Chapter 53

CAMPHOR
(*Cinnamomum camphora* T. Nees & Eberm.)

HISTORY

In China, camphor has been used for centuries as an antiseptic, antipruritic, cold remedy, and abortifacient. *Linimentum camphoratum* was officially recognized as medicinal product in the first edition of the *US Pharmacopeia*, published in 1820. Up until the 1930s, camphor was used as a circulatory stimulant and analeptic. Reports on camphor toxicity were first reported in the English medical literature in the late 19th century.[1] Gastrointestinal irritation, seizures and altered consciousness were well-recognized complications of acute camphor poisoning in the early 20th century.[2] By 1954, over 130 cases of camphor poisoning had been reported in the medical literature, primarily involving the accidental ingestion of camphorated oil or camphor-containing cough and cold preparations.[3] These reports included approximately 20 deaths attributed to camphor intoxication.[4] In 1982, the US Food & Drug Administration (FDA) declared camphorated oil as "not generally recognized as safe," and the concentration of camphor in US over-the-counter products was limited to <11%. Camphor is no longer listed in the *US Pharmacopeia* or the National Formulary.

BOTANICAL DESCRIPTION

Common Name: Camphor Tree, Camphor Laurel

Scientific Name: *Cinnamomum camphora* T. Nees & Eberm. (*Camphora camphora* Karst., *Laurus camphora* L.)

Botanical Family: Lauraceae (laurel family)

Physical Description: The camphor tree is a broad-leafed evergreen in topical and subtropical regions of the world that grows to 50–100 ft (~15–30 m) in height. Young branches are green with tinges of red. The tops of the leaves are shiny with three distinct veins, whereas the underneath side of the leaves are somewhat pale. Crushing the leaves produces an aromatic odor. The flowers are yellow-white, borne in small inflorescences. The fruits are round, pea-sized berries attached to the branchlets by small, cup-like green cones. The immature fruits first turn red, and then turn black during maturation. The fruits contain a single seed.

Distribution and Ecology: This plant is indigenous to Taiwan, China, and Japan, but camphor is widely cultivated in subtropical regions of the world.

EXPOSURE

Sources

The traditional source of camphor is the wood of *Cinnamomum camphora*, an evergreen tree native to eastern Asia. Camphor from natural sources is the dextrorotatory isomer. Currently, the primary source of camphor is the steam distillation and crystallization of pinene, which is converted to camphene by treatment with acetic acid and nitrobenzene.

Medical Toxicology of Natural Substances, by Donald G. Barceloux, MD

Medicinal Uses

Traditional

Traditional uses of camphor include administration as an antiseptic, antipruritic, rubefacient, mild anesthetic, abortifacient, contraceptive, inhalant for cold preparations, and suppression of lactation. Rarely, this compound has been used as a homicidal or suicidal agent.

Current

The use of camphor is limited to inhalation of vaporized solutions and dermal application of topical compounds. Camphor is used as a rubefacient/liniment (0.1–3%), a constituent of embalming fluid, and a dental antiseptic (65% camphor, 35% parachlorophenol) for root canals. Frequently, dermal products containing camphor also contain other potentially toxic ingredients (phenol, eucalyptus oil, menthol). Topical products containing camphor include Campho-Phenique® antiseptic gel (10.8% camphor and 4.7% phenol; Bayer Consumer Products, Morristown, NJ) and Vicks Vaporub® (5% camphor, 5% turpentine oil, 2.75% levomenthol, 1.5% eucalyptus oil; Procter & Gamble Consumer Products, Cincinnati, OH). Commercial products that may contain camphor include lacquers, varnishes, plasticizers for cellulose esters and ethers, moth repellents, explosives and pyrotechnics, and preservatives for pharmaceuticals and cosmetics.

Regulatory Status

Camphorated oil typically contained 20% camphor weight/volume (w/v) in cottonseed oil. However, in 1982, the FDA banned the sale of over-the-counter products containing this concentration of camphor due to serious central nervous system (CNS) toxicity following accidental ingestion.[5] Camphor now appears in US over-the-counter topical preparations in concentrations ranging up to 11%.[6]

PRINCIPAL INGREDIENTS

Chemical Composition

Camphor (CAS RN: 76-22-2) is a cyclic ketone of the hydroaromatic terpene group. Figure 53.1 displays the chemical structure of this compound ($C_{10}H_{16}O$). Twenty grams of Vicks Vaporub® (i.e., about 5% camphor) contains about 1 g of camphor, whereas 10 mL of Campho-Phenique® (i.e., about 10.8% camphor) contains about 1 g of camphor. Oxygenated monoterpene compounds were the major ingredients of the headspace constituents of a fresh sample of a Japanese specimen of *C. camphora* with camphor and 1,8 cineole accounting for 70% and 7.5% of the total, respectively.[7] The sample also contained about 8% monoterpenes (camphene, α-pinene, limonene) and approximately 9% sesquiterpene hydrocarbons. Cinnamomin is a ribosome-inactivating protein (RIP) isolated from mature seeds of the camphor tree that belongs to the type II class of RIPs.[8] These toxic proteins consist of an A-chain with RNA *N*-glycosidase activity and a galactose-specific lectin B-chain. The seeds of *Cinnamomum camphora* also contain a type I RIP, camphorin, with a molecular mass of 23 kDa.[9] These ribosome-inactivating proteins consist of a single peptide chain with a molecular mass between 25–30 kD.

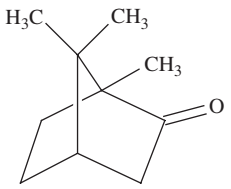

FIGURE 53.1. Chemical structure of camphor.

TABLE 53.1. Physiochemical Properties of Camphor

Physical Property	Value
Melting Point	180 °C/356 °F
Boiling Point	204 °C/399 °F
log P (Octanol-Water)	2.38
Water Solubility	1600 mg/L (25 °C/77 °F)
Vapor Pressure	0.65 mmHg (25 °C/77 °F)
Henry's Law Constant	8.10E-05 atm-m^3/mole (25 °C/77 °F)
Atmospheric OH Rate Constant	9.88E-12 cm^3/molecule-second (25 °C/77 °F)

Physiochemical Properties

Camphor is a translucent, crystalline solid at room temperature with a penetrating characteristic odor and a pungent, aromatic taste. Table 53.1 lists some of the physiochemical properties of camphor. Cinnamomin is a type II ribosome-inactivating protein, similar to other heterodimeric proteins (e.g., ricin) in this class. These 61 kD-proteins inhibit protein synthesis by acting as a RNA *N*-glycosidase to remove an adenine at A4324 in the sarcin/ricin domain (S/R domain) of the largest RNA in the 28S ribosome.[10] This domain is responsible for the interaction of elongation factors with ribosomes. Although cinnamomin A-chain inhibits protein synthesis similar to ricin, *in vitro* studies in BA/F3β cells

indicate that the cytotoxicity of cinnamomin is substantially lower than ricin.[8]

Mechanism of Toxicity

Following ingestion, camphor produces dose-dependent CNS excitation and gastrointestinal (GI) irritation. There are limited data on the toxic compounds in camphor, and the specific toxins (i.e., parent compound or toxic alcohol metabolite) in camphor that cause these CNS and GI effects remains unknown. Although cinnamomin is a type II ribosome-inactivating protein structurally and functionally similar to ricin, abrin, viscumin, ebulin 1, and nigrin b, the role of this protein in producing human toxicity remains undefined. Volunteer studies suggest that short-term exposure to vapors containing camphor stimulate cold receptors in the nose, but these vapors do not alter air flow through the nasal passages.[11]

DOSE RESPONSE

The ingestion of ≥1 g (5 mL of a 20% camphor solution) camphor by a child is a serious intoxication.[12] Camphor toxicity is unlikely to develop following the ingestion of ≤10 mg camphor/kg based a retrospective review of poison center data and the medical literature.[13] In a study from the 1920s of 80 postpartum women receiving camphor injections into engorged breasts, one women developed adverse reactions (nausea, vomiting) attributed to the treatment.[14] Adverse reactions were not reported for the rest of the subjects. The camphor regimen involved 195 mg camphor the first day, followed by three daily injections of 97 mg for a total camphor dose of 486 mg. Although minor GI symptoms may develop following the ingestion of 30–50 mg/kg, serious toxicity including convulsions is uncommon. The ingestion of 6–10 g camphor as a stimulant by two adults resulted in anxiety, agitation, and depersonalization.[15] There were no seizures. Major toxicity occurs following the consumption of camphor doses exceeding 150 mg/kg from preparations containing 20% camphor in cottonseed oil or camphor spirts (10% camphor in ethanol or isopropyl alcohol). The intentional ingestion of 68 mg camphor/kg (44 mL Campho-Phenique® – 10.8% camphor) by a 20-year-old man caused the onset of seizures within 10 minutes followed by vomiting and coma requiring intubation and respiratory support.[16] The patient recovered without sequelae within 24 hours. Another adult survived the ingestion of 42 g camphor with basic supportive care.[2] A survey of a camphor packaging facility indicated that exposure to ambient air concentrations exceeding 2 mg camphor/m^3 (2 ppm) causes mild to moderate eye, nose, and throat irritation.[17]

TOXICOKINETICS

Absorption

The absorption of camphor from the GI tract is relatively rapid based on limited data from volunteer studies and case reports. Seizures can develop within 5–10 minutes of the ingestion of camphor.[18] In fasted rats receiving one gram camphor as a 40% concentration of camphorated oil dissolved in cottonseed oil, the peak camphor concentration occurred about $1\frac{1}{2}$ hours after oral gavage.[19] The presence of food in the stomach delays absorption of camphor from the GI tract. Volunteer studies suggest that the systemic absorption of camphor from the skin is relatively low.[20]

Distribution

Camphor is highly tissue bound with a volume of distribution of approximately 2–4 L/kg.[21] This compound is widely distributed after absorption including diffusion into amniotic fluid, fetus, brain, liver, and kidney.

Biotransformation/Elimination

Based on animal studies, the biotransformation of camphor involves the oxidation of camphor to camphorols (2-hydroxycamphor, 3-hydroxycamphor, borneol) followed by conjugation of these alcohols with glucuronic acid.[22] These inactive glucuronide conjugates are excreted in the urine. Analysis of urine following the ingestion of camphor as a stimulant by two adults demonstrated hydroxylated metabolites (3-OH-, 5-OH-, 8-OH-, 9-OH-camphor), which subsequently underwent oxidation to the corresponding ketone and carbonic acid with the latter compounds excreted as a glucuronides.[15] Very small amounts of unchanged camphor appear in the urine after absorption. The plasma elimination half-life of camphor following oral administration of 200 mg camphor to two volunteers was approximately 1.5 and 2.5 hours.[21] The mean elimination serum half-life of camphor was approximately 2 hours in a group of 50 rats receiving one gram camphor as a 40% concentration of camphorated oil dissolved in cottonseed oil.[19] The lungs excrete small amounts of camphor, resulting in the characteristic pungent odor of exhaled breath.

CLINICAL RESPONSE

Acute Effects

The primary effects of camphor intoxication are gastrointestinal irritation, seizures, and CNS depression. As a

result of the rapid absorption of camphor, clinical effects begin soon (i.e., about 5–90 minutes) after ingestion.[23,24] The first symptoms of camphor intoxication occurred about 45 minutes after accidental ingestion of 1–1½ tablespoons of camphorated oil by a group of children (age 4–10 years).[25] Initially, local irritation of the GI tract occurred following ingestion, manifest by pain in the mouth and throat, nausea, vomiting, and a nonspecific feeling of warmth. Spontaneous nausea and vomiting occur in most patients ingesting more than a small amount of camphor. Other clinical features of camphor intoxication include headache, lightheadedness, anxiety, agitation, confusion, hallucinations, hyperreflexia, and myoclonus.[15,21,26] Toxicity following dermal application or inhalation is not usually associated with GI symptoms.[27] The odor of camphor may be present in the breath, vomitus, or urine, but the absence of this odor does not exclude camphor toxicity. Serious CNS effects include headache, confusion, vertigo, restlessness, delirium, hallucination, and muscle jerks that progress to tonic-clonic seizures.[28] Life-threatening effects include status epilepticus, coma, apnea, and pulmonary aspiration. Camphor is a local irritant of the eye and upper respiratory tract. Death from acute camphor ingestion is uncommon today,[29,30] but potentially fatal complications involve respiratory failure from status epilepticus or aspiration pneumonia. Symptoms usually resolve within 24 hours without sequelae.

Chronic Effects

Case reports have not associated camphor poisoning during pregnancy with teratogenesis or adverse neonatal effects. Serious camphor intoxication in a 32-year-old woman during the first trimester of pregnancy resulted in several seizures and altered consciousness.[31] Six months later she delivered a normal infant. Camphor is highly lipophilic, and this compound can cross the placenta. Although a neonatal death occurred shortly after the mother demonstrated signs of camphor poisoning, the role of camphor in the death of the child was unclear.[32] A 26-year-old mother with a history of preeclampsia delivered a normal infant one day after she demonstrated the effects of camphor intoxication (agitation, hyperreflexia, tremor, seizure, nausea, lethargy) following the accidental ingestion of 12 g camphorated oil.[33]

Authors have associated mild elevation of serum hepatic aminotransferases with the use of topical compounds containing camphor, but the contribution of camphor to these changes remains unclear.[34] A case report associated the development of acute hepatic encephalopathy (prodromal viral syndrome, diffuse interstitial infiltrates, rapid neurological deterioration, hypoglycemia, hepatomegaly) with the chronic administration of a home remedy containing camphor and whiskey to a 6-month child over 5 months.[35] Although certain features of this illness simulated Reye's syndrome, the liver histology did not demonstrate the pleomorphic, swollen mitochondria and loss of dense intramitochondrial bodies usually seen in fatal cases of Reye's syndrome.

DIAGNOSTIC TESTING

Analytical Methods

Near-infrared (NIR) technology is a sophisticated analytical method for the quality control of camphor-containing products that allows the rapid identification of individual components and the detection of adulteration.[36] Methods for quantitation of camphor in biological samples include reverse phase high performance liquid chromatography (RP/HPLC) with UV detection and gas chromatography with flame ionization detection (GC/FID).[37] The limits of detection and quantitation of camphor in plasma following analysis by GC/FID are about 1 ng/mL and 5 ng/mL, respectively.[38] The between-day coefficient of variation for camphor in this study was about 13.5%.

Biomarkers

There are few data correlating clinical effects with concentrations of camphor in blood. Consequently, the presence of camphor in blood samples confirms exposure, but these biomarkers are not usually available in clinical laboratories. Two adults developed agitation and anxiety after ingesting camphor about 2 hours prior to presentation to the emergency department. Their initial plasma samples contained 0.3 mg camphor/L and 0.4 mg camphor/L.[15] Approximately 3 hours after ingesting camphorated oil, the plasma camphor concentration in blood samples from a 60-year-old woman was 3.1 mg/L.[39] Prior to blood sampling, she had two grand mal seizures, and at the time the blood sample was drawn, she had stable vital signs and was comatose (i.e., responsive only to painful stimuli). Following the intentional ingestion about 44 mL Campho-Phenique® (68 mg camphor/kg) 5 hours prior to sampling, the urine sample from a 20-year-old man contained 1.5 µg/mL camphor as measured by GC.[16] The serum concentration of camphor 7 hours after the ingestion of Vicks Vaporub® by a 3-year-old girl was 1.95 mg/dL. Although the patient developed confusion, vomiting, and suffered a single seizure within 2 hours of the camphor ingestion, the

patient had stable vital signs and regular respirations at the time the blood sample was drawn.[40] By 21 hours after ingestion, camphor was not detectable in the serum as measured by gas liquid chromatography.

Abnormalities

The chest x-ray is usually normal unless aspiration occurred during a seizure or altered consciousness. Most cases of camphor poisoning are not associated with laboratory changes with the exception of laboratory abnormalities related to seizures or respiratory depression during serious camphor intoxication.[41,42] Although some case reports associated changes in serum hepatic enzymes with the absorption of camphor, the role of camphor in these changes is unclear and any observed hepatic changes are usually transient.

TREATMENT

Stabilization

Because of the potential for rapid absorption of camphor and the quick onset of seizures, intravenous access should be established immediately in any patient with serious camphor intoxication (>30–50 mg/kg, the presence of alteration of consciousness, severe vomiting, ataxia, or seizures). Vital signs including pulse oximetry should be monitored closely. Sinus tachycardia is a common effect of camphor intoxication, but hypotension and shock are rare complications of camphor poisoning. Seizures should be treated with the usual doses of intravenous benzodiazepines. The use of intravenous phenobarbital is a second-line anticonvulsant based on older experimental studies and limited case reports.[3]

Decontamination

Referral to a health care facility is appropriate for camphor ingestions exceeding 30 mg/kg.[43] Because of the rapid absorption of camphor and the potentially rapid onset of seizures, the use of decontamination measures is not recommended. Although the low molecular weight of camphor suggests good adsorption to activated charcoal, there are limited data on the efficacy of activated charcoal. *In vivo* experiments in rats suggest that camphor is not well adsorbed to activated charcoal at a charcoal to camphor ratio of 2:1.[19] However, this study used a suboptimal dose of charcoal rather than the typical charcoal to toxin ratio of 10:1. Skin contaminated with camphor should be copiously washed with soap and water to prevent local irritant effects, although system toxicity is not expected to occur following dermal exposure to camphor.

Enhancement of Elimination

Camphor is highly lipophilic, and the large volume of distribution suggests that measures to enhance the elimination of camphor are not effective. Very small amounts of camphor appear in the urine unchanged, and diuresis does not significantly increase the renal elimination of camphor. Although a few case reports suggest that lipid hemodialysis[44] or resin hemoperfusion[45] improve the CNS effects (hyperexcitability, increased neuromuscular activity, seizures, coma) associated with serious camphor intoxication, the clinical data documenting the efficacy of these procedures are lacking. In a 54-year-old woman with coma, seizures, and respiratory failure after ingestion of 10% camphor spirits, 4 hours of hemoperfusion with Amberlite XAD4® (Sigma-Aldrich, St. Louis, MO) removed only 35 mg camphor.[21] Her seizures stopped during hemoperfusion, but she remained deeply comatose. Four hours of charcoal hemoperfusion in a comatose 60-year-old woman removed only about 1% of the estimated internal dose of camphor.

Supplemental Care

Although most laboratory values remain normal during camphor poisoning, patients with seizures or clinically significant CNS depression should be evaluated for any acid–base, electrolyte, glucose, or hepatorenal abnormalities. There are no specific antidotes for camphor poisoning. Patients with seizures during camphor intoxication should be monitored for at least 24 hours. Symptoms of camphor toxicity usually develop within 3 hours of ingestion; therefore, asymptomatic patients with a history of camphor ingestion may be discharged after 3–4 hours of observation.[3] In symptomatic patients, recurrence of toxic effects is not expected once the symptoms resolve. These patients may be discharged when symptoms of toxicity resolve.

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