Poisoning Potpourri

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Objectives

After completing this article, readers should be able to:

1. Manage a suspected acetaminophen overdose.
2. Describe one of the metabolic effects of ethanol ingestion in young children.
3. Delineate the signs and symptoms of theophylline toxicity.
4. Determine the cardiac anomaly that may be associated with an overdose of tricyclic antidepressants and how to assess the risk.
5. Manage a child who has ingested a hydrocarbon substance.
6. Recognize the signs and symptoms of methanol ingestion and know how to treat it.

Introduction

Unintentional poisoning can occur following exposure to any of a very large number of pharmaceutical or nonpharmaceutical products. This article briefly reviews the clinical toxicity resulting from the acute ingestion of selected substances by young children; clinicians are encouraged to discuss poisoning cases with colleagues at their regional poison control centers to obtain further information.

Acetaminophen

Definition/Epidemiology

Acetaminophen is one of the most common medications used to treat fever and pain in children. It also is the most common analgesic overdose in children younger than 6 years of age; more than 37,000 cases were reported in 1998 by the American Association of Poison Control Centers (AAPCC) Toxic Exposure Surveillance System. Although 99.8% of these exposures resulted in no or minor toxicity, it is important for clinicians to understand the mechanism of toxicity, clinical presentation, and treatment of a serious ingestion.

Clinical Aspects

The toxicity from acetaminophen overdose arises from metabolism of the drug. During hepatic metabolism of large quantities of acetaminophen, as in an overdose, a toxic metabolite accumulates in the hepatocyte and binds to intracellular molecules, causing damage to the liver cells. The minimum toxic dose of acetaminophen is 140 mg/kg. A single ingestion of this quantity may cause transient reversible liver damage. More severe toxicity results from ingestions in excess of 250 mg/kg.

The initial symptoms and signs of an acetaminophen overdose are nonspecific, develop over the first few hours, and consist of nausea and vomiting. Within 18 to 24 hours after the overdose, hepatic damage may become evident as serum hepatic transaminases begin to rise. If not treated, hepatic damage may worsen over the next 2 to 3 days before gradually resolving. In rare instances, the hepatic toxicity may be so serious that it progresses to severe hepatic damage, including fulminant hepatic failure.

The only accurate predictor of hepatic toxicity is an acetaminophen serum level measured between 4 and 10 hours after the overdose. Acetaminophen serum levels that fall above the nomogram line (Fig 1) may be associated with hepatic damage; those falling below the line are not associated with significant hepatic damage. Hepatic toxicity has been defined by elevations in serum hepatic transaminases (alanine aminotransferase, aspartate aminotransferase) and prolongation of the prothrombin time. The usefulness of this

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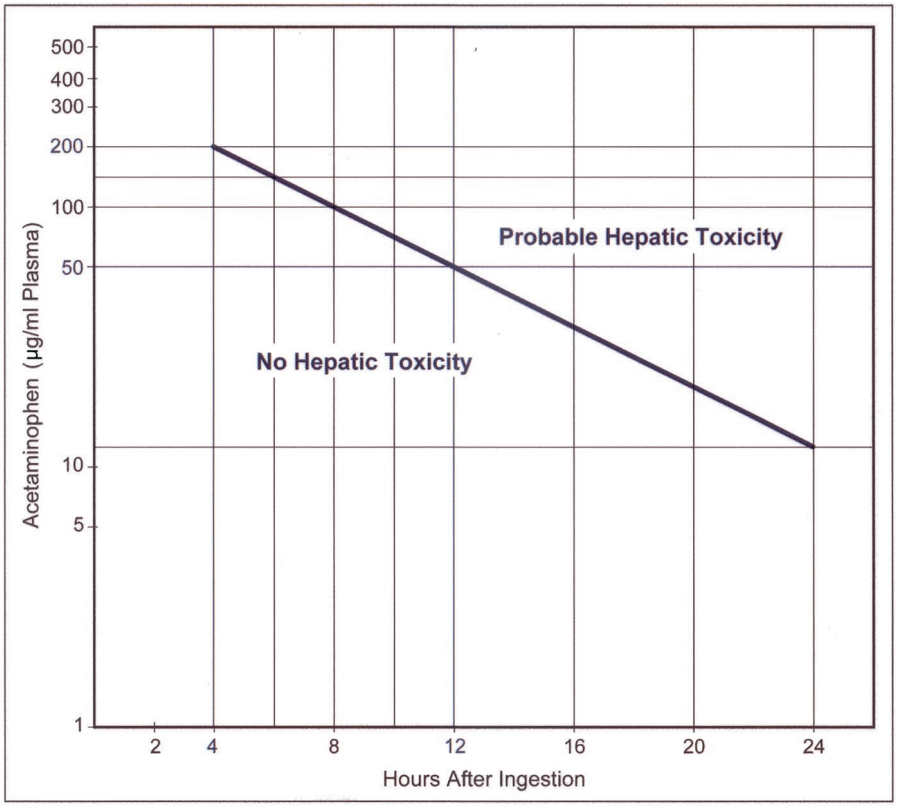

Figure 1. Acetaminophen nomogram.

nomogram in interpreting acetaminophen levels following the ingestion of modified-release preparations has not been established.

Diagnosis/Management

An acetaminophen overdose is diagnosed by a history of an ingestion of at least 140 mg of acetaminophen per kilogram body weight and a serum acetaminophen level above the therapeutic range. Acetaminophen toxicity is defined by a serum acetaminophen level above the line in the nomogram or by a positive history in association with elevations of serum hepatic transaminases.

Children who have a serum acetaminophen level in the toxic range should be treated with *N*-acetylcysteine. If an acetaminophen level cannot be determined, initiation of therapy should be based on the history of the ingestion of a toxic quantity of acetaminophen. Because the effectiveness of treatment declines with time after ingestion, treatment should be initiated within 10 hours of the ingestion. The current treatment regimen is summarized in Table 1. Alternate therapeutic regimens (parenteral administration, shorter duration) are being evaluated in the United States but are not yet approved for routine use.

The prognosis of an acute acetaminophen ingestion in a young child is good; few children develop hepatotoxicity, and only occasional cases progress to serious liver damage. If hepatic damage does occur and the child recovers, the hepatic damage resolves completely. Deaths are rare.

Ethanol

Definitions/Epidemiology

Ethanol (ethyl alcohol) is the traditional alcohol found in beverages such as wine (10% to 12% ethanol), beer (3% to 6%), and liquor (30% to 40%). Ethanol also may be found in some over-the-counter pharmaceutical products such as elixirs (5% to 15%) and cough medications (2% to 25%) as well as in some personal care products such as mouthwashes (10% to 25%) and aftershaves (15% to 80%).

Clinical Aspects

Ethanol is a dose-related general central nervous system (CNS) depressant that also induces hypoglycemia in young children. The metabolism of ethanol creates a relative lack of pyruvate, which blocks gluconeogenesis so that hypoglycemia may occur when dietary and hepatic sources of glucose have been depleted.

A single ingestion of 0.5 g/kg of ethanol (roughly equivalent to 1.5 mL/kg body weight) is sufficient to cause significant intoxication in a young child. The clinical effects of ethanol intoxication are those of dose-related progressive CNS depression and may range from inebriation, vomiting, and ataxia to coma, respiratory

Table 1. ***N*-acetylcysteine Dosing Schedule**

Initial dose	140 mg/kg orally once
Maintenance doses	70 mg/kg orally every 4 hours for 17 doses

depression, hypotension, and death. In addition, ethanol intoxication may mask toxicities from other coingested drugs. The effect of CNS stimulants may be blunted or masked in the presence of ethanol, and the clinical effects of other CNS depressants (eg, benzodiazepines, antihistamines) may be potentiated by its presence.

The clinical effects of ethanol poisoning are nonspecific and may be confused easily with the symptoms and signs of hypoglycemia. Ethanol-induced hypoglycemia occurs more frequently in young children than in adults, and its development or severity does not appear to be related to the dose or blood level of ethanol.

Appropriate laboratory blood tests to obtain in a patient who is suspected of being toxic from ethanol ingestion include measurement of serum electrolytes, glucose, and ethanol. A significant number of children admitted to the hospital for ethanol ingestion have potassium levels less than 3 mmol/L (3 mEq/L). If the patient exhibits signs of more severe CNS, respiratory, or cardiovascular depression than can be attributed to ethanol alone, a broad-spectrum drug screen may identify other CNS depressants.

Diagnosis/Management

Ethanol poisoning should be suspected in any patient who has CNS depression, particularly if there is a smell of "alcohol" on the breath. Any suspicion of ethanol ingestion must be confirmed with measurement of a blood ethanol level because it is difficult to predict the blood ethanol level based solely on clinical findings.

Management of the ethanol-poisoned patient is primarily symptomatic and supportive. Correction of hypoglycemia and electrolyte imbalance is important, as is the administration of parenteral fluids. There is no antidote to ethanol intoxication; extracorporeal removal techniques are reserved for the critically ill patient.

The prognosis for ethanol poisoning is good; recovery is expected with supportive care.

Hydrocarbons

Definitions/Epidemiology

Hydrocarbons are, by definition, substances comprised of hydrogen and carbon. In practice, these products are straight-chain (aliphatic), six-carbon ring-structure (aromatic), or mixtures of the two hydrocarbons. This review focuses on the unintentional ingestion of low-viscosity aliphatic (or petroleum distillate) hydrocarbon products by young children (Table 2); it does not cover aromatic hydrocarbons, halogenated hydrocarbons, or hydrocarbons with additives (eg, heavy metals, pesticides). Turpentine is distilled from wood that contains pine oil, and although the toxicity and management are similar to petroleum distillate hydrocarbons, it is not considered here.

Table 2. Examples of Hydrocarbons

	Aliphatic Hydrocarbons	Aromatic Hydrocarbons
Low-viscosity	Gasoline Kerosene Lighter fluid/naphtha Mineral spirits/varsol	Benzene Toluene Xylene
High-viscosity	Grease Motor oil Paraffin wax Petroleum jelly	

Of the more than 26,000 hydrocarbon exposures reported by the AAPCC for 1998, nearly 75% of the identified hydrocarbons involved gasoline, kerosene, lighter fluid/naphtha, and mineral spirits/varsol. Within this group, gasoline was the substance involved most commonly. Kerosene exposures appear to have had the highest morbidity (11% of exposures resulting in moderate or major clinical effects). Lighter fluid/naphtha was the only substance category that resulted in death (2 of 1,651 known outcomes).

Clinical Aspects

Petroleum distillate hydrocarbons irritate the gastrointestinal and respiratory tracts. The primary clinical effect of concern is chemical pneumonitis, and the major determinants of the degree of chemical pneumonitis are the volume of liquid aspirated and the product's viscosity (Table 2). Low-viscosity petroleum distillate hydrocarbon liquid that has been aspirated spreads over large areas of the lining of the lungs, destroying surfactant and causing widespread alveolar collapse, ventilation-perfusion mismatch, and hypoxemia. Direct capillary damage contributes to the chemical pneumonitis. Pneumonitis does not result from gastrointestinal absorption of the liquid or from inhalation of the fumes or vapors.

The initial symptoms and signs of hydrocarbon ingestion are oropharyngeal and gastric irritation. Coughing and choking may result from inhalation of the fumes and do not necessarily imply aspiration of the liquid. Vomiting may result from gastric irritation. Children who have aspirated a petroleum distillate hydrocarbon will demonstrate immediate significant coughing and respiratory distress. The physical examination may reveal a "petroleum" smell on the breath, tachypnea, retractions, bronchospasm, and wheezing and rales in the lungs; absence

of tachypnea is a good predictor for the absence of toxicity. Fulminant chemical pneumonitis may occur in more severe cases and is characterized by marked shortness of breath and hypoxemia. Fever occurring within 6 hours of the aspiration is due to tissue damage, not infection. Pulmonary damage reaches its clinical peak approximately 3 days after the aspiration.

Asymptomatic patients should be observed; there is no need for investigations. Symptomatic patients should undergo laboratory testing to evaluate and monitor their respiratory status. Arterial blood gas determinations may reflect hypoxemia, hypercarbia, and respiratory acidosis. Changes in the chest radiograph will lag behind findings on physical examination; chest radiography may not reveal abnormalities for 4 to 6 hours after the aspiration.

Diagnosis/Management

The diagnosis of petroleum distillate hydrocarbon aspiration should be based on a history of exposure, symptoms and signs of respiratory involvement, and findings on chest radiography that are compatible with diffuse chemical pneumonitis. Asymptomatic patients who remain so during 4 to 6 hours of observation and have normal chest radiographic findings may be discharged home. Asymptomatic patients who have abnormal chest radiographic findings should be admitted if good ambulatory follow-up is uncertain. Patients who are symptomatic should be admitted for observation and supportive care. The use of prophylactic corticosteroids and antibiotics is not recommended.

The majority of hydrocarbon ingestions result in either no or only minor clinical effects, and most patients who have petroleum distillate hydrocarbon aspiration chemical pneumonitis recover completely. Rarely, cases of pneumonitis have been associated with prolonged bronchospastic tendencies or pneumatoceles.

Theophylline

Definitions/Epidemiology

Theophylline (aminophylline) is used widely as a bronchodilator in the treatment of asthma. It is available in several different chemical forms (eg, salts) as well as in the free state. Sustained-release preparations are used more commonly than the regular-release products. Theophylline poisoning in young children is unusual; only 421 exposures were reported by the AAPCC for 1998, and more than 95% of outcomes were classified as no or minor effect. Children younger than 4 years of age appear to be at higher risk of developing serious toxicity than are older children.

Clinical Aspects

Theophylline has pharmacologic actions that result in diuresis, smooth muscle relaxation, and stimulation of the CNS and myocardium. In overdose, theophylline causes the release of endogenous catecholamines, resulting in sympathomimetic effects. Hypokalemia may result from diuresis, vomiting, and beta-adrenergic stimulation.

The acute ingestion of a dose of theophylline exceeding 10 mg/kg may result in some degree of clinical toxicity. The initial toxic effects include nausea, vomiting, and abdominal discomfort. Other clinical findings might include restlessness, agitation, tremors, peripheral vasodilation, and sinus tachycardia. Among the life-threatening clinical findings are generalized convulsions, hypotension, or hemodynamically significant cardiac dysrhythmias.

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Serum theophylline levels correlate well with clinical toxicity and should be measured every 2 hours until the levels are falling and the patient is showing clinical improvement. Theophylline levels may not peak for many hours after ingestion of a sustained-release product. Levels of 111 to 222 mcmol/L (20 to 40 mcg/mL) are associated with minor toxicity, levels of 222 to 333 mcmol/L (40 to 60 mcg/mL) cause moderate toxicity, levels of 388 to 444 mcmol/L (70 to 80 mcg/mL) result in severe toxicity, and levels greater than 555 mcmol/L (100 mcg/mL) may cause death if not treated promptly and appropriately.

Serum glucose and electrolyte levels should be measured, looking for hyperglycemia and hypokalemia. Arterial blood gases should be measured in patients who have experienced convulsions or who have other signs of serious toxicity. An electrocardiogram should be performed on patients who have cardiovascular findings; more seriously ill patients may require continuous cardiac monitoring.

Diagnosis/Management

The diagnosis of acute theophylline poisoning is based on a history of theophylline ingestion, compatible clinical findings, and an elevated serum theophylline concentration. Patients who have ingested a toxic amount of theophylline within the previous 2 hours should receive oral activated charcoal (at least 1 g/kg). Clinical signs of toxicity and serum theophylline concentrations should be used to determine disposition and further therapy. Patients who exhibit mild toxicity and have serum levels below 111 mcmol/L (20 mcg/mL) may be discharged. Those who have mild toxicity and higher levels should be treated with multiple doses of activated charcoal (0.125 g/kg per hour to a maximum of 12.5 g/h starting 1 hour after the initial dose of activated charcoal) until levels drop below 111 mcmol/L (20 mcg/mL). Vomiting that interferes with the administration of activated charcoal should be treated with ondansetron. Patients who have more severe or worsening toxicity and serum levels above 333 mcmol/L (60 mcg/mL) should be admitted for careful monitoring, supportive care, and multiple doses of activated charcoal. Patients whose serum theophylline levels are greater than 444 mcmol/L (80 mcg/mL) should be transferred to a facility where extracorporeal clearance can be performed. The prognosis for appropriately treated cases is good.

Tricyclic Antidepressants

Definitions/Epidemiology

The tricyclic antidepressants (TCA) include such compounds as amitriptyline, clomipramine, desipramine, doxepin, nortriptyline, protriptyline, and trimipramine. TCAs are used in pediatrics more for treating enuresis than depression. Treatment of a child with a TCA and the drug's candy-like appearance create a risk for unintentional ingestion by any children in the household.

Clinical Aspects

TCAs cause toxicity by blocking the cholinergic neurotransmitter acetylcholine, preventing reuptake of the adrenergic neurotransmitter norepinephrine, and by blocking sodium channels in the myocardium.

Clinical toxicity begins within 6 to 8 hours of an overdose and peaks within 24 hours of presentation; almost all reported deaths also occur within this 24-hour period. The initial clinical effects from an overdose include dry mouth, ileus, dilated pupils, urinary retention, and mild sinus tachycardia. CNS effects include delirium, agitation, restlessness, hallucinations, and convulsions. Brief, generalized convulsions most often develop within 1 to 2 hours of presentation. Life-threatening toxicity is due to cardiac dysrhythmias.

TCA serum levels can be measured, but the results do not contribute to treatment decisions. An electrocardiogram (ECG) may identify significant conduction defects (prolonged PR interval, QRS widening, QTc prolongation, rightward shift in the axis of the terminal 40 msec of the QRS complex [negative deflection in lead I and positive deflection in lead aVR], and atrioventricular block) and ventricular dysrhythmias (ventricular tachycardia, torsades de pointes, ventricular fibrillation). The single most useful predictor of convulsions or cardiac dysrhythmias is the QRS duration on a limb-lead electrocardiogram. Within the first 6 hours of TCA ingestion, a maximal limb-lead QRS duration of at least 100 msec is associated with a 33% incidence of convulsions; a QRS duration of at least 160 msec is associated with a 14% incidence of cardiac dysrhythmias (Fig. 2). An example of a rightward shift in the axis of the terminal 40 msec of the QRS complex is depicted in Figure 3.

Diagnosis/Management

TCA overdose should be suspected in any child who presents with the acute onset of nonfocal neurologic abnormalities and lives in a home in which TCAs are present.

Gastrointestinal decontamination with activated charcoal is important to minimize the absorption of ingested TCAs. Anticholinergic effects rarely need specific treatment. CNS toxicity, including convulsions, responds to the administration of benzodiazepines. Serial limb lead ECGs should be performed during the first 6 hours after ingestion. The maximal QRS duration is useful for predicting which patients are at risk for developing convulsions and cardiac dysrhythmias. As with any test, the ECG results should be interpreted within the general clinical picture of the patient. Intravenous bolus doses of sodium bicarbonate (1 to 2 mEq/kg) are the first choice for the treatment of cardiac dysrhythmias. For patients who have cardiac findings, cardiac monitoring should be continued until all toxic effects have resolved for 24 hours.

The prognosis for TCA toxicity generally is good, although major clinical toxicity and even fatalities continue to be reported. Resolution of clinical toxicity is expected in 24 to 48 hours. Although patients have been reported who developed late fatal dysrhythmias (2 to 5 days after overdoses), these patients were seriously ill prior to their death, had residual toxicity or coexisting

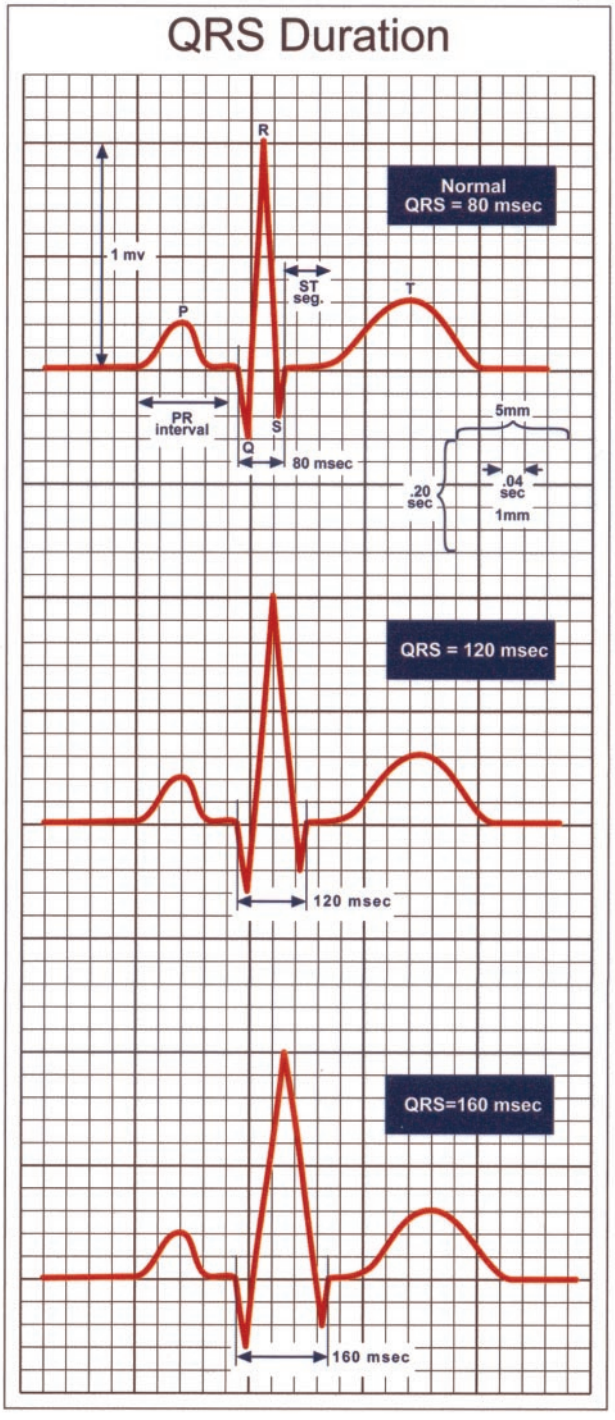

Figure 2. Examples of QRS widenings on electrocardiography.

disease, and frequently had not received adequate gastrointestinal decontamination; none was a child. No cases of late unexpected deterioration or death have been identified in retrospective reviews of TCA toxicity.

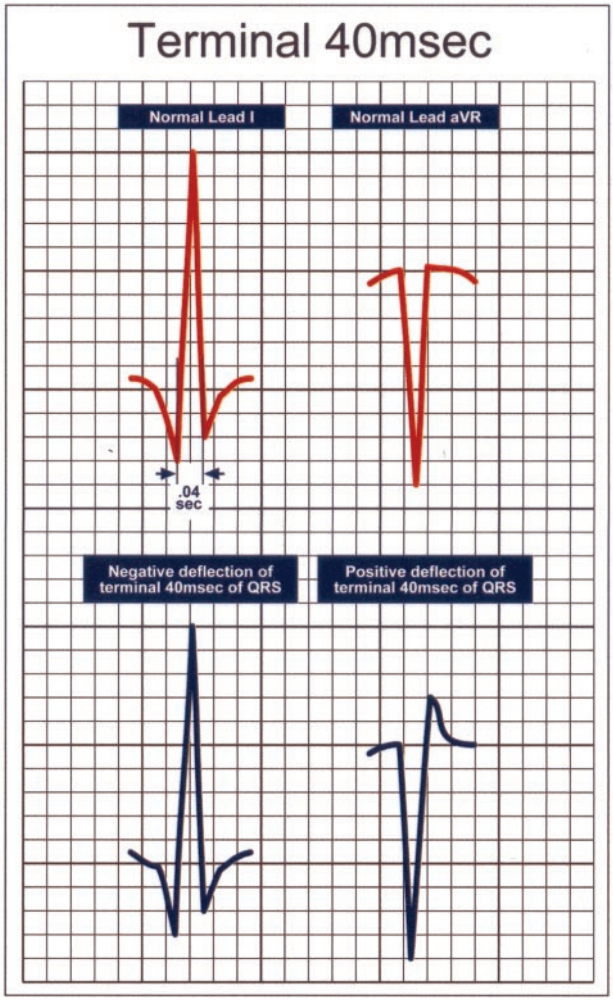

Figure 3. Right axis deviation of the terminal 40 msec of QRS complex.

Methanol

Definitions/Epidemiology

Methanol is found most commonly in automobile windshield washing fluid. Sometimes the diagnosis is delayed because methanol has many synonyms (Table 3). Windshield washer fluids containing methanol often come in large quantities and are brightly colored. Young children may ingest leftover windshield washer fluid that is put in a smaller container without a child-resistant cap after mistaking it for a familiar sugar drink. The unpleasant taste of methanol is not a deterrent to poisoning because only very small quantities are needed to result in serious toxicity. A potentially toxic amount of 100% methanol is approximately 0.15 mL/kg (equivalent to 3 mL in a 20 kg child).

Table 3. Methanol Synonyms

- Carbinol
- Colonial spirit
- Columbian spirit
- Methyl alcohol
- Methyl hydroxide
- Methylol
- Pyroxylic spirit
- Wood alcohol
- Wood naphtha
- Wood spirit

Clinical Aspects

Methanol causes inebriation but otherwise is not very toxic. Serious toxic effects develop when it is metabolized to formic acid, causing the severe metabolic acidosis and ocular findings characteristic of methanol poisoning. Early toxic effects from the ingestion of methanol are nausea, abdominal discomfort, vomiting, and inebriation. As the methanol is metabolized, metabolic acidosis and compensatory respiratory alkalosis develop. Approximately 18 to 24 hours after ingestion, effects related to the eyes develop and include blurred vision, "snow field" vision, and hyperemia and edema of the optic disks. Recommended laboratory tests include measuring the serum methanol level and arterial blood gases.

Management

Critical elements in the diagnosis of methanol poisoning are the history of methanol ingestion, severe metabolic acidosis, or evidence of eye toxicity. Any child who may have ingested any amount of methanol should be evaluated immediately in an emergency department. Metabolic acidosis, if present, should be treated with sodium bicarbonate. Administering ethanol (ethyl alcohol) in sufficient quantities to achieve a blood ethanol level of 22 mmol/L (100 mg/dL) blocks metabolism of methanol. Intravenous administration of leucovorin or folate (1 mg/kg up to a maximum of 50 mg) is recommended to hasten the elimination of formic acid. Hemodialysis is recommended for patients who have severe metabolic acidosis or methanol levels greater than 15.6 mmol/L (50 mg/dL).

If treated early and appropriately, recovery is expected from methanol toxicity. Permanent ocular damage, including blindness, has been reported in severe untreated cases in adults. Fatalities are uncommon among young children, but not among adults.

Suggested Reading

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PIR Quiz

Quiz also available online at *www.pedsinreview.org.*

1. You are evaluating a 14-year-old girl in the emergency department who is complaining of nausea, vomiting, and abdominal pain. Physical examination reveals mild agitation and tachycardia. Results of a capillary blood gas measurement are normal, and her blood glucose is 210 mg/dL (11.66 mmol/L). You suspect an ingestion. Of the following substances, which is the *most* likely to cause her symptoms?

   A. Acetaminophen.
   B. Ethanol.
   C. Imipramine.
   D. Methanol.
   E. Theophylline.

2. Which of the following statements regarding acetaminophen overdose is *true*?

   A. Hepatic damage is usually not evident until 18 to 24 hours after the overdose.
   B. More than 50% of affected children develop serious liver disease.
   C. *N*-acetylcysteine should be given intravenously for best results.
   D. The best predictor of hepatic toxicity is a prothrombin time measured 4 hours after overdose.
   E. Treatment initiated as late as 2 days after overdose is effective in preventing liver disease.

3. You are seeing a 7-year-old boy brought in to your office for vomiting and mental status changes that began at home last night. Findings on physical examination are normal, except for confusion and ataxia. There is a faint smell of alcohol on his breath. His blood glucose is 30 mg/dL (1.67 mmol/L). His mother denies the presence of liquor in the home. Of the following statements, you are *most* likely to tell her that:

   A. A urine toxicology screen will help confirm a diagnosis of ethanol poisoning.
   B. Ethanol intoxication only occurs after ingestion of ethanol-containing beverages.
   C. Hypoglycemia indicates that a large quantity of alcohol was ingested.
   D. Masked toxicities from other coingested drugs should be strongly considered.
   E. Ongoing neurologic deficits should be expected.

4. A worried mother brings in her 2-year-old daughter after finding her drinking from a gasoline container in the garage. The little girl is irritable and tachypneic. There are no obvious burns in the oropharynx. She has bilateral diffuse wheezing and retractions. Which of the following statements is *true*?

   A. Antibiotics and chest percussive therapy are indicated to prevent pneumonitis.
   B. Complete recovery is most likely.
   C. She may be discharged home if findings on chest radiography are normal.
   D. She should undergo immediate endoscopy to rule out esophageal burns.
   E. The presence of cough implies a chemical pneumonitis.

5. You are evaluating a 3-year-old boy who was brought to the emergency department because of a seizure at home. Further history reveals that his grandmother takes the antidepressant amitriptyline. He is tachycardic, his pupils are dilated, and he has a dry mouth. Electrocardiography reveals a QRS of 200 msec. Following this examination, he has another brief seizure. In determining the management for this patient, you remember that:

   A. Activated charcoal does not bind tricyclic antidepressants and, therefore, is not helpful.
   B. Physostigmine is indicated for the anticholinergic effects.
   C. Seizures generally respond well to the administration of dextrose.
   D. Sodium bicarbonate is the first-line treatment for cardiac dysrhythmias.
   E. Tricyclic antidepressant serum levels are an adequate predictor of toxicity.