In this chapter, a brief description of ethylene glycol poisoning, symptoms, and its treatment are given. Ethylene glycol is readily available and is easy to obtain. It is stored in most car garages. For this reason, accidental ingestion by people as well as by pets happens quite frequently. Thousands of exposures and several deaths are reported every year by poison centers. Ethylene glycol is the ingredient that makes antifreeze tasty. It is a colorless, odorless syrup-like alcohol that tastes sweet. It can mix easily with sodas, juices, and other sugary beverages. Pets and children are prone to lap up a puddle of antifreeze left on garage floors. Every year 90,000 animals and 4000 children ingest this toxic liquid. Several states require manufacturers to add a bittering agent to antifreeze (1). Two published cases in newspapers illustrate a mother who was wrongly convicted based on a false-positive identification of ethylene glycol and a young woman's suicide attempt with ethylene glycol.

5.1 Mechanism of Toxicity

Most of the ingestions of ethylene glycol happen in children. A small percentage of these ingestions are accidental and the rest are intentional or suicide attempts. The toxic consequences are quite severe and include renal and cardiovascular failure, brain damage, and death. The toxicity is because of the metabolites generated by ethylene glycol due to the action of alcohol dehydrogenase (ADH). Ethylene glycol is metabolized to glycoaldehyde and then to glycolic acid (glycolate), which is responsible for severe metabolic acidosis. Glyoxylate is further metabolized to glyoxylic acid (glyoxylate), which also undergoes metabolic conversion via several pathways (Figure 5.1). Finally glyoxalate is formed, which combines with calcium forming calcium oxalate crystals in many tissues and urine (2,3).

5.2 Clinical Symptoms

Ethylene glycol poisoning causes central nervous system (CNS) depression within 12 h after ingestion. The patient may experience ataxia, slurred
speech, and altered mental status. Anion gap acidosis and formation of oxalate crystals may also be seen. After approximately 12 to 24 h, cardiopulmonary symptoms such as hypertension, tachycardia, and heart failure may occur. After 24 to 72 h, renal failure may occur. Severe acidosis hyperkalemia, seizures, and coma indicate a poor prognosis (2,3).

5.3 Diagnosis

Metabolic acidosis and respiratory distress are present in most cases. Most institutions do not have the facilities to perform ethylene glycol determination. Therefore, treatment is started based on patient history and symptoms. The presence of oxalate crystals in urine and hypocalcemia are highly suggestive symptoms of ethylene glycol poisoning. Finally, the most conclusive evidence is the determination of serum and urine ethylene glycol (2,3).
5.4 Laboratory Monitoring

Simultaneous determination of ethylene glycol and its metabolites in serum needs to be monitored to follow the progress of detoxification. Briefly, the procedure is as follows. After serum proteins are precipitated by acetonitrile, the supernatant is derivatized by trimethylsilyl and the resulting derivatives are analyzed by capillary column GC. The internal standard is 3-bromo-1-propanol. Details of the exact procedure are published elsewhere (4).

5.5 Treatment

Where there are no facilities to monitor ethylene glycol and its metabolites, infusion of IV ethanol is started and the serum samples are sent to central referral research hospitals. Since ethanol blocks ADH, patients are treated with ethanol to maintain serum concentration of ethanol at 100 mg/dL to 150 mg/dL. This is accomplished by using 10% ethanol over a period of 20 to 60 min. If fomepizole is available, it is administered as a 50 mg/kg loading dose. This is followed by four bolus doses of 10 mg/kg every 12 h. The treatment is continued until ethylene glycol concentration is less than 20 mg/dL (2,3).

5.6 False-Positive Ethylene Glycol

This case illustrates false-positive ethylene glycol identification by two independent laboratories. It appeared that a male child died due to presumptive ethylene glycol poisoning. The mother was accused of poisoning her child by feeding the baby formula containing ethylene glycol. She was sentenced to life in prison, but while in prison gave birth to a second son who was found to have methylmalonic acidemia (MMA). On reexamination of the serum stored from the first child, it became evident that the first child also had MMA. Apparently, the two independent laboratories mistakenly identified propionic acid as ethylene glycol in the serum of the first child. The mother eventually was released from prison once the authorities realized that her first child died due to an inborn error of metabolism (5).

5.7 Positive Ethylene Glycol

This published newspaper report on the Combs trial illustrates that a positive identification of ethylene glycol poisoning resulted in Joe Combs, a Baptist preacher, and his wife receiving a sentence of 179 years in prison.
for child abuse. The Combses had taken a girl from an Indiana children's home, but never adopted her. This girl, Esther Combs, 19 years of age in 2007, told the police and the doctors in the hospital where she was admitted for ethylene glycol poisoning that her parents beat, tortured, and abused her. Joe Combs repeatedly raped her over the years. The doctors found both horizontal and vertical scars all over her body. The nurses, social workers, and the doctors found burn marks on her body. She had broken teeth. She could not take it anymore, so on February 18, 2007 she drank a 24-oz cup of antifreeze, brushed her teeth, and went to bed. She said she wanted to die. She was transported by ambulance to the hospital after a 911 call reported that a young woman was having seizures. This was followed by several investigations, the trial, and conviction of Joe Combs and his wife (6).

References