There has been considerable concern over the systemic absorption of mercury from *dental amalgam*, which typically contains between 40 and 70% of mercury. However, the quantity absorbed from amalgam fillings is reported to be relatively small[9,10] and current evidence suggests that the use of dental amalgam for tooth restoration is both safe and effective.[11-14] The main risks appear to be occupational exposure of dental staff and environmental pollution. Some patients with hypersensitivity to mercury (manifest most commonly as local lichenoid reactions) may benefit from removal of amalgam fillings.[15-17]

Ethylmercury is contained in *thiomersal*, which is used as a preservative in some vaccines for infants and children, thus representing a potential source of mercury exposure. The safety of these vaccines has been a matter of considerable debate worldwide for many years although it is now usually accepted that there is no evidence of neurotoxicity. However, some countries have phased out vaccines containing thiomersal in favour of alternative preservatives. For further details and references, see Thiomersal p.1664.

The symptoms of *acrodynia* have been mistaken for those of phaeochromocytoma.[18-21]


1. Bluhm RE, *et al.* Elemental mercury vapour toxicity, treatment, and prognosis after acute, intensive exposure in chloralkali plant workers part I: history, neuropsychological findings and chelator effects. *Hum Exp Toxicol* 1992; **11:** 201–10.
2. Bluhm RE, *et al.* Elemental mercury vapour toxicity, treatment, and prognosis after acute, intensive exposure in chloralkali plant workers part II: hyperchloraemia and genitourinary symptoms. *Hum Exp Toxicol* 1992; **11:** 211–15.
3. dell'Omo M, *et al.* Long-term toxicity of intravenous mercury injection. *Lancet* 1996; **348:** 64.
4. Zippel D, *et al.* Quicksilver cholecystitis. *Ann Intern Med* 2006; **144:** 941.
5. Grandjean P, *et al.* Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury. *Neurotoxicol Teratol* 1997; **19:** 417–28.
6. Murata K, *et al.* Delayed brainstem auditory evoked potential latencies in 14-year-old children exposed to methylmercury. *J Pediatr* 2004; **144:** 177–83.
7. Grandjean P, *et al.* Cardiac autonomic activity in methylmercury neurotoxicity: 14-year follow-up of a Faroese birth cohort. *J Pediatr* 2004; **144:** 169–76.
8. Myers GJ, *et al.* Prenatal methylmercury exposure from ocean fish consumption in the Seychelles child development study. *Lancet* 2003; **361:** 1686–92.
9. Eley BM. The future of dental amalgam: a review of the literature. Part 3: mercury exposure from amalgam restorations in dental patients. *Br Dent J* 1997; **182:** 333–8.
10. Eley BM. The future of dental amalgam: a review of the literature. Part 4: mercury exposure hazards and risk assessment. *Br Dent J* 1997; **182:** 373–81.
11. FDI/WHO. Consensus statement on dental amalgam. *FDI World* 1995; **4** (July/Aug): 9–10.
12. Eley BM. The future of dental amalgam: a review of the literature. Part 6: possible harmful effects of mercury from dental amalgam. *Br Dent J* 1997; **182:** 455–9.
13. Bellinger DC, *et al.* Neuropsychological and renal effects of dental amalgam in children: a randomized clinical trial. *JAMA* 2006; **295:** 1775–83.
14. DeRouen TA, *et al.* Neurobehavioral effects of dental amalgam in children: a randomized clinical trial. *JAMA* 2006; **295:** 1784–92.
15. Ibbotson SH, *et al.* The relevance and effect of amalgam replacement in subjects with oral lichenoid reactions. *Br J Dermatol* 1996; **134:** 420–3.
16. McGivern B, *et al.* Delayed and immediate hypersensitivity reactions associated with the use of amalgam. *Br Dent J* 2000; **188:** 73–6.
17. Laeijendecker R, *et al.* Oral lichen planus and allergy to dental amalgam restorations. *Arch Dermatol* 2004; **140:** 1434–8.
18. Henningsson C, *et al.* Acute mercury poisoning (acrodynia) mimicking pheochromocytoma in an adolescent. *J Pediatr* 1993; **122:** 252–3.
19. Velzeboer SCJM, *et al.* A hypertensive toddler. *Lancet* 1997; **349:** 1810.
20. Wößmann W, *et al.* Mercury intoxication presenting with hypertension and tachycardia. *Arch Dis Child* 1999; **80:** 556–7.
21. Torres AD, *et al.* Mercury intoxication and arterial hypertension: report of two patients and review of the literature. Abstract: *Pediatrics* 2000; **105:** 627. Full version: http://pediatrics.aappublications.org/cgi/reprint/105/3/e34.pdf (accessed 14/07/06)


**Effects on the kidneys.** The kidneys are one of the primary sites for the accumulation of mercury in the body. All forms of mercury (liquid mercury, inorganic mercury, and organic mercury) may be toxic to the kidney although the inorganic forms are the most nephrotoxic.[1]


1. Zalups RK. Molecular interactions with mercury in the kidney. *Pharmacol Rev* 2000; **52:** 113–43.


**Hypersensitivity.** Systemic contact dermatitis in a patient apparently sensitised by red, mercury-based tattoo pigments, developed when he ate raw swordfish and alfonsino, both renowned for high mercury levels.[1]


1. Tsuruta D, *et al.* A red tattoo and a swordfish supper. *Lancet* 2004; **364:** 730.


### Treatment of Adverse Effects

The treatment of **acute** mercury toxicity depends on the form of mercury, the route of exposure, and the dose. Supportive measures may be needed with all types of toxicity. Ingestion of elemental mercury seldom requires active treatment since it is poorly absorbed by this route, although inhalation or aspiration of elemental mercury vapour is a potentially serious problem. Poisoning due to organic mercury is difficult to treat and supportive measures are the mainstay of treatment. Acute exposure to mercury by injection requires mainly supportive therapy, although excision of the affected area has been recommended after subcutaneous or intramuscular injection. Mercurials on the skin should be removed by copious washing with soap and water; for contamination in the eye, irrigation with water at room-temperature for at least 15 minutes should be performed.

Acute oral poisoning due to inorganic mercury salts should be treated if appropriate by activated charcoal or gastric lavage to reduce absorption if within 1 hour of ingestion of a potentially life-threatening dose. Decontamination is not usually necessary after ingestion of small amounts such as elemental mercury from a thermometer. Some inorganic mercury compounds may be corrosive to the gastrointestinal tract when ingested, which can make gastrointestinal decontamination problematic; induction of emesis is not recommended. Gastric decontamination may be tried for organic mercury compounds, although the benefits are uncertain.

In severe cases of toxicity with all forms of mercury poisoning, chelation therapy may be required to facilitate the removal of mercury from the body. Unithiol (p.1468) is the chelating agent of choice in the UK and should be considered in symptomatic patients with a blood-mercury concentration of 100 micrograms/litre, and in asymptomatic patients with a blood-mercury concentration of 200 micrograms/litre. Other chelating agents that may be used are succimer (p.1466), dimercaprol (p.1444), and penicillamine (p.1456). The treatment of choice may vary in other countries depending on local policies and availability of the above chelating agents. Penicillamine may be associated with more adverse effects than other chelating agents and should be reserved for use when others are unavailable or not tolerated. Dimercaprol should be avoided in poisoning with metallic mercury or methylmercury because it may exacerbate neurological effects through redistribution of mercury to the brain from other sites.

Some centres start haemodialysis early in the course of treatment; others wait until renal failure develops. Giving a thiol resin complex to prevent the reabsorption of mercury from the bile has also been tried.

The management of **chronic** toxicity is generally symptomatic although chelation therapy has been used in some patients if the blood-mercury concentration is raised or the patient is symptomatic.

◊ References.


1. Kostyniak PJ, *et al.* Extracorporeal regional complexing haemodialysis treatment of acute inorganic mercury intoxication. *Hum Exp Toxicol* 1990; **9:** 137–41.
2. Ferguson L, Cantilena LR. Enhanced mercury clearance during hemodialysis with chelating agents. *Clin Pharmacol Ther* 1991; **49:** 131.
3. Florentine MJ, Sanfilippo DJ. Elemental mercury poisoning. *Clin Pharm* 1991; **10:** 213–21.
4. Bluhm RE, *et al.* Elemental mercury vapour toxicity, treatment, and prognosis after acute, intensive exposure in chloralkali plant workers part I: history, neuropsychological findings and chelator effects. *Hum Exp Toxicol* 1992; **11:** 201–10.
5. Toet AE, *et al.* Mercury kinetics in a case of severe mercuric chloride poisoning treated with dimercapto-1-propane sulphonate (DMPS). *Hum Exp Toxicol* 1994; **13:** 11–16.
6. Houeto P, *et al.* Elemental mercury vapour toxicity: treatment and levels in plasma and urine. *Hum Exp Toxicol* 1994; **13:** 848–52.
7. Aaseth J, *et al.* Treatment of mercury and lead poisonings with dimercaptosuccinic acid and sodium dimercaptopropanesulfonate: a review. *Analyst* 1995; **120:** 853–4.
8. Isik S, *et al.* Subcutaneous metallic mercury injection: early, massive excision. *Ann Plast Surg* 1997; **38:** 645–8.
9. Baum CR. Treatment of mercury intoxication. *Curr Opin Pediatr* 1999; **11:** 265–8.
10. Satar S, *et al.* Intoxication with 100 grams of mercury: a case report and importance of supportive therapy. *Eur J Emerg Med* 2001; **8:** 245–8.
11. Risher JF, Amler SN. Mercury exposure: evaluation and intervention — the inappropriate use of chelating agents in the diagnosis and treatment of putative mercury poisoning. *Neurotoxicology* 2005; **26:** 691–9.


### Pharmacokinetics

There is little absorption of elemental mercury from globules in the gastrointestinal tract. The main hazard of elemental mercury is from absorption after inhalation of mercury vapour; this mercury is widely distributed before being oxidised to the mercuric form. Concentrations can be detected in the brain.

Soluble inorganic mercuric salts are absorbed from the gastrointestinal tract and can also be absorbed through the skin. The mercury is distributed throughout the soft tissues with high concentrations in the kidneys; it is mainly excreted in the urine and faeces with an elimination half-life of about 60 days, although it may take years to eliminate mercury from the brain; elimination from other tissues can take several months.

Organic alkyl mercury compounds are more readily absorbed from both the gastrointestinal and the respiratory tracts. They are widely distributed and can produce high concentrations in the brain. Alkyl mercury compounds are excreted in urine and in the faeces with extensive enterohepatic recycling. The biological half-life varies but is longer than that of inorganic mercury.

Organic mercury, and to some extent inorganic mercury, diffuse across the placenta and are distributed into breast milk.

### Uses and Administration

The hazards associated with mercury generally outweigh any therapeutic benefit and its clinical use has largely been abandoned. The use of mercurial diuretics such as mersalyl (p.1333) has generally been superseded by other diuretics. Ointments containing mercurials, such as ammoniated mercury (p.1604) have also generally been replaced by less toxic preparations. Mercurials were formerly used as spermicides.

Some ionisable inorganic mercury salts and certain organic compounds of mercury have been used as disinfectants, and some mercury salts are effective parasiticides and fungicides. Organic mercurials such as phenylmercuric acetate, borate, and nitrate are also used as preservatives (p.1657). Mercury is a component of dental amalgams.

Other mercury salts that have been used for their antibacterial activity include mercuric chloride, yellow mercuric oxide, and mercurous chloride (above).

**Homoeopathy.** Mercury and some mercury compounds have been used in homoeopathic medicines under the following names:

- Mercury: Hydrargyrum metallicum; Mercurius vivus; Merc. viv.
- Mercuric cyanide: Hydrargyrum bicyanatum; Mercurius cyanatus
- Mercuric iodide: Mercurius iodatus flavus; Merc. i. f.
- Red mercuric iodide: Hydrargyrum biiodatum; Mercurius bijodatus; Mercurius iodatus ruber; Merc. i. r.
- Mercuric nitrate: Hydrargyrum nitricum oxydulatum; Mercurius nitricus oxydulatus; Mercurius nitricus; Merc. nit.
- Red mercuric sulfide: Hydrargyrum sulfuratum rubrum; Cinnabaris; Cinbar.
- Ammoniated mercuric nitrate: Mercurius solubilis; Mere. sol.
- Potassium mercuric iodide: Mercurius et kali iodatus; Merc. ki.

See also Ammoniated Mercury (p.1604), Mercuric Chloride (above), and Mercurous Chloride (above).

### Preparations

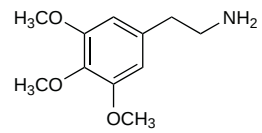
**Proprietary Preparations** (details are given in Part 3)
***Arg.:*** Lagrimas de Santa Lucia†.

**Multi-ingredient:** ***Austria:*** Coldophthal; ***Belg.:*** Ocal; ***Spain:*** Oftalmol Ocular.

## Mescaline

Mescalina; Mescalinum; Meskaliini; Meskalin. 3,4,5-Trimethoxyphenethylamine.

$C_{11}H_{17}NO_3$ = 211.3.
*CAS — 54-04-6.*




NOTE. The following terms have been used as 'street names' (see p.vi) or slang names for mescaline or peyote:
Bad seed; Beans; Big chief; Black button; Blue caps; Britton; Buttons; Cactus; Cactus buttons; Cactus head; Chief; Crystal; Dusty; Full moon; Green button; Half moon; Hikori; Hikula; Hikuli; Hyatari; Indians; M; Mesc; Mesca; Mescal; Mescalito; Mescap; Mescy; Mese; Mess; Mezc; Moon; Musk; Nubs; P; Peyote; Peyotl; Pixie sticks; San Pedro; Seni; Shaman; Topi; Tops.

### Profile

Mescaline is an alkaloid obtained from the cactus *Lophophora williamsii* (*Anhalonium williamsii*, *A. lewinii*) (Cactaceae), which grows in the northern regions of Mexico. The cactus is known in those areas by the Aztec name 'peyote' or 'peyotl' and dried slices of the cactus are called 'mescal buttons'.

Mescaline produces hallucinogenic and sympathomimetic effects similar to those produced by lysergide (see p.2335), but it is less potent. Its effects last for up to 12 hours. It has no therapeutic use. Both Mexican and North American Indians have used peyote in religious ceremonies on account of its hallucinogenic activity.

**Botulism.** Peyote consumed during a ceremonial ritual was believed to have caused botulism in three men.[1] The sample was found to contain type B botulinum toxin when assayed.


1. Hashimoto H, *et al.* Botulism from peyote. *N Engl J Med* 1998; **339:** 203–4.


## Mesoglycan Sodium

Mesoglicano sódico.

### Profile

Mesoglycan sodium is a mucopolysaccharide complex (glycosaminoglycan) extracted from calf aorta, containing mainly suleparoid (heparan sulfate) (p.1406) and dermatan sulfate (p.1256). It has been claimed to have antithrombotic, antiplatelet, and antihyperlipidaemic properties.

◊ References.


1. Forconi S, *et al.* A randomized, ASA-controlled trial of mesoglycan in secondary prevention after cerebral ischemic events. *Cerebrovasc Dis* 1995; **5:** 334–41.