

# POISONING BY THE VOLATILE OILS IN CHILDHOOD

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The volatile oils play an important part in accidental poisoning in childhood, as can be seen from the figures quoted in a recent review (Craig and Fraser, 1953). The purpose of the present paper is to draw attention to the dangers of this form of poisoning, on which the recent literature is scanty, to suggest measures for its treatment and control, and to consider any points which may be applicable to the wider field of poisoning as a whole.

Craig and Fraser showed that, of 502 cases of accidental poisoning in childhood which had occurred in Edinburgh and Aberdeen between 1931 and 1951, the common toxic agents were volatile oils in 74 cases, household disinfectants in 39, barbiturates in 36, kerosene in 31 and iron in 24. The group of acids and alkalis caused only 11 cases. Of 454 deaths from accidental poisoning in childhood which had occurred in Britain during a similar period, 54 were caused by the volatile oils, the next commonest being strychnine in 41, acids and alkalis in 39, iron in 31 and disinfectants in 28. Only 10 deaths were due to barbiturate poisoning. That the volatile oils should top both the morbidity and mortality figures is surprising, considering the relatively slight attention which has been paid to this group.

The 74 cases seen in hospital were made up of camphor 29, turpentine 25, sassafras 5, eucalyptus 4, methyl salicylate (oil of wintergreen) 3, others 8. The 54 deaths were due to methyl salicylate 36, camphor 12, turpentine 2, eucalyptus 1, others 3.

## Material

The present paper is based on the 74 cases of poisoning mentioned above. In 24 of these the records are either missing or so exiguous that analysis is unwarranted. The remaining 50 cases are otherwise unselected. Many of them are mild, but even these can contribute information on such points as the mode of ingestion and the first aid that was carried out.

## Camphor

Camphor is a crystalline ketonic substance, closely related to the terpenes, which is obtained by distillation from *cinnamomum camphorae*. As a by-product of this distillation, essential oil of camphor (ol. camph. rect.) is also obtained. The main constituent of this oil is cineole (35%), with safrole, camphor, phellandrene, pinene, etc. Camphorated oil, on the other hand, is pure camphor (20%) in a vegetable oil (arachis oil). It is thus not strictly a volatile oil, but is sufficiently closely related to be treated clinically as such. Spirits of camphor contain 10% of camphor.

The toxic effects of camphor are mentioned in pharmacological works of reference, but little material is given which particularly applies to the problem in childhood. Single cases in adults have been described by Haft (1925) and Klingensmith (1934). A case in a mentally defective boy of 10 years was described by Cottrell (1931). Benz (1919) briefly described the chaos which reigned in a children's dormitory after all the occupants had been dosed with camphorated oil instead of castor oil, but unfortunately this graphic report is not susceptible of scientific analysis. Single cases in childhood have been reported by Davies (1887), Barker (1910), Haas (1916), Clark (1924), Blair (1929) and others. In a general survey of poisoning, Rubin, Recinos, Washington and Koppanyi (1949) include some discussion of 14 cases of camphor poisoning and 11 of turpentine poisoning.

Case records are available in 19 patients with camphor poisoning, all of whom took camphorated oil, and these are summarized in Table 1.

Two cases are reported in detail:

CASE 2. A boy of 5 years was given one teaspoonful of camphorated oil instead of olive oil for a cold. This happened about 7.45 a.m. He complained of the nasty taste and the mother discovered her error, but did nothing about it. However, the boy took a good breakfast and went to school. Shortly after 9 a.m. he vomited and began

TABLE I  
POISONING BY CAMPHORATED OIL

Case No.	Age (years and months)	Amount Taken	If in Error, Given for	Vomiting	Emesis	Gastric Lavage	Pallor or Collapse	Convulsion	Stupor	Coma	Muscular Excitability	Ataxia	Mental Changes	Cyanosis
1	1 yr. 5	dr. 1	Castor oil	30+	—*	—	—	25	—	—	—	—	—	—
2	5 yr. 5	dr. 1	Olive oil	90	—	—	—	—	—	—	—	—	—	—
3	9	<oz. ½	Castor oil	—	—	45	—	—	—	—	—	—	—	—
4	2	?	'In error'	—	—	—	—	—	—	—	—	—	—	—
5	1 yr. 4	dr. ½	Glycerine	—	—	15	—	—	—	—	—	—	—	—
6	4½ yr.	oz. 1	Olive oil	10	6	—	—	4	—	—	—	—	—	—
7	1 yr. 11	?	—	70	—	100	65	60	—	—	—	—	—	—
8	2 yr.	dr. 2	—	5	—	—	—	—	—	—	—	—	—	—
9	1 yr. 8	?	—	—	—	—	—	—	—	—	—	—	—	—
10	1 yr. 1	?	—	—	—	—	—	—	—	—	—	—	—	—
11	2 yr. 11	dr. 1	Castor oil	5	—	50	—	—	—	—	—	—	—	—
12	2	?	Olive oil	—	—	—	—	—	—	—	—	—	—	—
13	2½ yr.	?	—	125	—	—	—	120	—	—	—	—	—	—
14	2 yr.	oz. ½	—	45	—	180	—	125	—	—	—	—	—	—
15	1 yr. 2	dr. 1	'Cough mixture'	—	—	60	—	?	—	—	—	—	—	—
16	1 yr. 4	dr. 1	—	35	32	—	—	30	—	—	—	—	—	—
17	1 yr. 4	dr. 1	'By mistake'	—	—	—	—	—	—	—	—	—	—	—
18	1 yr. 3	dr. 1	Cod liver oil	5	—	90	—	60	—	?	—	—	—	—
19	2 yr.	dr. 2	—	45	—	—	—	120	—	—	—	—	—	—

\* In the tables, — sign means only that the symptom was not mentioned in the case records. The dosages quoted are for the most part parental estimates, and in only a few cases should they be taken at their face value.

† Where numbers are quoted instead of — signs in the tables, they indicate the approximate time in minutes after ingestion at which a symptom appeared. Although some of these estimates must be inaccurate, they are correct in so far as they establish the sequence of events.

to stagger about the classroom. His legs seemed to be twitching. He did not talk, and seemed sleepy, though conscious. On admission, he was slightly cyanosed. He was confused but obeyed simple commands. He vomited again. No specific therapy was given and he was completely well within 24 hours.

CASE 6. At 8.40 a.m. a boy of 4 years was given one ounce of camphorated oil in mistake for olive oil. Four minutes later, he went into a generalized convulsion which lasted for two minutes. He then seemed comatose, but his mother induced vomiting by putting her finger down his throat, and he became brighter thereafter. He had a further two vomits before admission at 9.15 a.m. On admission, he seemed bright and interested, but his hands were jerking. Gastric lavage gave a return smelling strongly of camphorated oil. If left alone, the boy remained quiet, but he became very excited when disturbed. He was completely well within 24 hours.

It will be seen that in 11 of the 19 cases the camphor was administered by the parents. This proportion does not appear with any other toxic agent, and why it should happen with camphor, which has such a distinctive smell, is difficult to understand. If the cases in which the drug was given by the parents are excluded, the age incidence of camphor poisoning corresponds with that for poisoning as a whole.

Five patients in the present series developed no symptoms. It is of interest to note that four of these (Cases 3, 4, 10, and 12) were the youngest patients in the series. This raises the possibility that the youngest children splutter out most of what they are given because of the offensive taste, which in

turn raises the whole question of taste in relation to accidental poisoning, a matter which will be dealt with at greater length later. Death has, however, been recorded in the youngest age-group.

**Symptoms.** There are four cardinal signs in camphor poisoning. These are:—

(1) INCREASED MUSCULAR EXCITABILITY. Case 2 affords a good example of abnormal muscular activity occurring without a convulsion supervening, but for the most part a case which shows muscle jerking will also show convulsions. These movements may herald a convulsion, or may follow a convulsion and gradually die away without a further convulsion occurring. They interfere with voluntary activity.

(2) CONVULSIONS. Although it is an established fact that convulsions occur in camphor poisoning, it does not seem to be generally realized how common these convulsions are. It will be seen that nine (or possibly 10) patients of the present series had one convulsion or more.

Rubin *et al.* report convulsions in five of their 14 cases. A convulsion may be the first sign of poisoning, or it may occur after other symptoms have appeared. Camphor convulsions do not carry the serious prognosis of strychnine convulsions, as all of the patients who convulsed subsequently recovered. A large dose may be expected to produce a prompt convulsion (Case 6).

(3) VOMITING. This may be the only symptom, or it may precede or follow other symptoms. It may occur within five minutes of ingestion, or may be delayed for as much as 90 minutes. There is evidence

that delayed vomiting leads to serious symptoms, as, of the seven patients who did not vomit for 30 minutes or more after ingestion, six developed convulsions and the seventh showed marked jactitations (Case 2). It is obvious, however, that a patient who has not vomited may not produce any symptom. But if vomiting has occurred before the patient has been seen, and the vomiting has been delayed, then the case must be regarded as potentially serious.

(4) **MENTAL CHANGES.** These are difficult to assess in children, but terror (Klingensmith) and maniacal behaviour (Haft) have been recorded. Confusion and irritability are common.

Pallor or cyanosis and collapse may occur, but seem usually to be secondary to other symptoms. Death from camphor poisoning is said to occur commonly from respiratory failure. This may follow a series of convulsions (Barker) or may occur early (Blair). In Blair's case, respiratory failure occurred within 45 minutes of ingestion and the child was kept alive by artificial respiration for 30 minutes before spontaneous respiration was resumed, accompanied by tonic muscular contractions. On the other hand, death has apparently occurred in status epilepticus (Clark, Davies). The situation is paralleled in experimental work. The rats used by Grove (1910) died in respiratory failure, the guinea-pigs used by Carnot and Cairis (1914) in the prostration produced by status epilepticus. It appears that children may react in either way, which has to be considered in treatment. From the present series, respiratory failure seems to be rare, but this may only mean that the series is composed of mild cases.

Sequelae seem to be rare. The mentally defective boy described by Cottrell had marked meningitic signs for four days. Klingensmith's case was readmitted on the fourth day because of severe headache. All the cases in the present series appeared to recover rapidly, and none was readmitted.

**Treatment.** It is startling to observe that artificial emesis was induced by the parent in only two of the 19 cases, and in these two only after a convulsion had occurred. The need for speedy emesis was underlined above, when it was pointed out that the more severe symptoms appear regularly in those cases in which vomiting has been delayed. A practitioner

who has been 'phoned about a case of poisoning cannot assume that a parent will carry out the 'obvious' measure of stimulating vomiting.

Gastric lavage is also indicated, whether vomiting has occurred or not. Lavage in a patient liable to have a convulsion is not always a wise practice, but camphor, being in liquid form, is relatively easily removed from the stomach, and much may be gained from a successful lavage.

Many cases need otherwise only be treated on expectant lines. However, convulsions cannot be taken lightly. It is true that a convulsion is not a particularly grave prognostic sign, yet convulsions occurring with increasing frequency are ominous (Carnot and Cairis). On the other hand, sedation must be undertaken with care, because of the risk of eventual respiratory failure. Barker found chloral ineffective, but only gave one small dose. He was also unable to make full use of chloroform, as the patient's breathing deteriorated, cyanosis became marked, and the fits, though less frequent, were more severe. In a severely poisoned child any anaesthetic may act as a respiratory depressant except when given by the most skilled hands. The short-acting barbiturates, given intravenously, or paraldehyde given intramuscularly, seem to be the most rational form of treatment.

**Toxicity.** One drachm of camphorated oil has caused the death of a child of 16 months, and the same dose caused marked symptoms in a boy of 6, quoted in the present series. A convulsion occurred four minutes after a boy of 4 had taken one ounce, and this child must be considered fortunate because his mother at once induced vomiting, which was quickly followed by further spontaneous vomiting. A review of the present series and of the available literature suggests that 1 drachm is a dangerous dose for a toddler, and 2 drachms for a school child.

#### Eucalyptus

Single cases of eucalyptus poisoning have been described by Wood (1900), Myott (1906), Benjamin (1906), Orr (1906), Allan (1910), Garrett (1925), Sewell (1925) and Gibbin (1927). Kirkness (1910) described two cases. It is an uncommon poison in childhood, but death has been recorded. The principal findings in three further cases are summarized in Table 2.

One case is reported in detail.

TABLE 2  
POISONING BY EUCALYPTUS OIL

Case No.	Age (years and months)	Amount Taken	Vomiting	Emesis	Gastric Lavage	Respiratory Signs	Colour	Stupor	Shock	Vertigo	Ataxia	Myosis
1	3 yrs.	oz. 1	90	—	120	—	Flush	100	—	?	90	—
2	4 yrs.	?	—	15	60	—	Flush	—	—	10	10	—
3	7	dr. 1	—	—	30	—	Pale	5-10	—	—	—	—

CASE 3. A boy, aged 7 months, was given a teaspoonful of oil of eucalyptus at 8.15 a.m. He coughed and choked, and some of the oil was spilled. A few minutes later he became drowsy. When first seen, 25 minutes after ingestion, he was pale and collapsed, with rapid, shallow respirations, and a rapid and feeble pulse. The limbs were flaccid, the reflexes present but weak. The pupils were pin-point, with no reaction to light. Rhonchi were heard at both bases. The stomach was washed out, the return smelling strongly of eucalyptus. By 11 a.m. his breathing had improved, but there was considerable exudation in the respiratory tract, and the back of the throat required frequent swabbing. His temperature rose to 103.4° F. By 4 p.m. the child was showing spontaneous movement, and was beginning to cry. He had a stool smelling strongly of eucalyptus at 11 p.m. After 24 hours the respiratory tract was almost dry, the temperature normal, the general state good. The pupils were no longer pin-point, but were not reacting well to light. The odour of eucalyptus did not leave the breath until 72 hours after ingestion. He was discharged on the sixth hospital day.

**Symptoms.** There are three major manifestations of eucalyptus poisoning:

(1) A DEPRESSANT EFFECT. A toxic dose of eucalyptus usually leads to drowsiness in a few minutes, and the patient may be deeply unconscious in a quarter of an hour. Vertigo and ataxia were observed in two of our patients, neither of whom became shocked, and it seems that these symptoms are indicative of a relatively mild degree of poisoning, the more serious cases being quickly overcome by stupor. This is borne out by Gibbin's patient who stated that he had experienced epigastric pain, nausea, weakness in the legs, giddiness, cold sweats, rigors and headaches before becoming unconscious. A constricting type of abdominal pain seems common.

(2) RESPIRATORY SYMPTOMS. Although Case 3 of the present series resembled Gibbin's case in having shallow respirations, the majority show stertorous breathing. That the moisture in the respiratory tract is probably a direct effect of the eucalyptus is suggested by the work of Boyd and Pearson (1946), who showed experimentally on a number of mammals that eucalyptus or oil of turpentine instilled into the stomach significantly increased the output of respiratory tract fluid. They further showed that there was an optimal dose (about 50 mg. kg.) in producing this effect, and that a larger dose led to an actual fall in the effect. This may explain the variability of the appearance of respiratory symptoms in poisoned children.

(3) MYOSIS. Pin-point pupils have frequently been recorded in eucalyptus poisoning. This is of some importance, as the conjunction of pin-point pupils and stertorous breathing might suggest

morphine poisoning, as has been pointed out by Wood, or poisoning by amidone or an organic phosphate. On the other hand, the pupils may be dilated and the breathing shallow (Kirkness). However, the smell of eucalyptus should make the diagnosis obvious.

Urinary symptoms have been reported.

**Treatment.** Emesis should be induced and gastric lavage carried out. As there is a central depression of respiration, it seems logical to use a stimulant such as nikethamide, although it should be used with care if the eucalyptus has been taken in liniment which also contains camphor.

Due attention should be paid to the respiratory tract, as Myott's patient died of respiratory complications, the lungs being grossly congested and friable at necropsy. Excess mucus should be removed from the respiratory passages, and oxygen given where necessary. Antibiotics should be given prophylactically.

**Toxic Dose.** The toxic dose of oil of eucalyptus is difficult to determine. An adult male died after taking 6 drachms (Myott), and the baby who has just been described was severely ill after taking less than one teaspoonful. However, one must remember that all the children brought to hospital with reputed eucalyptus poisoning developed symptoms, and the same applies to sassafras poisoning. This is not at all usual with other poisons, as many of the patients show no signs or symptoms of having actually taken the drug. The conclusion to be drawn is that even a few drops of eucalyptus or sassafras may be sufficient to cause symptoms. As symptoms may rarely take as long as two hours to appear (Case 1), it would seem advisable to keep all cases of reputed eucalyptus poisoning under observation for that time, unless gastric lavage has been carried out within a short time of ingestion without eucalyptus being detected in the washings.

#### Sassafras

Oil of sassafras is sometimes used against headache, although its popularity is waning. It also exists in a synthetic form, which may be expected to have the same properties as the natural substance. Oil of sassafras is 80% safrol, with small amounts of pinene, phellandrene and camphor. Crude oil of camphor may be divided into a light fraction, from which medicinal camphor is prepared, and a heavy fraction with a high safrol content, from which synthetic oil of sassafras is derived. One of the patients in the present series (Case 4) ingested the synthetic form.

The literature on sassafras poisoning is scanty. The U.S. Dispensary records the case of a young

man who ingested one teaspoonful of oil of sassafras, developed vomiting and collapse with dilated pupils, passed into stupor, and died (Cincinnati Lancet-Clinic, December, 1888). Five cases are now reported, the results being summarized in Table 3.

All five children took the oil themselves, so no reliance can be placed on the estimated amount taken. It is probable that all the figures are over-estimates.

**Symptoms.** The U.S. Dispensary records that experimental work, indicating that sassafras kills by a central paralysis of respiration preceded by a greatly depressed circulation, has been done by Heffter (1894).

Symptoms first appear within 10 to 90 minutes of taking the oil. The clinical picture in many ways resembles that of eucalyptus poisoning, except that vomiting and signs of shock are more common in sassafras poisoning, while myosis and respiratory symptoms were not apparent in the five cases here discussed. All the patients vomited, all but one showed signs of shock, three of the five showed vertigo, two were stuporose and one was aphasic.

**Treatment.** The use of emetics is well exemplified in Case 2. The mother sought the help of the police as soon as the accident occurred, and the police gave repeated emetics until the vomitus no longer smelt of oil. This therapy, more energetic than is applied to the majority of poisoning cases before referral to

hospital, may well have averted serious symptoms, as the child was stuporose within 10 minutes of ingestion. Once in hospital, the child responded dramatically to an injection of nikethamide.

**Toxic Dose.** As an adult male has died after taking one teaspoonful of oil of sassafras, it is probable that a few drops would be sufficient to kill a toddler. It seems remarkable that the Registrar General's returns for the last 20 years contain no record of a death from sassafras poisoning.

**Turpentine**

Oil of turpentine (spirits of turpentine) is composed largely of terpenes. Its main constituent is pinene, and from pinene there can be derived synthetic camphor. Rectified oil of camphor contains small quantities of safrol, pinene and phellandrene, all of which are also found in oil of sassafras. Such links are common throughout the volatile oil series, and are mentioned here to show why turpentine is best considered as a poison in its relation to the other volatile oils.

Recent reports of the toxic effects of oil of turpentine have largely referred to its use in industry and to its absorption through the skin. Single cases of ingestion have been described by Grapel (1901), Stanwell (1901) and Harbeson (1936). Sixteen further cases are summarized in Table 4.

TABLE 3  
POISONING BY OIL OF SASSAFRAS

Case No.	Age (years and months)	Amount Taken	Vomiting	Emesis	Gastric Lavage	Pallor	Shock	Stupor	Vertigo	Aphasia
1	2 yr.	dr. $\frac{1}{2}$	60	—	120	—	—	—	—	—
2	1 yr. 2	dr. 2	15	5	—	15	—	10	—	—
3	1 yr. 9	oz. 2	< 30	—	> 60	< 60	—	30	< 60	—
4	2 $\frac{1}{2}$ yr.	dr. 2	120	*F	140	120	120	—	90	90
5	1 yr. 3	dr. 2	65	—	120	60	—	—	65	—

\* F = emesis attempted but failed.

TABLE 4  
POISONED BY OIL OF TURPENTINE

Case No.	Age (years and months)	Quantity	Choking = Respiratory Signs	Vomiting	Emesis	Gastric Lavage	Shock	Stupor	Convulsion	Ataxia	Urine	Pyrexia
*1	5	dr. 1	—	—	F+	60	—	—	—	—	—	—
2	1 yr. 7	< oz. 3	—	—	F	190	—	90	—	—	R.B.C.s	—
3	1 yr. 9	oz. 2	—	5	3	—	—	60	—	—	Smell Few R.B.C.s	—
*4	2 yr.	oz. $\frac{1}{2}$	—	10	—	—	—	—	—	—	—	—
5	2 yr.	?	—	—	—	30	—	—	—	—	—	—
6	2 yr. 10	?	—	—	—	—	—	—	—	—	—	—
7	1 yr. 2	oz. 4	—	—	F	430	—	240	550	240	—	—
8	1 yr. 1	oz. 1	—	—	—	20	—	—	—	—	—	—
*9	5 yr. 7	?	—	5	3	—	—	—	—	—	—	—
10	2 yr. 8	?	—	—	3	—	—	—	—	—	—	—
11	1 yr. 1	?	—	245	240	15	—	—	180	—	—	—
12	1 yr. 8	?	—	30	—	35	35	—	—	—	—	—
13	1 yr. 1	?	—	—	—	30	—	—	—	—	—	—
14	1 yr.	?	—	—	—	—	—	—	—	—	—	—
15	3 yr.	oz. 1	—	5	—	15	—	—	—	—	—	—
16	1 yr. 3	oz. 3	—	10	—	90	—	—	—	—	R.B.C.s	—

\* Cases 1, 4, and 9 took 'turpentine substitute' which contains hydrocarbons derived from petroleum.

† F = emesis attempted but failed.

One case is described in detail.

CASE 7. A girl of 14 months was reputed to have drunk 4 ounces of turpentine. The mother attempted to induce vomiting by giving salt and water, but failed. The child then gradually 'became drunk'. She was sleepy, and would neither sit nor stand. The child also seemed 'jumpy, and likely to have a convulsion'. She was admitted to hospital six hours after ingestion, when her temperature was 101.6° F., pulse 140, respiration 50. The child was sleeping, but twitching markedly. Gastric lavage was done, and the result smelt strongly of turpentine. The pulse rose to 170, the respirations to 70, and the child then had a convulsion lasting for 15 minutes. She was stuporose and screaming for half-an-hour after the convulsion. She was well within 24 hours, and no sequelae appeared.

In view of the relative mildness of most cases of turpentine poisoning, Harbeson's case is also described. This was a child of 11 months who was given two teaspoonfuls of spirits of turpentine as an anthelmintic. She had a convulsion a few hours later and became comatose, with cyanosis, hyperpyrexia and marked tachypnoea and tachycardia. She died a few hours after the convulsion without having regained consciousness.

**Symptoms.** Turpentine produces a wide range of toxic effects. It is difficult to describe a 'classical case' of turpentine poisoning. Symptoms which may appear are:

(1) **VOMITING.** This occurred spontaneously in only four of the 16 cases.

(2) **STUPOR.** This appears to be the commonest of the more severe symptoms. It occurred in six patients, four of whom had not vomited spontaneously and five of whom had not had vomiting induced or gastric lavage before the symptom supervened.

(3) **CONVULSIONS.** These are not commonly described as signs of turpentine poisoning, yet a convulsion occurred in Harbeson's case and in two children in the present series. Both of the latter had previously had a febrile convulsion, and the inference seems to be that turpentine has a mild convulsant action which may cause a convulsion in a pre-disposed child, even in the absence of pyrexia. Convulsions do not seem to occur till some hours after ingestion.

(4) **IRRITATION OF URINARY TRACT.** It is well known that the urine smells of violets in turpentine poisoning (and also in eucalyptus poisoning). This sign was observed in only one of the 16 cases, and excess of red cells was observed in three only.

(5) **RESPIRATORY SYMPTOMS.** There was a history of choking at the time of ingestion in seven cases, and respiratory symptoms appeared in two.

Pyrexia, ataxia, shock, tachycardia, sweating and leucocytosis may also appear.

In their 11 cases, Rubin *et al.* found coma or drowsiness in six, fever in four, vomiting in four, and pneumonia in one, findings which correspond well with my own.

**Treatment.** Treatment in the first instance is by emesis and gastric lavage. Case 7 shows that it is almost true to say that 'it is never too late to wash out the stomach', as turpentine was found there six hours after ingestion.

The treatment of stupor which is liable to be punctuated by a spontaneous convulsion is something of a problem. However, convulsions do not seem to lead to death, whereas coma may, so that careful use of stimulants seems to be indicated. Other symptoms seem to be so uncertain in their appearance that treatment can only be symptomatic.

**Toxicity.** In Harbeson's case, 2 drachms caused the death of a 7-month-old baby. In only one case of the present series (Case 1) is the dose known with any accuracy, the parent having given turpentine instead of gripe water. One drachm was sufficient to cause stupor in a 5-month-old baby, which roughly coincides with Harbeson's case. One ounce produced very severe urinary symptoms in a woman of 20 years, the urine still smelling of violets 24 days after ingestion (Grapel). A woman of 46, who had her stomach washed out within 15 minutes of taking 10 ounces of turpentine in a suicide attempt, developed no symptoms (Stanwell). It is noteworthy that her breath did not smell of turpentine and that the lavage was undertaken initially as a means of diagnosis. This small clinical point deserves attention.

It seems likely that 1-2 drachms would be necessary to produce severe symptoms in a toddler, which presumably accounts for the mildness of turpentine poisoning relative to that of the other volatile oils.

#### **Methyl Salicylate (Oil of Wintergreen)**

Oil of wintergreen (ol. gaultheria, lin. meth. sal.) is almost unique among the volatile oils in that it is very nearly a pure substance, containing 98% methyl salicylate. Methyl salicylate is converted in the stomach to methyl alcohol, which is absorbed as such, and salicylic acid, which is absorbed after being converted to sodium salicylate in the intestine. The methyl alcohol is seldom produced in sufficient quantities to play much part in the picture of poisoning, the effect produced being that of salicylism, as 75% of the methyl salicylate is available as salicylate. A teaspoonful of oil of wintergreen will supply 45 grains of salicylate, obviously enough to endanger the life of a small child, particularly as

young children have a notoriously poor tolerance for salicylates. The symptoms produced by oil of wintergreen are thus quite different from those due to other volatile oils.

The literature on methyl salicylate poisoning is copious. Reviews have been published by Stevenson (1937), Macready (1943), and Mouren (1949), and the biochemistry of salicylate poisoning has been thoroughly investigated by Erganian, Forbes and Case (1947). Nevertheless, three further cases are now reported, as post-mortem examination was carried out in one, and as attention should be drawn to this extremely dangerous poison in the British literature.

In two of these three cases, no symptoms appeared. The children were aged  $1\frac{1}{2}$  and 6 years. Gastric lavage was carried out in one within 20 minutes of ingestion. In the other, the precise time of lavage is uncertain, but it was certainly less than one hour after ingestion. The gastric contents in both cases smelt strongly of oil of wintergreen. Both children were admitted to hospital, but remained perfectly well. The third case presents a striking contrast.

**CASE REPORT.** At 11 o'clock one morning, 34 hours before admission to hospital, a girl aged 25 months drank oil of wintergreen. The precise amount was unknown, but it was less than half an ounce. Castor oil was given. She remained well until five hours after ingestion, when she began to vomit and had two loose stools. She then became irritable and developed dyspnoea, but she had had a cold for two days and these signs were not considered serious. She was given a further dose of castor oil, but during the night she vomited repeatedly and had several loose motions. About 17 hours after ingestion, she was pale and dyspnoeic and did not recognize her mother. By the time she was admitted, she was flaccid and deeply unconscious. Her breathing was markedly acidotic, and she showed occasional muscle twitchings (temperature  $100^{\circ}$  F., pulse 160, respiration 40). There were no added signs to be heard in the chest although her own doctor had noted bronchitic signs before sending her in. An intravenous drip of glucose was set up, but the child died soon after, her temperature rising to  $106^{\circ}$  F. before her death, which occurred 36 hours after ingestion. No biochemical studies were made.

At necropsy Dr. Kenneth Rhaney found numerous submucous haemorrhages in the stomach.

The spleen was considerably enlarged, pale grey and soft, with very prominent Malpighian bodies. Microscopically, some of the Malpighian bodies showed necrosis and hyaline material in their centres. The pulp contained an excess of polymorph leucocytes.

There was oedema of the bases of the lungs.

The liver was slightly enlarged, soft, pale orange-yellow with a mosaic of delicate brown lines, apparently the result of severe fatty change. Microscopically, there was a moderate degree of fatty change in all parts of the lobules, most severe in the peripheral zone. Many of the cells had pyknotic nuclei.

In the kidney there was moderately severe venous congestion. Microscopically, the cells of the convoluted tubules and the ascending limbs of Henle's loops showed widespread cloudy swelling, with narrowing or obliteration of the lumen. Many cells had separated one from another and some had been desquamated. Urine taken at necropsy gave a positive Gerhardt test and a negative Rothera test, and contained sheets of cells, but no casts.

The changes were consistent with chemical poisoning.

**Symptoms.** It is not intended to give here a detailed description of the signs of salicylism. On the other hand, it is desirable to stress the particular danger of wintergreen poisoning. The poison is a rare one, yet, over the last 20 years, it is second only to strychnine as a lethal poison in childhood.

There is usually vomiting shortly after ingestion, although this was absent in the case described above. There then ensues a period of several hours during which there may be no untoward signs (Stevens and Kaplan, 1945). There may be mild subjective symptoms during this period (Crossland, 1948), but not such as a toddler is likely to explain in words. After this delay, the signs of severe acidosis complicated by liver damage gradually appear, and the patient is likely to die unless energetic therapy is undertaken.

Dérobot and Gascoin (1950) described a child in whom the march of symptoms was more rapid. He was put to bed after his midday meal within reach of a bottle of oil of wintergreen, and at 2.30 was found pale and reeking of wintergreen. Copious vomiting was immediately induced, but by 4 p.m. he showed muscular jerking, respiratory embarrassment, cyanosis of the whole body, profuse sweating, cold extremities, a rapid pulse, projecting eyes and strabismus. He was sent at once to hospital, but died within 45 minutes of admission. There was no smell of wintergreen in the gastric washings, but there were large quantities in the urine at necropsy. Although it is impossible to work out a precise timetable for the early part of the history, it seems that this child probably took a very large quantity of wintergreen and, although all that was in the stomach was removed by emesis within an hour of ingestion, enough had passed into the intestine to cause rapid death. A similar case is described by Gaburro (1952).

**Treatment.** Emesis should be induced pending gastric lavage, in which sodium bicarbonate should not be used as it is said to increase the solubility of methyl salicylate (Aikman, 1949). At the first sign of acidosis intravenous therapy should be begun, glucose being given to counter the toxic effect of salicylate on the liver and M-6 sodium R-lactate to

counter the acidosis. Therapy can be controlled by studying the CO<sub>2</sub> combining power of the blood. Vitamins K and C should also be given. If pulmonary signs appear, antibiotics should be exhibited.

Methyl salicylate poisoning is a pitfall for the inexperienced resident medical officer. He should remember that a clear return from gastric lavage does not necessarily preclude severe toxic symptoms if the child has already vomited, or if the poison has been taken some time before. He must also remember that a child who has no abnormal signs four hours after ingesting wintergreen may be beyond medical aid a few hours later. All reputed cases of wintergreen poisoning must be admitted to hospital.

#### Other Volatile Oils

The volatile oils so far discussed include all those known to have caused deaths in Great Britain during the last 20 years, except for oil of citronella, which has been responsible for one fatality. Oil of chenopodium has caused six deaths in children in Padua in the last 10 years (Gaburro). There remain to be mentioned four cases in which the oils were taken in compound form.

CASE 1. A girl of 20 months took an unknown quantity of camphor and eucalyptus. She was admitted to hospital 90 minutes later, showing pallor, stupor and collapse, but made a good recovery. This cannot be taken to indicate that camphor and eucalyptus together invariably combine to produce a depressant effect, as the relative amounts of the two oils taken in this case were unknown.

CASE 2. A girl of 35 months swallowed some 'three oils', vomited twice in the next half hour, and immediately thereafter began to have a series of short convulsions. She then became comatose, and remained drowsy the next day, but was well in 48 hours. 'Three Oils' has no fixed formula, and the oils may be chosen from arachis, olive, cajuput, eucalyptus, camphor, lavender, turpentine, amber, red oil, etc. In the present case the main constituent seems to have been camphor, but possibly a depressant oil, even eucalyptus, was also present.

CASE 3. A boy who swallowed 'chilblain liniment' had his stomach washed out within 15 minutes and developed no symptoms.

CASE 4. A boy of 2½ years swallowed an unknown amount of linimentum A.B.C. When admitted, he was pale, twitching and delirious, with widely dilated pupils which did not react to light. He recovered.

This case has been included with the liniments only as a warning, as the symptoms are primarily those of aconite poisoning. One teaspoonful of linimentum A.B.C. contains approximately 1/60 gr. of aconite (toxic dose for an adult, 1/300 gr.), and gr. 1/16 belladonna alkaloids, in addition to camphor and chloroform. The extreme toxicity of this preparation is obvious.

#### Discussion

The volatile oils are not chemically pure substances. Broadly speaking, they contain alcohols, esters, aldehydes, ketones, phenols, ethers, hydrocarbons usually in the form of terpenes, and nitrogen or sulphur compounds; certain solid oxygenated principles are called camphors. One chemical substance may be present in several of the oils. Consequently, it is not surprising to find that there is considerable overlapping in the toxic symptoms of the oils.

Camphor causes nervous excitability and convulsions. Turpentine occasionally causes convulsions, but usually causes depression with occasional respiratory symptoms. Eucalyptus causes a depression of rapid appearance, respiratory symptoms often being severe. Sassafras causes ataxia, pallor and shock, with some depression. Separate from the others in the group is oil of wintergreen, which gives symptoms of salicylate poisoning.

The present survey frequently emphasizes the need for prompt emesis and gastric lavage in treating poisoning cases, and it may have been felt that too great stress has been laid on what is obvious. However, one has only to consider the records of these 50 children to realize how seldom emesis is induced or gastric lavage carried out as first-aid measures. Gastric lavage is often difficult in toddlers, and its omission by practitioners living close to hospital is understandable, but failure to induce emesis in so many cases is surprising. It is of interest that emesis was carried out more often in turpentine poisoning than in the other forms, which suggests that the general public may believe that poisoning by 'household' poisons is more serious than poisoning by medicines or medicinal applications. This would be a dangerous opinion to hold.

It has been pointed out that five of the cases of camphor poisoning discussed showed no symptoms, and that four of these were the youngest patients in the series, which raised the possibility that the youngest patients recognize and reject an offensive taste. Another consideration is that convulsions occurred in only three of the eight patients over the age of 13 months who were given camphor in error, whereas convulsions occurred in six of the seven patients over the age of 13 months who took the camphor themselves. Can it be concluded from this that a bad taste is no great deterrent to a normal toddler who is investigating a new substance or does it mean that children who poison themselves tend to have abnormalities of oral sensation? The evidence is insufficient to sustain any conclusion, but the point might become one of considerable importance in devising means of cutting down accidental poison-



ing in childhood, so that further investigation seems desirable.

The prevention of poisoning by the volatile oils involves two considerations; how to stop parents giving their children camphor by mistake, and how to stop children ingesting volatile oils.

The first half of the problem almost defies solution. If a mother cannot recognize a poison bottle, a label 'Not to be taken internally', and the strong and characteristic smell of camphor, what *will* she recognize? A distinctive colouring? Such a suggestion might be worth a trial, but it is doubtful if it would be effective.

The great danger of the volatile oils to children lies in their extreme toxicity, and the problem is not to make the oils unpalatable or difficult of access, but to keep them away from children altogether. It is necessary to educate the public, and it seems that the best way to educate the public in the present instance is to give it a little trouble. Some curb should be put on these oils, which can all be sold across the counter of a chemist's shop. It seems desirable to remove such highly dangerous substances as oil of wintergreen and linimentum A.B.C. from the pharmacopeia, and substitute similar ointments, as ointments appear to be very rarely ingested by children. The other oils, except for oil of turpentine which has relatively mild toxic effects, should either be obtainable only on a doctor's prescription, or else the buyer should have to sign a 'poison book' to draw his attention to the danger of what he is buying. It is also necessary for the doctor to be aware of the dangers of the volatile oils before prescribing them in a household which contains children, and for a chemist to stress the danger when selling them.

### Summary

It is pointed out that the volatile oils as a group head the morbidity and mortality lists for accidental

poisoning in childhood. Camphorated oil and oil of turpentine are the oils most commonly ingested, but oil of wintergreen accounts for three times as many deaths as camphor, the second commonest lethal agent. The symptoms and treatment of poisoning by camphor, eucalyptus, sassafras, turpentine and oil of wintergreen are discussed. It is suggested that the sale of these dangerous poisons, with the exception of turpentine, be controlled.

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