Chelators Antidotes and Antagonists

The drugs included in this chapter act in a variety of ways to counter the toxic effects of exogenous and endogenous substances in the body. They are therefore used in the management of poisoning and overdosage, to protect against the toxicity of drugs such as antineoplastics, and in the management of metabolic disorders such as Wilson's disease where toxic substances accumulate.

The main groups of drugs used include:

- antagonists, such as the opioid antagonist naloxone, that compete with the poison for receptor sites. Other antagonists act by blocking substances that mediate the effects of the toxin; atropine (p.1219) acts in this way
- chelators and other drugs that form complexes with the toxin. This may reduce absorption of the toxin from the gastrointestinal tract, inactivate or reduce the activity of the toxin, or increase its elimination
- drugs that affect the metabolism of the toxin. Some antidotes, such as fomepizole in methyl alcohol poisoning, act by reducing the rate of metabolism to a toxic metabolite; alcohol (p.1625) has a similar action. Others, such as methionine and glutathione, promote the formation of inactive metabolites; acetylcysteine (p.1548) also acts in this way
- drugs that bypass the effect of the toxin. Calcium folinate (p.1943) is used for this purpose in methotrexate overdosage.

Acute poisoning

In the management of suspected acute poisoning it is often impossible to determine the identity of the poison or the size of the dose received with any certainty. Moreover, few poisons have specific antidotes or methods of elimination, and the mainstay of treatment for patients with suspected acute poisoning is therefore supportive and symptomatic therapy; in many cases nothing further is required. Symptoms of acute poisoning are frequently non-specific, particularly in the early stages. Maintenance of the airway and ventilation is the most important initial measure; other treatment, for example for cardiovascular or neurological symptoms, may be added as appropriate. Patients who are unconscious or who have respiratory depression may be given naloxone, particularly if opioid overdosage is a possibility. Some centres also recommend the routine administration of glucose to all unconscious patients since hypoglycaemia may be a cause of unconsciousness, although blood glucose measurements should be obtained first where facilities are immediately available; thiamine may be given in addition since glucose may precipitate Wernicke's encephalopathy.

Specific antidotes are available for a number of poisons and are the primary treatment where there is severe poisoning with a known toxin. They may be life-saving in such cases but their use is not without hazard and in many situations they are not necessary; their use does not preclude relevant supportive treatment.

Measures to reduce or prevent the absorption of the poison are widely advocated. For inhalational poisoning the victim is removed from the source of poisoning. Some toxins, in particular pesticides, may be absorbed through the skin, and clothing should be removed and the skin thoroughly washed to avoid continued absorption. Caustic substances are removed from the skin or eyes with copious irrigation. However, for orally ingested poisons the best method for gastrointestinal decontamination remains controversial.

Activated charcoal adsorbs a wide range of toxins and is often given to reduce absorption from the gastrointestinal tract. A single dose is generally effective, particularly if it is given within one hour of ingestion, although delayed use may be beneficial for modified-release preparations or for drugs that slow gastrointestinal transit time, such as those with antimuscarinic properties. Charcoal is generally well tolerated, although vomiting is common and there is a risk of aspiration if the airway is not adequately protected. Repeated doses may be of use to eliminate some substances even after systemic absorption has occurred.

Active removal of poisons from the stomach by induction of emesis or gastric lavage has been widely used, but there is little evidence to support its role (see under Ipecacuanha, p.1563). Induction of emesis with an emetic such as syrup of ipecacuanha has been used but is no longer recommended for either the home or hospital situation since there is no evidence that it improves outcomes and it may increase the risk of aspiration. If used at all, it should only be in fully conscious patients, where a potentially toxic amount has been ingested within the previous hour, and where other measures are unavailable or inappropriate. Emesis should not be induced if the poison is corrosive or petroleum based, nor if the poison is removable by treatment with activated charcoal. Gastric lavage may occasionally be indicated for ingestion of non-caustic poisons that are not absorbed by activated charcoal, but only if less than one hour has elapsed since ingestion; it should not be attempted if the airway is not adequately protected.

Whole-bowel irrigation using a non-absorbable osmotic agent such as a macrogol has also been used, particularly for substances that pass beyond the stomach before being absorbed, such as iron preparations or enteric-coated or modified-release formulations, but its role is not established.

Techniques intended to promote the elimination of poisons from the body, such as haemodialysis or haemoperfusion, are only of value for a limited number of poisons in a few severely poisoned patients. Forced diuresis is no longer recommended, although alkalinisation of the urine using sodium bicarbonate infusion may have a role for selected poisons. Repeated oral doses of activated charcoal may be as effective as these more invasive methods for some drugs that undergo enterohepatic or enteroenteric recycling.

Poisons Information Centres exist in many countries and should be consulted for more detailed information in specific situations.

Activated Charcoal

Aktif Kömür; Aktiivihiili; Aktivált szén; Aktyvintosios anglys; Carbo activatus; Carbo Medicinalis; Carbón activado; Charbon activé; Decolorising Charcoal; Kol, aktivt; Medicinal Charcoal; Uhlí aktivní; Węgiel leczniczy.

CAS — 16291-96-6 (charcoal).

ATC — A07BA01.

ATC Vet — QA07BA01.

Pharmacopoeias. In *Chin.*, *Eur.* (see p.vii), *Int.*, *Jpn*, *US*, and *Viet.*

Ph. Eur. 6.2 (Charcoal, Activated). It is obtained from vegetable matter by suitable carbonisation processes intended to confer a high adsorption power. A black, light powder free from grittiness. Practically insoluble in all usual solvents. It adsorbs not less than 40% of its own weight of phenazone, calculated with reference to the dried substance. Store in airtight containers.

USP 31 (Activated Charcoal). The residue from the destructive distillation of various organic materials, treated to increase its adsorptive power. A fine, black, odourless, tasteless powder, free from gritty matter. The USP 31 has tests for adsorptive power in respect of alkaloids and dyes.

Adverse Effects and Precautions

Activated charcoal is relatively non-toxic when given by mouth but gastrointestinal disturbances such as vomiting, constipation, or diarrhoea have been reported. It may colour the faeces black. Activated charcoal should be used with caution in patients at risk of gastrointestinal obstruction as it may reduce gastrointestinal motility.

Haemoperfusion with activated charcoal has produced various adverse effects including platelet aggregation, charcoal embolism, thrombocytopenia, haemorrhage, hypoglycaemia, hypocalcaemia, hypothermia, and hypotension.

Care is needed if activated charcoal is used in patients receiving specific oral antidotes such as methionine (see Interactions, below). As with any treatment given by mouth for poisoning the risk of aspiration should be considered in drowsy or comatose patients.

Effects on the gastrointestinal tract. Gastrointestinal adverse effects are the main complication of oral activated charcoal. Vomiting may occur and is a risk factor for pulmonary aspiration (see Effects on the Lungs, below). Although some preparations may cause diarrhoea, activated charcoal may reduce gastrointestinal motility and multiple doses have been associated with intestinal obstruction or faecal impaction,[1-4] in some cases leading to ulceration[5] or perforation;[6] overdosage with drugs that reduce gastrointestinal motility may increase the risk.[2,3,6] Two cases of pseudo-obstruction, one of which was fatal, have also been reported[7] after the use of activated charcoal and sorbitol with opioid sedation for theophylline poisoning. In another report,[8] severe peritonitis developed in a patient given oral activated charcoal following gastric lavage; charcoal was found in the peritoneum, although the site of perforation could not be detected. Acute appendicitis has also been reported after multiple doses of activated charcoal.[9]

1. Watson WA, *et al.* Gastrointestinal obstruction associated with multiple-dose activated charcoal. *J Emerg Med* 1986; **4:** 401–7.
2. Anderson IM, Ware C. Syrup of ipecacuanha. *BMJ* 1987; **294:** 578.
3. Ray MJ, *et al.* Charcoal bezoar: small-bowel obstruction secondary to amitriptyline overdose therapy. *Dig Dis Sci* 1988; **33:** 106–7.
4. Atkinson SW, *et al.* Treatment with activated charcoal complicated by gastrointestinal obstruction requiring surgery. *BMJ* 1992; **305:** 563.
5. Mizutani T, *et al.* Rectal ulcer with massive haemorrhage due to activated charcoal treatment in oral organophosphate poisoning. *Hum Exp Toxicol* 1991; **10:** 385–6.
6. Gomez HF, *et al.* Charcoal stercolith with intestinal perforation in a patient treated for amitriptyline ingestion. *J Emerg Med* 1994; **12:** 57–60.
7. Longdon P, Henderson A. Intestinal pseudo-obstruction following the use of enteral charcoal and sorbitol and mechanical ventilation with papaveretum sedation for theophylline poisoning. *Drug Safety* 1992; **7:** 74–7.
8. Mariani PJ, Pook N. Gastrointestinal tract perforation with charcoal peritoneum complicating orogastric intubation and lavage. *Ann Emerg Med* 1993; **22:** 606–9.
9. Eroglu A, *et al.* Multiple dose-activated charcoal as a cause of acute appendicitis. *J Toxicol Clin Toxicol* 2003; **41:** 71–3.

Effects on the lungs. Vomiting after oral activated charcoal for acute poisoning has been associated with pulmonary aspiration of gastric contents, sometimes with fatal results.[1-3] Vomiting may be related to the formulation used and may be increased with sorbitol-containing preparations,[4] although a study in children[5] failed to confirm this. The use of a cuffed endotracheal tube has been recommended for any patient with impaired laryngeal reflexes to prevent aspiration;[3] however, there have been reports of aspiration despite a protected airway, including a case of obstructive laryngitis in a child.[6] Acute[7] and chronic[8] pulmonary toxicity has also been reported after accidental administration of charcoal into the lung due to misplacement of the nasogastric tube.

1. Harsch HH. Aspiration of activated charcoal. *N Engl J Med* 1986; **314:** 318.
2. Menzies DG, *et al.* Fatal pulmonary aspiration of oral activated charcoal. *BMJ* 1988; **297:** 459–60.
3. Rau NR, *et al.* Fatal pulmonary aspiration of oral activated charcoal. *BMJ* 1988; **297:** 918–19.
4. McFarland AK, Chyka PA. Selection of activated charcoal products for the treatment of poisonings. *Ann Pharmacother* 1993; **27:** 358–61.
5. Osterhoudt KC, *et al.* Risk factors for emesis after therapeutic use of activated charcoal in acutely poisoned children. *Pediatrics* 2004; **113:** 806–10.
6. Donoso A, *et al.* Activated charcoal laryngitis in an intubated patient. *Pediatr Emerg Care* 2003; **19:** 420–1.
7. Harris CR, Filandrinos D. Accidental administration of activated charcoal into the lung: aspiration by proxy. *Ann Emerg Med* 1993; **22:** 1470–3.
8. Graff GR, *et al.* Chronic lung disease after activated charcoal aspiration. *Pediatrics* 2002; **109:** 959–61.

Interactions

Activated charcoal has the potential to reduce the absorption of many drugs from the gastrointestinal tract and simultaneous oral therapy should therefore be avoided. In the management of acute poisoning, concurrent medication should be given parenterally. Care is needed if a specific oral antidote such as methionine is given since adsorption of the antidote may decrease its effectiveness; it has been recommended that activated charcoal should be cleared from the stomach or avoided if oral antidotes are to be used.

Uses and Administration

Activated charcoal can adsorb a wide range of plant and inorganic poisons and many drugs including salicylates, paracetamol, barbiturates, and tricyclic antidepressants; when given orally it reduces their systemic absorption from the gastrointestinal tract and is therefore used in the treatment of acute oral poisoning. It is of no value for poisoning by strong acids, alkalis, or other corrosive substances and its adsorptive capacity is too low to be of use in poisoning with iron salts,

cyanides, lithium, malathion, clofenotane, and some organic solvents such as methyl alcohol or ethylene glycol. Adsorption characteristics can be influenced by the charcoal's particle size, thus different responses may be obtained with different preparations.

Activated charcoal is given by mouth usually as a slurry in water. A usual adult dose for reduction of absorption is 50 g, but higher doses have been used. Children 1 to 12 years old may be given 25 to 50 g and infants under 1 year 1 g/kg. For maximum efficacy, activated charcoal should be given as soon as possible (within 1 hour) after ingestion of the toxic compound. However, it may be effective several hours after poisoning with certain drugs that slow gastric emptying. In the case of drugs that undergo enterohepatic or enteroenteric recycling (e.g. phenobarbital and theophylline) *repeated doses* of activated charcoal are of value in enhancing faecal elimination. Adult doses for repeated administration in active elimination have varied but typically 50 g may be given every 4 hours or 25 g every 2 hours. Doses in children and infants are similar to those used above for reduction of absorption and may be given every 4 to 6 hours. Administration may also be via a nasogastric tube.

Mixtures such as 'universal antidote' that contained activated charcoal, magnesium oxide, and tannic acid should not be used; activated charcoal alone is more effective and tannic acid may cause hepatotoxicity.

In treatment of poisoning using charcoal haemoperfusion, activated charcoal is used to remove drugs from the bloodstream. Where available, it may be of value in acute severe poisoning by drugs such as the barbiturates, glutethimide, or theophylline when other intensive measures fail to improve the condition of the patient.

Activated charcoal is used in dressings for ulcers and suppurating wounds (p.1585) to reduce malodour and may improve the rate of healing.

Activated charcoal has been used as a marker of intestinal transit and has also been tried in the treatment of flatulence. Both activated charcoal and vegetable charcoal (wood charcoal; carbo ligni) are included in preparations for various gastrointestinal disorders.

Technical grades of activated charcoal have been used as purifying and decolorising agents, for the removal of residual gases in low-pressure apparatus, and in respirators as a protection against toxic gases.

Administration. Activated charcoal is most commonly given as a slurry in water but this is often unpalatable because of the colour, gritty taste, lack of flavour, and difficulty in swallowing.[1] Flavourings and other excipients are often added in an attempt to improve palatability, although the effect of any additives on the adsorptive capacity of charcoal needs to be considered. Studies *in vitro* or in healthy subjects indicated that some foods such as ice cream, milk, and cocoa might inhibit the adsorptive capacity of activated charcoal, whereas starches and jams appeared to have no effect.[2,3] Carmellose has improved palatability although it might also reduce adsorptive capacity.[4-6] Saccharin sodium, sucrose, or sorbitol may be suitable additives,[7] although there may be problems associated with sorbitol-containing products (see under Poisoning, below). Chocolate syrup has also been used but the sweetness and flavour may disappear after a few minutes of contact with the activated charcoal.[1] A more recent study[8] of charcoal use in children with suspected poisoning found no evidence that use of flavourings improved the success of administration.

1. Scholtz EC, *et al.* Evaluation of five activated charcoal formulations for inhibition of aspirin absorption and palatability in man. *Am J Hosp Pharm* 1978; **35:** 1355–9.
2. Levy G, *et al.* Inhibition by ice cream of the antidotal efficacy of activated charcoal. *Am J Hosp Pharm* 1975; **32:** 289–91.
3. De Neve R. Antidotal efficacy of activated charcoal in presence of jam, starch and milk. *Am J Hosp Pharm* 1976; **33:** 965–6.
4. Mathur LK, *et al.* Activated charcoal–carboxymethylcellulose gel formulation as an antidotal agent for orally ingested aspirin. *Am J Hosp Pharm* 1976; **33:** 717–19.
5. Manes M. Effect of carboxymethylcellulose on the adsorptive capacity of charcoal. *Am J Hosp Pharm* 1976; **33:** 1120, 1122.
6. Mathur LK, *et al.* Effect of carboxymethylcellulose on the adsorptive capacity of charcoal. *Am J Hosp Pharm* 1976; **33:** 1122.
7. Cooney DO. Palatability of sucrose-, sorbitol-, and saccharin-sweetened activated charcoal formulations. *Am J Hosp Pharm* 1980; **37:** 237–9.
8. Osterhoudt KC, *et al.* Activated charcoal administration in a pediatric emergency department. *Pediatr Emerg Care* 2004; **20:** 493–8.

Poisoning. The management of acute poisoning is discussed on p.1435. The use of a *single oral dose* of activated charcoal has become a widespread method of preventing the absorption of ingested compounds and may be superior to gastric emptying. The American Academy of Clinical Toxicology (AACT) and the European Association of Poisons Centres and Clinical Toxicologists (EAPCCT) consider[1] that activated charcoal may be used if a patient presents within 1 hour of ingesting a potentially toxic amount of a poison known to be adsorbed by charcoal. There are insufficient data to support general use beyond 1 hour after ingestion.[1-3] In addition, *multiple oral doses* of activated charcoal have been found to enhance the elimination of some drugs and toxic substances even after systemic absorption. Mechanisms by which activated charcoal may increase drug elimination from the body include interruption of the enterohepatic circulation of drugs excreted into the bile, reduction of the reabsorption of drugs which diffuse or are actively secreted into the intestines, and increased elimination of the drug via the gastrointestinal tract when given with a laxative to decrease gastrointestinal transit time, although the practice of using charcoal with a laxative has been questioned.[4-6] Repeated oral doses of activated charcoal may therefore be considered for compounds that undergo enterohepatic or enteroenteric circulation, have a small volume of distribution, are not extensively bound to plasma proteins, and have a low endogenous clearance. Following a review of the literature[6] the AACT and EAPCCT recommended that multiple doses of charcoal should be considered only if a patient has ingested a life-threatening amount of carbamazepine, dapsone, phenobarbital, quinine, or theophylline. Anecdotal reports and studies in acutely poisoned patients indicate that a technique of giving multiple doses of charcoal may offer an alternative to charcoal haemoperfusion or haemodialysis. However, while activated charcoal is generally well tolerated, major complications do occasionally occur, including pulmonary aspiration and bowel obstruction.[7] Also, use of multiple doses of charcoal preparations containing sorbitol or sodium bicarbonate can result in increased vomiting[8] or in electrolyte disturbances.[4,9,10]

1. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Position paper: single-dose activated charcoal. *J Toxicol Clin Toxicol* 2005; **43:** 61–87. Also available at: http://www.clintox.org/Pos_Statements/SingleDoseActivatedCharcoal.pdf (accessed 27/09/05)
2. Green R, *et al.* How long after drug ingestion is activated charcoal still effective? *J Toxicol Clin Toxicol* 2001; **39:** 601–5.
3. Cooper GM, *et al.* A randomized clinical trial of activated charcoal for the routine management of oral drug overdose. *QJM* 2005; **98:** 655–60.
4. Neuvonen PJ, Olkkola KT. Oral activated charcoal in the treatment of intoxications: role of single and repeated doses. *Med Toxicol* 1988; **3:** 33–58.
5. Neuvonen PJ, Olkkola KT. Effect of purgatives on antidotal efficacy of oral activated charcoal. *Hum Toxicol* 1986; **5:** 255–63.
6. American Academy of Clinical Toxicology; European Association of Poisons Centres and Clinical Toxicologists. Position statement and practice guidelines on the use of multi-dose activated charcoal in the treatment of acute poisoning. *J Toxicol Clin Toxicol* 1999; **37:** 731–51. Also available at: http://www.clintox.org/Pos_Statements/MultipleDoseActivatedCharcoal.pdf (accessed 27/09/05)
7. Palatnick W, Tenenbein M. Activated charcoal in the treatment of drug overdose: an update. *Drug Safety* 1992; **7:** 3–7.
8. McFarland AK, Chyka PA. Selection of activated charcoal products for the treatment of poisonings. *Ann Pharmacother* 1993; **27:** 358–61.
9. McLuckie A, *et al.* Role of repeated doses of oral activated charcoal in the treatment of acute intoxications. *Anaesth Intensive Care* 1990; **18:** 375–84.
10. Tenenbein M. Multiple doses of activated charcoal: time for reappraisal? *Ann Emerg Med* 1991; **20:** 529–31.

HAEMOPERFUSION. Haemoperfusion involves the passage of blood through an adsorbent material such as activated charcoal or synthetic hydrophobic polystyrene resins that can retain certain drugs and toxic agents. Early problems with charcoal haemoperfusion such as charcoal embolism, marked thrombocytopenia, fibrinogen loss, and pyrogen reactions have been largely overcome by purification procedures and by coating the carbon with biocompatible polymers. However, transient falls in platelet count, leucocyte count, and circulatory concentrations of clotting factors, calcium, glucose, urea, creatinine, and urate have been reported during haemoperfusion. While there is no substitute for supportive measures, haemoperfusion can significantly reduce the body burden of certain compounds with a low volume of distribution within 4 to 6 hours in some severely poisoned patients; haemoperfusion is not effective for drugs or poisons with very large volumes of distribution.

References.

1. Winchester JF. Dialysis and hemoperfusion in poisoning. *Adv Ren Replace Ther* 2002; **9:** 26–30.

Porphyria. Activated charcoal may be used as part of the management of erythropoietic protoporphyria, one of the non-acute porphyrias (p.1448). It acts as a sorbent in the gut lumen, interrupting the enterohepatic recycling of protoporphyrin. It may also have a role in congenital erythropoietic porphyria, a very rare porphyria. In a patient[1] with photomutilation due to this condition, activated charcoal 30 g given orally every 3 hours for 36 hours reduced the plasma-porphyrin concentration to normal values by 20 hours and was more effective than colestyramine or red cell transfusion. After discontinuation of activated charcoal, plasma-porphyrin concentrations rose rapidly to near pretreatment levels within 10 days. Long-term treatment with oral charcoal over a 9-month period produced a clinical remission with low concentrations of plasma and skin porphyrin and an absence of photocutaneous activity. The optimal dose was determined to be 60 g three times daily. However, exacerbation after an initial period of remission has been reported in another patient[2] and total lack of efficacy in a third.[3]

Activated charcoal has also been tried in variegate porphyria, but a study[4] in 8 patients found that oral charcoal led to clinical and biochemical deterioration with an increase in skin lesions and in urinary and plasma porphyrins.

1. Pimstone NR, *et al.* Therapeutic efficacy of oral charcoal in congenital erythropoietic porphyria. *N Engl J Med* 1987; **316:** 390–3.
2. Hift RJ, *et al.* The effect of oral activated charcoal on the course of congenital erythropoietic porphyria. *Br J Dermatol* 1993; **129:** 14–17.
3. Minder EI, *et al.* Lack of effect of oral charcoal in congenital erythropoietic porphyria. *N Engl J Med* 1994; **330:** 1092–4.
4. Hift RJ, *et al.* Administration of oral activated charcoal in variegate porphyria results in a paradoxical clinical and biochemical deterioration. *Br J Dermatol* 2003; **149:** 1266–9.

Pruritus. Activated charcoal has been tried in pruritus (p.1582) associated with renal failure. In a double-blind crossover study,[1] activated charcoal 6 g daily by mouth for 8 weeks was more effective than placebo in relieving generalised pruritus in 11 patients undergoing maintenance haemodialysis. Another study[2] found that activated charcoal completely relieved pruritus in 10 of 23 haemodialysis patients, with a partial response in a further 10; treatment was generally well tolerated.

1. Pederson JA, *et al.* Relief of idiopathic generalized pruritus in dialysis patients treated with activated oral charcoal. *Ann Intern Med* 1980; **93:** 446–8.
2. Giovannetti S, *et al.* Oral activated charcoal in patients with uremic pruritus. *Nephron* 1995; **70:** 193–6.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Mamograf; Minicam Carb; ***Austral.:*** Ad-Sorb; Carbosorb; Charcocaps; Karbons†; ***Austria:*** Norit; Norit-Carbomix; ***Belg.:*** Norit; Norit-Carbomix; ***Braz.:*** Neocarbon†; ***Canad.:*** Charac; Charcodote Aqueous; ***Cz.:*** Carbosorb; Norit; ***Fin.:*** Carbomix; ***Fr.:*** Alione Charbon; Carbactive; Carbomix; Carbonet; Charbon de Belloc; Colocarb; Formocarbine; Splenocarbine; Toxicarb; ***Ger.:*** Kohle-Compretten; Kohle-Hevert; Kohle-Pulvis; Kohle-Tabletten; Ultracarbon; ***Gr.:*** Carbomix†; Norit; Toxicarb; ***Hong Kong:*** Charcodote; ***Indon.:*** Bekarbon; ***Irl.:*** Carbomix; Carbonet; ***Israel:*** Norit; ***Ital.:*** Carbomix; ***Mex.:*** Carbotural; ***Neth.:*** Norit; ***Norw.:*** Kohle-Compretten; ***NZ:*** Carbosorb X; ***Port.:*** Askina Carbsorb†; Norit; ***Singapore:*** Aqueous Charcodote; Ultracarbon; ***Spain:*** Arkocapsulas Carbon Vegetal; Ultra Adsorb; ***Swed.:*** Carbomix; Kolsuspension; Medikol; ***Thai.:*** Ca-R-Bon; Ultracarbon; ***Turk.:*** Charflo Aqua; ***UK:*** Actidose-Aqua; Bragg's Medicinal Charcoal; Carbomix; Carbonet; Charcodote; Clinisorb; Legius; Lyofoam C; Modern Herbals Trapped Wind & Indigestion; Norit; ***USA:*** Actidose-Aqua; Charcoaid; Charcoal Plus; Charcocaps; Liqui-Char.

Multi-ingredient: ***Arg.:*** Carbogasol; Carbon Tabs; Diarrocalmol; Estreptocarbocaftiazol; Karbonetas; Lefa Enteril†; Opocarbon; ***Austral.:*** Carboflex†; Carbosorb S; No Gas†; ***Austria:*** Eucarbon; Eucarbon Herbal; Intestinol; Sabatif; ***Belg.:*** Carbobel; Carbolactanose; ***Canad.:*** Carboflex†; Charac Tol; Charcodote; ***Chile:*** Carbon Sulfaguanidina; ***Cz.:*** Carbocit; Carbotox; ***Fr.:*** Acticarbine; Actisorb Ag ; Carboflex; Carbolevure; Carbophos; Carbosylane; Carbosymag; Notgaz; ***Ger.:*** Actisorb Silver†; ***Gr.:*** Carbosylane; ***India:*** Distenil; Molzyme†; Nutrozyme; Papytazyme; Unienzyme; ***Irl.:*** Actisorb Silver; ***Israel:*** Carbosylane; Charcodote; Eucarbon; Kaltocarb; Novicarbon; ***Ital.:*** Actisorb Plus; Carbone Composto; Carbonesia; Carboyoghurt; Eucarbon; No-Gas; ***Malaysia:*** Eucarbon†; ***Mex.:*** Acilin; Dipecfur; ***NZ:*** Carbosorb S†; Carbosorb XS; ***Pol.:*** Rapacholin AC; Rapacholin C; ***Port.:*** Carboflex†; ***S.Afr.:*** Rubilax†; ***Switz.:*** Carbolevure; Carboticon; Carvon†; ***Thai.:*** Belacid; Bicobon†; Biodan†; Carbonpectate; Delta Charcoal; Papytazyme†; Pepsitase; Polyenzyme-I; ***Turk.:*** Charflo Sorbitol; Eucarbon; Intestinol; Karboseptin; ***UK:*** Acidosis; Actisorb Silver; Carbellon; ***USA:*** Actidose with Sorbitol; Flatulex; Poison Antidote Kit; ***Venez.:*** Carbargal; Carbargal con Atropina; Guanicar.

Amifostine *(BAN, USAN, rINN)*

Amifostiini; Amifostin; Amifostina; Amifostinum; Ethiofos; Gammaphos; NSC-296961; WR-2721. *S*-[2-(3-Aminopropylamino)ethyl] dihydrogen phosphorothioate.

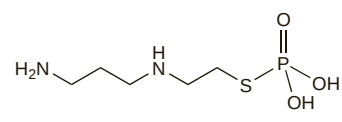
Амифостин

$C_5H_{15}N_2O_3PS$ = 214.2.

CAS — 20537-88-6 (amifostine); 63717-27-1 (amifostine monohydrate).

ATC — V03AF05.

ATC Vet — QV03AF05.



Pharmacopoeias. *US* includes the trihydrate.

USP 31 (Amifostine). The trihydrate is a white crystalline powder. Freely soluble in water. pH of a 5% solution in water is between 6.5 and 7.5. Store in airtight containers at a temperature of 2° to 8°. Protect from light.

Incompatibility. Amifostine has been reported[1] to be physically incompatible with aciclovir sodium, amphotericin B, cefoperazone sodium, chlorpromazine hydrochloride, cisplatin, ganciclovir sodium, hydroxyzine hydrochloride, miconazole,