

# CAMPHORATED OIL: STILL ENDANGERING THE LIVES OF CANADIAN CHILDREN

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## Abstract • Résumé

Camphor is a volatile, aromatic compound familiar to many people as a principal ingredient in topical home remedies for colds. It is highly toxic when ingested. Although camphorated oil in concentrations of 11% or greater is no longer sold in the United States, preparations containing concentrations of up to 20% are still sold over the counter in Canada. The authors describe two children who suffered severe poisoning after accidental ingestion of a small amount of camphorated oil. Both children exhibited generalized tonic-clonic seizures with subsequent respiratory depression. Treatment was symptomatic, consisting of seizure control and respiratory assistance. The authors argue that because camphorated oil is of questionable benefit and poses a danger to the public it should be removed from the market.

Le camphre est un composé aromatique volatil que beaucoup de gens connaissent bien comme principal ingrédient des médicaments maison anti-rhume d'application externe. Il est très toxique lorsqu'il est ingéré. Même si l'huile camphrée dont la concentration est supérieure à 11 % n'est plus vendue aux États-Unis, des préparations contenant des concentrations qui peuvent atteindre 20 % sont toujours vendues sans ordonnance au Canada. Les auteurs décrivent les cas de deux enfants qui ont été victimes d'une intoxication grave après avoir ingéré accidentellement une faible quantité d'huile camphrée. Les deux enfants ont été victimes de crises de grand mal généralisées suivies d'une défaillance respiratoire. Le traitement symptomatique visait à lutter contre les attaques et à fournir de l'assistance respiratoire. Les auteurs soutiennent qu'il faudrait retirer l'huile camphrée du marché parce qu'elle présente un avantage douteux et qu'elle est dangereuse pour la population.

Camphor has been used for hundreds of years as an antiseptic, antipruritic, rubefacient, abortifacient, aphrodisiac, contraceptive and lactation suppressant.<sup>1,2</sup> Today, many people continue to use topical preparations containing camphor because they create a local sensation of heat and mild anesthesia and carry a penetrating, characteristic odour that is associated with "strong medicine."<sup>3</sup> Most familiarly, it is used in ointments, oils and inhalants for the home treatment of colds. Camphorated oil is a preparation of 19% or 20% camphor in a carrier oil and is used mainly as a chest rub to relieve cold symptoms.

The highly toxic effects of camphor have long been established. A statement reflecting the level of concern among pediatricians and poison centre staff with regard to the use of camphor was issued by the Committee on Drugs of the American Academy of Pediatrics in 1978.<sup>4</sup>

In 1983 the US Food and Drug Administration (FDA) ruled that the concentration of camphor in medicinal products must not exceed 11%.<sup>5</sup> Recently, the Committee on Drugs of the American Academy of Pediatrics readdressed the issue and expressed further concern about the continued use and toxicity of camphor even at concentrations below 11%.<sup>6</sup> The committee recommended that parents be made aware of common camphor-containing products and of the potential dangers of these products. Alternative therapeutic agents should be considered whenever camphor might commonly be used.

Meanwhile, camphorated oil in concentrations of up to 20% is still stocked in many Canadian drug stores. For some brands, the only warning about the hazardous nature of the product is the statement on the label, in small print, "For external use. Harmful if swallowed."

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This does little to communicate how toxic camphorated oil is: ingestion of as little as 5 mL can be enough to kill a child.<sup>7</sup> Because it is an over-the-counter product, no advice is given by a pharmacist at the time of purchase. To highlight the extreme danger associated with camphor, especially camphorated oil, we describe two children who ingested camphorated oil and were admitted to the Hospital for Sick Children, Toronto, during a single week in September 1994.

## CASE REPORTS

### CASE 1

A 3-year-old girl weighing 15 kg who had had a productive cough with clear sputum and nasal congestion for 1 week had been given chest rubs with 19% camphorated oil. While taking care of her two other children, the mother instructed her husband to give the child her camphorated oil. The father, mistakenly thinking that it was to be administered like cough syrup, gave the child one teaspoonful of the camphorated oil orally. Within 20 minutes the child had a generalized tonic-clonic seizure lasting 5 minutes that was followed by an irritable postictal state.

Upon arrival at a peripheral hospital the child experienced a second generalized tonic-clonic seizure. Her heart rate was 122 beats/min and her respiratory rate 8 breaths/min. The oxygen saturation was measured to be 97% with the child breathing room air, but the lips were noted to be cyanotic. She was treated with 5 mg diazepam intravenously and was given oxygen by mask, which resulted in a rapid improvement. After the generalized seizure the patient exhibited myoclonic movements and increased muscle tone but no focal neurologic signs.

The child was subsequently airlifted to our hospital for observation and further treatment. Upon arrival she was alert, happy and active. No neurologic sequelae were found on examination. Laboratory values, including blood counts, electrolyte concentrations and blood glucose concentrations, were normal.

### CASE 2

A 15-month-old boy weighing 10 kg had been receiving chest rubs with 20% camphorated oil for his cold. On one occasion, while his father was on the phone, the toddler, who was playing on the floor, opened the bottle of camphorated oil despite its "child-proof" cap and consumed approximately 20 mL of the oil. After checking the product label carefully, the father called the poison information service and was told to summon an ambulance immediately. Upon arrival of the ambulance, about 10 minutes later, the child had a gen-

eralized tonic-clonic seizure. Oxygen was given, and shortly afterward the seizure resolved.

The child was taken to a peripheral hospital, where he had a second generalized tonic-clonic seizure. He was given 2.5 mg diazepam intravenously, and the seizure subsided after 45 seconds. Vital signs assessed shortly after were stable: the temperature was 36.6°C, heart rate 140 beats/min, respiratory rate 28 breaths/min, blood pressure 118/60 mm Hg and oxygen saturation 98%. The child was irritable but consolable and responsive. Two and a quarter hours after the ingestion of the camphorated oil the child had a third generalized tonic-clonic seizure, which was treated with 3.5 mg diazepam intravenously. Subsequently the child's respiratory rate dropped, and oxygen was given by continuous positive airway pressure for a short time. He was given a loading dose of 15 mg/kg phenobarbital and was transferred to our hospital. Venous blood gases taken before transport while the child was breathing 100% oxygen were as follows: pH 7.24, partial pressure of carbon dioxide 47 mm Hg, partial pressure of oxygen 123 mm Hg, bicarbonate 20 mmol/L and base excess -7 mmol/L.

Upon arrival at our hospital the child had a blood pressure of 74/56 mm Hg, for which 200 mL of normal saline was given in rapid infusion. Oxygen saturation was 100% on respiratory assistance. The child responded to pain stimuli only. He localized pain and withdrew, but made no verbal response. Findings for the remaining physical examination were normal. The leukocyte count was  $18.2 \times 10^9/L$ , the hemoglobin level 101 g/L and the platelet count  $471 \times 10^9/L$ . The electrolyte concentrations were normal except for a potassium concentration of 3.3 mmol/L. The lactate dehydrogenase level was elevated at 258 U/L, the aspartate aminotransferase level was 37 U/L, the alanine aminotransferase level 32 U/L, the alkaline phosphatase level 278 U/L and the bilirubin level 4  $\mu\text{mol/L}$ .

The child was transferred to a stepdown unit and his condition closely monitored. A chest x-ray film taken 16 hours after the ingestion appeared normal. The patient remained drowsy for about a day and then recovered fully.

## COMMENTS

The toxic effects of camphor are well documented. By 1954 camphor poisoning had been reported more than 130 times in the medical literature<sup>8</sup> and camphorated oil was involved in many cases reported thereafter.<sup>2</sup> Frequently, it was ingested after being mistaken for castor oil, which was packaged similarly and has some similarity in spelling.<sup>9,10</sup> The highly toxic effects of camphor, the incidence of severe poisonings and the lack of any established role for camphorated oil in scientific medicine led the FDA to remove products containing more

than 11% camphor from the market in 1983.<sup>5</sup> Despite this measure, camphor intoxications continued to occur.<sup>3,6</sup> In Canada, camphorated oil in concentrations of up to 20% is still produced. It is sold in packaging that does not adequately distinguish it from other products and that does not give sufficient warning of its toxic nature. As a result, accidental ingestions are still causing life-threatening intoxications.

Camphor is rapidly absorbed when taken orally, but a considerable amount can also be absorbed via inhalation or through intact skin.<sup>11</sup> Typically, symptoms begin 5 to 90 minutes after ingestion of a toxic dose.<sup>1-3,12</sup> Seizures occurred as soon as 4 minutes after ingestion of 28 g of camphorated oil by a 4½-year-old girl.<sup>13</sup> The onset of seizures can be sudden and may be followed by postconvulsive depression and coma.<sup>1-3,10,14</sup> Death may result from respiratory depression or complications of status epilepticus.<sup>2,6</sup> Other neurologic symptoms include confusion, vertigo, restlessness, delirium and hallucinations. Increased muscular activity, tremors and jerky movements often progress to epileptiform convulsions.<sup>1-3,10,12,13,15</sup> Gastrointestinal symptoms occur in most patients and consist of oral and intestinal burning, nausea and vomiting.<sup>1-3,10,12,13,15</sup> Other clinical manifestations that have been reported are tachycardia, mydriasis, visual disturbances, urinary retention, albuminuria, mild transient elevations of the aspartate dehydrogenase and lactic dehydrogenase concentrations, and, rarely, hepatic failure.<sup>1-3,8,10</sup> Although a variety of other conditions or intoxications can exhibit similar symptoms, a strong odour of camphor on the breath or a history of recent treatments with camphor-containing agents will strongly suggest camphor intoxication.

It is crucial to realize that severe symptoms may arise very rapidly. Consequently, emergency transportation to a medical facility is warranted.<sup>3,12</sup> There are conflicting views as to the merits of attempting to prevent absorption. Siegel and Wason<sup>15</sup> suggested that ipecac be used to try to prevent absorption if more than 1 g is ingested, the patient is seen within 2 hours, and no symptoms are present. If symptoms are present and the patient is seen within 2 hours, they recommended the use of gastric lavage, charcoal and cathartics; if the patient is seen later they recommended the use of only charcoal and cathartics. This approach, however, is not supported by experimental data. Given that seizures can occur suddenly and without warning, others have recommended that the use of ipecac be abandoned and that gastric lavage be used with caution.<sup>2,3</sup> Activated charcoal has been given, but no study has demonstrated its effectiveness as a decontaminant in cases of camphor poisoning. Studies involving animals have shown no difference in the absorption of camphor when activated charcoal was used.<sup>16</sup>

Supportive care, usually focusing on prevention and control of seizures and on protection of the airway,

should be given as needed. Diazepam and phenobarbital have been recommended for seizure control.<sup>2,3,10,12,17</sup>

Camphor is metabolized in the liver and subsequently excreted in the urine as a pharmacologically inactive glucuronide compound. Enhancement of elimination in severely poisoned patients has been attempted with the use of lipid hemodialysis,<sup>17,18</sup> resin hemoperfusion<sup>13</sup> and activated charcoal hemoperfusion.<sup>19</sup> However, most of the camphor is distributed into body tissues; only a very small amount is present in the blood. Although all of these methods can remove the camphor from the blood as it passes through dialysis, they are not effective in removing camphor from the body. Mascie-Taylor, Widdop and Davison<sup>19</sup> reported that although charcoal hemoperfusion efficiently cleared the blood of camphor, the total amount of camphor removed was less than 1% of the ingested dose.<sup>19</sup> It must be concluded that such extracorporeal procedures are of limited benefit in serious cases of camphor poisoning.

Hence treatment is predominantly symptomatic. Very few means of causal treatment are available once a toxic dose of camphor has been ingested. Inhibition of absorption may be ineffective in most cases because camphor is rapidly absorbed, enhancement of elimination is prevented by the agent's highly lipophilic nature, and an antidote is not available. The only effective causal "treatment" is the prevention of accidental ingestion.

In Canada camphorated oil in concentrations of 19% and 20% is still available despite its documented high toxicity and unproven efficacy. Moreover, it is still inappropriately labelled: its high toxicity is not indicated, and it can easily be confused with castor oil or olive oil preparations that are similarly packaged and labelled. The crucial common features of the two cases we have described are the use of camphorated oil — an obsolete substance in modern evidence-based medicine — as a cold remedy and the parents' lack of awareness of the extreme oral toxicity of this product. How many of these life-threatening accidents will it take before camphorated oil is removed from the Canadian market?

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*After this case report was accepted for publication another child was admitted to our hospital with severe camphor poisoning. A 15-month-old girl had ingested an unknown amount of Vicks Vaporub (containing 4.73% camphor, 2.6% menthol and 1.2% eucalyptus oil). One and three-quarter hours after the ingestion the child had a generalized tonic-clonic seizure lasting 20 minutes. After she was treated with 1 mg diazepam rectally, the seizure subsided. Subsequent respiratory depression necessitated intubation and ventilation. Two further seizures were treated with lorazepam, phenobarbital and phenytoin. The child was admitted to our intensive care unit and eventually recovered fully. This additional case underlines the immense toxicity of camphor even when formulated in lower concentrations.*

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