; ***Swed.:*** Pentothal; ***Switz.:*** Pentothal; ***Thai.:*** Pentothal; ***Turk.:*** Pental; Pentothal; ***USA:*** Pentothal; ***Venez.:*** Pentothal†.

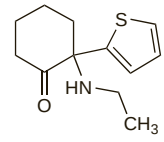
Tiletamine Hydrochloride *(BANM, USAN, rINNM)*

CI-634; CL-399; CN-54521-2; Hidrocloruro de tiletamina; Tilétamine, Chlorhydrate de; Tiletamini Hydrochloridum. 2-Ethylamino-2-(2-thienyl)cyclohexanone hydrochloride.

Тилетамина Гидрохлорид

$C_{12}H_{17}NOS,HCl = 259.8$.

CAS — 14176-49-9 (tiletamine); 14176-50-2 (tiletamine hydrochloride).



(tiletamine)

Pharmacopoeias. In *US* for veterinary use only.

USP 31 (Tiletamine Hydrochloride). A white to off-white crystalline powder. Freely soluble in water; slightly soluble in chloroform; practically insoluble in ether; soluble in methyl alcohol;