

CRIMINAL POISONING

*INVESTIGATIONAL GUIDE
FOR LAW ENFORCEMENT,
TOXICOLOGISTS,
FORENSIC SCIENTISTS,
AND ATTORNEYS*

Second Edition

By

JOHN HARRIS TRESTRAIL, III

RPh, FAACT, DABAT



 **HUMANA PRESS**

Criminal Poisoning

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John Harris Trestrail, III, RPh, FAACT, DABAT

Center for the Study of Criminal Poisoning, Grand Rapids, MI



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DEDICATION

I would like to humbly dedicate this work to my parents, John Harris and Edith (McClay) Trestrail, for emigrating from the United Kingdom and Ireland to America, which provided unlimited professional opportunities for their two children, and for their continued support and educational encouragement over the years, which allowed us to grow within and contribute to the vocations that we chose to follow.

This work is also dedicated to my wife, Mary, my children, John and Amanda, and my grandchildren, Olivia and Owen, who have allowed me to passionately follow my love for the subject of toxicology, even though, at many times, they must have considered it somewhat eccentric and bizarre.

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John Harris Trestrail, III, RPh, FAACT, DABAT

PREFACE

I am very pleased with the success of the first edition of *Criminal Poisoning: Investigational Guide for Law Enforcement, Toxicologists, Forensic Scientists, and Attorneys*, and would like to thank those who contributed constructive comments with regard to typographical errors and what they would like to see expanded. Ideas have also been provided by attendees to my symposia on “Murder by Poison!” and “Poisoners Throughout History,” which have been presented at venues around the world.

In this revised and expanded edition, the reader will find more discussion on what has been revealed about the poisoner and his victims since the 2000 publication of the first.

In addition, I have expanded the Appendix to include more commonly used poisons, as well as the use of antifreeze as a poison, to reflect some cases that have recently come to light.

The bibliography has been expanded by more than 200 additional citations of texts and journal articles from the international literature related to criminal poisoning.

John Harris Trestrail, III, RPh, FAACT, DABAT

DISCLAIMER: This publication is intended to serve the reader with general background information representing various aspects of toxicology as it applies to modern litigation. However, this is not intended to serve as a substitute for intensive research respecting various issues, as each case must be approached on a case-by-case basis. Some cases will require intensive research independent of this work.

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INTRODUCTION

“Passion [poison] often makes fools of clever men; sometimes even makes clever men of fools.” — La Rochefoucauld

In dealing exclusively with poisons as weapons in the hands of the murderer, tamperer, and terrorist, *Criminal Poisoning: Investigational Guide for Law Enforcement, Toxicologists, Forensic Scientists, and Attorneys, Second Edition* continues to represent a pioneering work.

The following are my goals in bringing forth this book:

- To make it much more difficult for poisoners to get away with their crime.
- To help investigators develop a mindset that will make their poisoning investigations more efficient.
- To help attorneys successfully prosecute the poisoning offender.
- To raise the degree of suspicion in the first place!
- To ultimately reduce the number of homicidal poisonings by generating awareness in forensic communities.

In order to encourage international communication on the subject of homicidal poisons, I have founded the Center for the Study of Criminal Poisoning, which performs the following activities:

- Maintains an international database on criminal poisoning cases.
- Maintains a collection of printed resources and references on forensic toxicology.
- Provides training workshops on the investigation of murder by poison.
- Conducts forensic research projects.
- Encourages international communication on homicidal poisoning.

Some of the oldest offensive killing weapons used by humankind, after the stick, stone, and fist, were the poisons. Yet, after nearly 5000 years of

recorded history, no in-depth work has ever been produced that deals exclusively with poisons as weapons for homicide. My review of the international literature has revealed only a few scattered scientific papers dealing with the psychology of the poisoner. In addition, books on medical jurisprudence and forensic investigation have devoted only a few pages to this important subject, usually stating that it is a *rare* occurrence. How do we know the true rarity of the use of poisons or homicide? All one has to do is look at the number of poisonings that are first documented only after the exhumation of the deceased in order to raise the logical question, "If we missed this one, how many more have been missed?" If all those buried in our cemeteries who were poisoned could raise their hands, we would probably be shocked by the numbers!

For years, the homicidal poisoner has remained shrouded in mystery. What little we think we know about the criminal poisoner represents only the tip of an iceberg, with the majority of our knowledge still remaining hidden under the surface. It is hoped that this work will serve as a valuable tool in the hands of the criminal investigator, forensic scientist, toxicologist, and attorney, and better increase our chances of avenging each victim of a homicidal poisoner.

The reader should know that for brevity and consistency, I have chosen to use the pronoun "he" in referring to a poisoner throughout most of this work. If the reader wants to mentally substitute "she" for "he," that is perfectly appropriate.

Unfortunately, a copy of this work is eventually bound to wind up in the hands of a potential criminal poisoner, who may gain some information to use in planning their crime and attempting to escape punishment. But the trade-off is that at the same time, law enforcement will now have detailed information that, it is hoped, will assist them in dealing more effectively with this almost invisible crime.

For, as I try to tell in the following poem, we each must become a *Toxic Avenger*, and bring to justice those individuals who have chosen this most secretive mode of homicide. It makes no difference whether a victim was killed with a knife, a gun, a bomb, or a poison; they each and every one deserve the full extent of justice that our society can deliver.

Homicide by poisoning is one of the most difficult types of cases to prove, both for the death investigator and medical expert. It has been stated that poisonings occur rarely, accounting for only about 3 to 6% of homicides. In this statement, we should add the word "known" before homicides, as I believe that they do not rarely occur, but rather are rarely detected!

The main problem is that in poisonings, the investigator often has no visible signs of trauma to indicate that the death is other than natural. Bullets

The Toxic Avenger

by

John Harris Trestrail, III, RPH, FAACT, DABAT

Toxicology

*From the grave, if lips could speak
the person who was, pleads—you must seek
the individual who had my trust,
and through deceit and cunning into grave did thrust
this body once alive and well,
now silenced by death, who cannot tell
my death was NOT what all thought then,
for a poison brought my life to end!
Avenge me now, for you alone
can find the truth beneath this stone.
Look close and the clues you will see
that tell the tale of what killed me.
For you must tell all others now,
that this was MURDER—and tell them how!
For if no one looks to find what's here,
an injustice was done to a life so dear.
If now only you could hear,
my muted pleading to make wants clear.
I'd speak as plain as it could be.
Since I can't—YOU must AVENGE ME!*

leave holes, knives leave cuts, clubs leave bruises, but the poisoner covers the murder with a blanket of invisibility! Important clues are usually buried with the victim. As poisons are offensive weapons, not defensive weapons, often the crime scene may seem nonexistent.

As the French scientist Louis Pasteur once said, “Chance favors the prepared mind,” and in this spirit, it is hoped that the contents of *Criminal Poisoning: Investigational Guide for Law Enforcement, Toxicologists, Forensic Scientists, and Attorneys, Second Edition* will so prepare the mind of every criminal investigator, and therefore greatly increase their chances of solving this type of criminal event. After reading this reference work, it is hoped that each criminal investigator will become a “toxic avenger”!

John Harris Trestrail, III, RPH, FAACT, DABAT

Chapter 1

Poisoners Throughout History

“I maintain that though you would often in the fifteenth century have heard the snobbish Roman say, in a would-be off-hand tone, ‘I am dining with the Borgias tonight’, no Roman was ever able to say, ‘I dined last night with the Borgias.’ ” —And Even Now, Max Beerbohm

It is safe to say that poisoners have always been part of society, continue to be with us now, and will likely be with us in the future. To better understand these offenders, it is important for us to understand how our knowledge of poisons has developed and been passed down throughout history, in the various cultures and societies of the world.

1.1. POISONS IN ANCIENT TIMES

1.1.1. Introduction

The first homicidal poisoner is now clearly lost in the mists of time, living as early as 70,000 BC, yet one can certainly speculate on the type of person and incident that led to the possession of this knowledge. Certainly he or she was a member of an early tribe of ancient humanity who first noticed the negative effects that exposure to certain substances had on living organisms. Perhaps it began with the observation that shortly after consuming a plant, fungus, or mineral an animal or fellow tribal member became ill and possibly died. This reasoning individual was able to conclude cause and effect by the method *post hoc ergo propter hoc* (after this, therefore on account of this); this method is usually considered an illogical form of reasoning but here is quite correctly applied. This observation allowed proper determination of the potential for the deleterious effects that would result from exposure

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to a certain substance originating from an animal, a vegetable, or a mineral source. Early man believed that flowers drew their toxicity from the vapors emanating from the entrances to the “Underworld,” that snakes developed their venom by devouring plants, and that stinging insects increased their potency by drawing venom from dead snakes. Any individual who obtained knowledge of the effects of poisons would certainly possess a great power among fellow tribal members. Perhaps the knowledge could have been used for the good of the group, as with the development of poisons for hunting, but the knowledge could certainly have been as easily used for homicidal purposes. This knowledge of poisons led eventually to power, the power to mystery, and the mystery to the fear of those individuals holding the ability to kill with such an invisible weapon. It is probable that this powerful knowledge was kept secret among a very select group of shamanistic individuals. Whoever this individual was, the knowledge he possessed was passed among selected members of the inner power circle, by word of mouth, down through countless generations.

Let us travel back in time to look at various ancient peoples and their knowledge of poisons, and especially the use of these substances for the purpose of homicide.

1.1.2. The Sumerians

Our first stop on our trip to the past is with the Sumerians, peoples living in Mesopotamia (modern-day Iraq) around 2500 BC. It is known that there has been a knowledge and interest in the subject of poisons as far back as their early recorded history. Deciphering of the Sumerians’ ancient cuneiform clay documents has revealed that they worshiped a deity of noxious poisons known to them as “Gula.” She was the first known, recorded spirit associated with poisons and was also called the “Goddess of Healing,” the “Mistress of Charms and Spells,” and the “Controller of Noxious Poisons.”

1.1.3. The Ancient Egyptians

As early as 3000 BC, the Egyptian King Menes studied the properties of poisonous plants (Smith, 1952). The “Ebers’ Papyrus” (ca. 1534 BC), an ancient Egyptian document, recorded the incantation, “repeat not the name I.A.O., under the penalty of the peach.” The Egyptians may have known that the seed kernel of the peach, and other members of the botanical genus *Prunus* (including the cherry, apricot, and bitter almond), contain plant compounds known today as “cyanogenic glycosides,” which can release toxic cyanide compounds in the presence of water and the proper plant enzyme. The Egyptians even believed that their gods were susceptible to the effects of poisonous entities.

They believed that their god “Ra” nearly succumbed from the effects of a venomous snakebite, and that “Horus” suffered a fatal outcome from the sting of a scorpion. In 525 BC, Psammetichus, the king of Egypt, was forced to drink the then-believed poisonous substance “bull’s blood,” which allegedly caused his immediate death. Zopyrus, a physician in Alexandria, concocted a general poison antidote that consisted of 30–50 various ingredients.

1.1.4. The Hebrews

Some scholars believe that the witches mentioned in the Old Testament were sorcerers and poison vendors. The Hebrews even had words for some of our dangerous poisons: *sam* (arsenic), *boschka* (aconite), and *son* (ergot). Although the use of poisoned arrows is mentioned in the Bible (in the book of Job), there are no references in the Old or New Testaments to the homicidal use of poisons (Bombaugh, 1899).

1.1.5. The Asian Indians

In the earliest writings from ancient India, one can begin to see discussions of the incidence of homicidal poisoning and its investigation. Two of the earliest writings on the subject of poisons, dating from 600 to 100 BC, were the *Charaka Samhita* and the *Susruta Samhita*. Another document, the *Veda*, gives the physician specific directions in the detection of poisoners:

“He does not answer questions, or the answers are evasive. He speaks nonsense, rubs the great toe along the ground and shivers. His face is discolored. He rubs the roots of the hair with his fingers and he tries by every means to leave the house. The food which is suspected should be given to animals. It is necessary for the practitioner to have knowledge of the symptoms of the different poisons and their antidotes, as the enemies of the Raja, bad women and ungrateful servants sometimes mix poison with food.”

It has even been speculated by some scholars that the Indian practice of “*suttee*,” in which the living widow was burned along with the corpse of her late husband, might have had some basis in attempting to discourage conjugal homicide (Meek, 1928, p. 1).

1.1.6. Nicander of Colophon

Nicander (204–135 BC), a physician, compiled the first poison pharmacopoeia while serving as personal attendant to Attalus III, the king of Pergamum, Greece. His favorite antidote consisted of viper parts seasoned with aromatic herbs and fruits (consisting mostly of ginger, cinnamon, myrrh, iris, and gentian). This antidote probably has no toxicological basis for its

effectiveness by today's standards. He also wrote two poems on poisons: *Theriaca*, consisting of 1000 lines dealing with poisonous animals, plants, and remedies, and *Alexipharmaca*, consisting of 600 lines dealing with poisons in general (8 animal and 11 plant), along with the subject of cures and antidotes.

1.1.7. Philon of Tarsus

Philon was a physician who developed one of the most long-lived antidotal potions for poisoning, which was called the "Philonium Romanorum." This antidotal concoction consisted of multiple herbs (spikenard, henbane, pyrethrum, euphorbia, and saffron). Once again, it had no toxicological basis for its effectiveness by today's standards.

1.1.8. Mithridates

Mithridates, king of Pontus (in modern Turkey), lived from 132 to 63 BC and had the reputation of knowing more about poisons and their proper antidotes than any other person of his time. He was very concerned with the possibility of being assassinated by poison, so he experimented with poisons and antidotes on himself as well as captured prisoners. He developed a so-called universal antidote, which was called "Mithridatum" in his honor. This antidote remained so popular in the minds of the people that it was still available in Italian pharmacies up through the 17th century. Once again, considering the ingredients of this mixture with today's toxicological knowledge, it is obvious that little protection could be obtained from Mithridates' antidote.

1.1.9. The Greeks

The Greeks gave us the word *toxicon*, used to denote poison, from their word "*toxon*," signifying a bow, which in warfare was used to shoot poisoned arrows at the enemy. From this Greek word come all of the words in use today to denote poison: *toxicology*, *toxic*, *intoxicated*, and so on. However, the word "intoxicated" today does not have the same meaning as it did in ancient Greece. If one were to ask an ancient Greek what it means to be intoxicated, that person would describe a physical condition resulting from being poisoned by an arrow.

Medea, a sorceress and the priestess of Hecate in Greek mythology, was credited as the first to use the plant known as "Meadow Saffron" (*Colchicum autumnale* L.) as a poison. Today, we know that this plant contains the very potent poison colchicine, used in modern medicine as a remedy for gout. In the classic literary work of the Greek Homer's *Odyssey*, one finds a discussion of one of the first great sorceresses, Circe, who used poisons and potions to subdue men to her ways.

The most famous of the Greek poisoners was Olympias, the wife of Philip of Macedon and the mother of Alexander the Great. She was involved in the deaths of Aridaeus, his wife Eurydice, Nicanor, and many other prominent men of Macedonia.

The Greeks also developed what was known as the “Athenian State Poison,” concocted from the very poisonous plant more commonly known today as poison hemlock (*Conium maculatum* L.). This very toxic plant containing the poison coniine, was reportedly used to execute the philosopher Socrates for his crime of corrupting the youth of Athens with his philosophical teachings.

Aristotle, in his writings of the period, described the preparation and use of arrow poisons by the Scythians, in which they allowed the bodies of snakes to decay and combined the exuding liquid with the clear fluid from decomposing blood. This mixture was then applied to arrows for use in battles. The greatest danger from this material was likely owing to septicemia (blood poisoning) from bacterial invasion.

It is interesting to note that a review of the writings of the famous Greek physician Hippocrates reveals no information on criminal poisoning. He did, however, make his students swear that they would not traffic in poisons in their practice of the medical arts.

During the Greek period, the Court of Areopagus was assigned the function of dealing with trials for poisoning cases.

The physician Galen (129–ca. 199 AD) compounded an antidote called “Nut Theriac,” which was to be used as a remedy for bites, stings, and other poisons. This antidote consisted of plant parts and salt, mixed into porridge.

1.1.10. The Romans

The ancient Romans documented the grand-scale use of poisons for homicidal purposes. As early as 131 BC, according to the writer Livy, there was an outbreak of homicidal poisoning in high circles of Roman society. One of the most infamous poisoners of the time was a woman named Locusta, who was the personal poisoner for Emperor Nero. With her assistance and advice, Nero murdered his brother Britannicus with cyanide containing natural compounds, and he also murdered his mother and several wives. Livia, who was the wife of Emperor Augustus, used the belladonna plant (*Atropa belladonna*) as a homicidal weapon. Agrippina, the wife of Claudius, killed him by injecting poisons into figs he then ate. Eventually 170 Roman women were convicted and punished for their homicidal poisoning activities.

So prevalent was the use of the wolfsbane plant (*Aconitum napellus* L.), with its highly toxic alkaloid aconitine, that the emperor Trajan (98–117 AD)

eventually banned the growing of this plant in all Roman domestic gardens. In fact, the writer Ovid referred to aconite as the “step-mother’s poison.”

In the work *Metamorphoses*, by Ovid, one finds this description of this perilous time in Roman history:

“Guest was not safe from host, nor father-in-law from son-in-law; even among brothers it was rare to find affection. The husband longed for the death of his wife, she of her husband; and murderous step-mothers brewed deadly poisons, and sons inquired into their fathers’ years before the time.”

In 82 BC, the ruler Sulla issued an edict known as “Lex Cornelia” against assassination by poison. This edict was the first legislative enactment in history against the use of poison as a means of homicide.

1.1.11. The “Italian School of Poisoners”

Deep within the psyche of the Italians of the Middle Ages existed the knowledge and will to use poisons to obtain wealth and power. In the year 1419, members of a group known as the Venetian “Council of Ten” carried out murder by poison for a fee. Three of their recipes for poison weapons are preserved as the “secreta secretissima,” in archives dating from 1540 to 1544 AD. Chief ingredients included corrosive sublimate (mercuric chloride), white arsenic (arsenic trioxide), arsenic trisulfide, and arsenic trichloride. In Venice and Rome, in the 15th to 17th centuries, there were schools for students who wished to become poisoners.

The name Borgia is the first that comes to mind regarding this location and period of time. The leader of this poisoning clan was one Rodrigo Borgia, born in 1431, who went on to become Pope Alexander VI. Among his five children were Cesare and Lucrezia, whom most people associate with murder-by-poison plots. In fact, Lucrezia, who died at the age of 39, probably never killed anyone. However, her brother Cesare, who died at the age of 32, was responsible for the murders of dozens of people in which poison was used as the instrument. The poison most frequently used by the Borgias was arsenic, which they used in the form of a poison that they called “La Cantrella,” a mixture of arsenic and phosphorus. It is believed that their weapon was prepared as follows:

“A hog was killed with arsenic. Its abdomen was opened and sprinkled with more of the same drug. The animal was then allowed to putrefy. The liquor which trickled from the decaying carcass was collected and evaporated to a powder.” (Meek, 1928, p. 7)

With the secret popularity of a piece of jewelry known as a poison ring, certainly no one aware of the Borgia’s knowledge of poisons would want to

take dinner with them without some consternation about the possible consequences.

Around 1650 another famous Italian poisoner of the era, Madame Giulia Toffana, produced and sold a mixture to would-be users called “Aqua Toffana,” thought to have been a solution of arsenic trioxide. She was credited with more than 600 successful poisonings and admitted to being involved in the poisoning of two popes, Pius III and Clement IV.

In 1659, the poisoner Hieronyma Spara formed a society in which she taught women how to murder their husbands by means of poisons. She dispensed her poison in small vials labeled “Manna of St. Nicholas of Bari.”

Catherine de Medici, who became the bride of the French King Henry II, was credited with carrying the Italian knowledge about poisons and methods of poisoning into France, by means of her accomplices, the Florentines Rene Bianco and Cosme Ruggieri. In fact, the king was so afraid of her poisoning powers and abilities that a “unicorn’s horn” (most likely the tusk of a marine mammal called the narwhal), then thought to be an antidote against poisons, became part of the official regal dowry. Catherine is usually also credited with being involved in the homicidal poisonings of Jeanne d’Albret, queen of Navarre; the Cardinal of Lorraine; Coffe, a marshal of France; and the Duc d’Anjou.

1.1.12. The “French School of Poisoners”

The most notorious maker of poisons in the 17th century was a man named Antonio Exili (a.k.a. Nicolo Eggidio), who was a professional poisoner once in the employment of Queen Christina of Sweden. During his imprisonment in the Bastille, he taught his skills to a fellow prisoner named Jean-Baptiste de Gaudin de Sainte-Croix. On release from prison, Sainte-Croix teamed up with a very greedy woman by the name of Marie-Madeleine d’Aubray, the Marquise de Brinvilliers. They soon experimented with many poisonous compounds, such as arsenic, sugar of lead, corrosive sublimate, tartar emetic, and copper sulfate. In fact, the marchioness even took their formulations into the hospitals of the time and mixed them in gifts of food and drink for the sick, in order to study the effectiveness of their poisonous weapons. To gain property and wealth, she allegedly murdered her father, two brothers, and a sister. Found guilty of these crimes, she was executed in 1676, at the Place de Grève, in Paris.

Another of the French poisoners was Catherine Deshayes Monvoisin (a.k.a. La Voisin) (1640–1680), who was an abortionist, and considered a sorceress of the time. She provided poisons to women so that they could do away with their spouses. One of her popular poisons was known as “La Poudre

de Succession” (inheritance powder). This poison was thought to have had a base of arsenic, mixed with aconite, belladonna, and opium. She was probably one of the last poisoners for hire. Deshayes accepted a sizeable commission to poison Louis XIV, but her efforts were unsuccessful, and she was found guilty of the attempt on the king’s life. Her punishment, after severe torture, was that she be burned at the stake.

From 1679 to 1680, there occurred in France what came to be known as the “Affair of the Poisons,” which involved numerous high-society murders. An investigative organization known as “La Chambre Ardente” (The Fiery Room), operating in France (1679–1682) under the reign of Louis XIV, was formed to deal with murder suspects during this rein of criminal poisonings. During the Chambre’s operation, it investigated 442 persons and ordered 367 arrests. Of those individuals investigated, 36 were executed, 23 banished, and 218 imprisoned. It was, in effect, a poisoner’s “Inquisition.”

Through the ages, many other poisoners operated around the world, in various countries:

- In 1596, Edward Squires was hired by Spain to poison Queen Elizabeth I by smearing an opium-based poison on the pommel of her horse saddle.
- In 1613, the Countess of Somerset was found guilty of utilizing “corrosive sublimate” (mercuric chloride) in a mass conspiracy to murder Sir Thomas Overbury while he was imprisoned in the Tower of London.
- In 1776, Thomas Hickey attempted to assassinate George Washington by poisoning a dish of green peas. Foiled in his attempt, he was hanged, becoming the first American executed for treason.

1.2. POISONERS IN THE MODERN ERA

Before 1800, most poisonings were confined to the very wealthy, as a means of speeding the departure of an individual who stood in the way of an offender obtaining an inheritance or power. But, beginning in 1830, with the development of the life insurance industry and “burial clubs” for the lower classes, there was now a monetary value on the life of common individuals. Then, it was killing for a one-time monetary reward (Watson, 2004).

We should not fool ourselves into thinking that poisoners operated only in the past, because they have continued their nefarious crimes into the present day. What follows are brief vignettes of some infamous poisoners that have, fortunately, been caught in their evil deeds, and we can learn a great deal from their cases. I have selected these cases, arranged in chronological order, from my collection of incidents of homicidal poisonings that have revealed various important facets of this type of crime.

1.2.1. William Palmer, MD, “The Rugeley Poisoner” (1855)

In 1855, Dr. William Palmer, of Rugeley, Staffordshire, was a physician with a gambling problem. Motivated by the gain of easy money, he poisoned a fellow horse-racing gambler named John Parsons Cook. Palmer’s poison of choice was the heavy metallic element antimony. Ultimately uncovered in his crime, he was forced to stand trial. Interestingly enough, a change in venue was deemed necessary, in order to obtain a more fair trial, so the trial was moved from the small town of Rugeley to London. (The legislative action for this move is still called the Palmer Act in England.) Dr. Palmer was convicted, and it is very possible that he was involved in as many as 14 other murders. As the crowd hissed “poisoner!” he was hanged for his crime on June 14, 1856 (Lewis, 2003).

1.2.2. Edward William Pritchard, MD, “The Philandering Poisoner” (1865)

In 1865 in Glasgow, Scotland, Dr. Edward William Pritchard took a mistress. To eliminate his wife Mary Jane Palmer, he poisoned her and her mother, Mrs. Taylor, by using antimony in the form of the compound “Tartar Emetic.” As the attending physician, he then conveniently certified the deaths of both women as resulting from gastrointestinal (GI) disturbances. An anonymous letter sent to the authorities eventually led to the arrest of Dr. Pritchard, and having been found guilty of the crimes, he was hanged on July 28, 1865, the last public hanging in Scotland (Roughead, 1925).

1.2.3. George Henry Lamson, MD, “The Slight-of-Hand Poisoner” (1881)

Dr. George Henry Lamson was an English physician who, after the Crimean War, suffered from an addiction to morphine and was in need of funds. To bring family estate funds into his domestic control, in December 1881, he selected as his victim his 18-year-old handicapped brother-in-law, Percy Malcolm John. While visiting John, and having tea and a Dundee raisin cake, he made a big deal of showing his relative a new American invention, the gelatin capsule, stating that it would make taking medicine much easier. To illustrate his point, he filled a capsule with sugar and asked John to take it. A few hours later, after Dr. Lamson left by return train for London, John began to suffer from severe stomach distress and soon died. Dr. Lamson was eventually caught and charged, after trying to bribe the newspapers with inside knowledge of John’s death. How did the poison get into the victim? Not in the capsule; Dr. Lamson had carefully tampered with some of the raisins in

the slice of Dundee cake given to John, using the powerful alkaloidal poison aconite. His reward for this crime was his death by hanging on April 28, 1882 (Adam, 1951).

1.2.4. Thomas Neill Cream, MD, “The Lambeth Poisoner” (1891)

The case of Dr. Thomas Neill Cream presents us with a rather unique motive. Dr. Cream was a sadist and moral degenerate who took out his perverse feelings on prostitutes in the Lambeth area of London. His modus operandi was to offer capsules containing strychnine to the unfortunate victims under the guise that it was a medication to improve their complexions. The victims quickly died agonizing deaths. London, in its post-Jack-the-Ripper climate, soon named this unknown and demented serial killer the “Lambeth Poisoner.” Cream eventually drew attention to himself when he offered to reveal to the authorities the identity of this infamous killer for a sum of many thousands of pounds. Cream was placed on trial, and it took the jury only 12 minutes to return a guilty verdict. He was hanged on November 15, 1892 (McLaren, 1993).

1.2.5. Cordelia Botkin, “The Scorned Poisoner” (1898)

A femme fatale, Cordelia Botkin chose to poison her feminine rival, the wife of her paramour. In San Francisco, California, Cordelia had begun a romantic relationship with John Dunning, a correspondent for the Associated Press. In 1898, John was assigned to cover the breaking Spanish-American War in Puerto Rico and Cuba and informed his mistress that he would not be returning to San Francisco, but to his wife (the daughter of John Pennington, a congressman and former state attorney general) and family in Dover, Delaware. On August 9, 1898, an unsolicited box of chocolate candies addressed to Mrs. Dunning arrived with the mail at the Pennington home in Delaware. Mary Dunning shared the candies with her sister Mrs. Ida Henrietta Deane and two children. Shortly thereafter, they all became violently ill, and subsequently the two women succumbed from severe stomach ailments. With four people becoming ill at the same time, the candies became suspect, and some of the remaining candy, as well as the victims’ bodies, were found to contain large quantities of arsenic. John Dunning quickly returned from Cuba, and he identified the handwriting on the package as Cordelia Botkin’s; she was quickly arrested in California. After a jurisdictional fight between the states of California and Delaware, the trial was held in California, the location of the poisoner. Cordelia was found guilty of the crime and sentenced to life imprisonment. She eventually died in San Quentin prison in 1910, from “soft-

ening of the brain due to melancholy.” This was the first known case of the US mail being used to transport a poisonous weapon to be used to commit murder (Alstadt, 2001).

1.2.6. Johann Otto Hoch, “The Stockyards Bluebeard” (1892–1905)

Johann Otto Hoch was an opportunistic serial killer who used arsenic as his weapon of choice. Between 1892 and 1905, in various US states, he is thought to have murdered possibly 12 of his 24 wives, in order to obtain control of their financial assets. Hoch moved from town to town, gaining the affections of new widows; endearing himself; marrying them; and, soon after, taking control of their finances. Each new wife would soon become ill, suffering from tremendous GI upsets. After the wife’s death, Johann would leave town with all the assets of the deceased. He would then move to a new town, check the obituaries in the local newspaper for widows, select a new target, and begin the process again. Eventually, authorities were alerted to the similarities in the deaths. Hoch was arrested, whereupon it was found that he carried in his pocket a hollow fountain pen containing a white powder, which proved to be arsenic. He claimed that the poison was his “exit dose” to be used when he intended to commit suicide; however, on further interrogation, he confessed to the many murders. Hoch stated: “Marriage was purely a business proposition to me. When I found they had money, I went after that.” He was found guilty of homicidal poisoning and was hanged on February 23, 1906, in Chicago, Illinois (Gaute and Odell, 1979, p. 128).

1.2.7. Hawley Harvey Crippen, MD, “The Mild Mannered Murderer” (1910)

The Crippen case is one that contains many unusual aspects, and many unanswered questions. Dr. Crippen, born in Coldwater, Michigan, in 1862, eventually went on to represent the firm Munyon’s Homeopathic Remedies, in London.

Dr. Crippen was a relatively small man, standing only 63 inches tall, and very quiet in demeanor. His second wife, Kunigunde Mackamotzki (a.k.a. Cora Turner, a.k.a. Belle Elmore), on the other hand, was a rather loud and brassy woman, with a very domineering personality. For several years prior to the disappearance of his wife, Dr. Crippen had been carrying on an affair with his office secretary, Ethel Le Neve. Sometime after the evening of January 31, 1910, Cora simply vanished. Things might have gone better for him if his mistress Ethel had not quickly moved into the Crippen home and begun to wear Crippen’s wife’s clothes and jewelry. Soon, social acquaintances of the

Crippens became suspicious and took their concerns to Scotland Yard. On questioning, Dr. Crippen changed his story of his wife's whereabouts numerous times, at first claiming that she had returned to America and died. Eventually he said that she had left him for another man. He might have gotten away with the crime if he had not panicked after being interrogated and made a dash for Canada by ship. Ethel traveled with Crippen disguised as his young son, with her hair cut and dressed in a young man's suit of clothes. On returning to the empty Crippen home, Inspector Walter Dew of Scotland Yard serendipitously came upon pieces of human tissue wrapped in a man's pajamas, along with hair in hair curlers, buried under the floor of the coal cellar.

An alarm quickly went out all across Europe for Crippen and Le Neve. On board ship, Dr. Crippen and Le Neve were soon identified by the captain, and a radio message was sent back to England alerting the authorities of the fugitives' presence among the passengers. This was the first time in history that the newly developed Marconi radio wireless was used in the apprehension of a criminal. Inspector Dew boarded a faster ship and was waiting for the fugitive pair as they were ready to disembark in Canada. They were arrested and taken back to England to stand trial for the murder of Mrs. Crippen. The case is interesting in that no head, no limbs, no bones, and no organs of gender were found, and the case hinged on the fact that the tissue discovered was found to contain the toxic alkaloidal compound hyoscyne (scopolamine), which had not been known ever to have been used in a poisoning homicide up to that time. It was also proven that Dr. Crippen had purchased hyoscyne, to use—he claimed—in the preparation of his homeopathic formulations. Ethel Le Neve was found not guilty of any involvement in the death of Mrs. Crippen, but Dr. Crippen was found guilty, and he was hanged on November 23, 1910.

More than 37 books have been written on the Crippen murder, and the name Crippen has even become a synonym for poisoner in the British language. Many students of the case have asked, why did Dr. Crippen not simply walk away from his unhappy marriage in the first place? Also, why did he apparently dismember his wife's body, which certainly did not point to a natural death? Hyoscyne was used at the time, in certain institutions, for a sedative effect. It is possible that he might have found it difficult to be sexually involved with two women at the same time and in an effort to depress her sexual appetite by giving her hyoscyne he had accidentally overdosed her (which would not be a hanging offense). Perhaps he had shot her—there was a gun in the home (which never came out at the trial)—and then realized that he had to get rid of the body. We probably will never have the true answers to these questions. But Crippen and Le Neve have been immortalized in wax in Madame

Tussaud's Waxworks' "Chamber of Horrors" in London, for all visitors to look upon their visages and wonder.

I was a member of the London research team investigating the many unanswered questions about the Crippen case in a documentary made for the History Channel's *The Real Dr. Crippen* and am now involved in a research project to utilize mitochondrial DNA to examine the actual remains removed from the grave from the Crippen property (Cullen, 1977).

1.2.8. Frederick Seddon, "The Poisoning Miser" (1911)

Frederick Seddon was a miser. In an attempt to gain easier access to the financial assets of another person, he took a boarder named Elizabeth Barrow into his London home. Frederick soon convinced the woman to assign him controlling interest in her annuities in exchange for his promise to care for her for the rest of her life. After several episodes of severe stomach distress, Elizabeth died in the Seddon home on September 14, 1911. Suspicious relatives soon arrived to take possession of the dead woman's estate, but Seddon told them that there was nothing left to turn over. They went to the police with their suspicions, and it was soon determined that the victim's body contained massive amounts of arsenic. Frederick and his wife, Mary, became prime suspects in Elizabeth's untimely death. It was proven that Mary Seddon had purchased a large number of insecticidal fly papers, which contained arsenic, and it was speculated that the deadly poison had been soaked from the product and given to the deceased. Mary was eventually found innocent of any crime, but Frederick was found guilty and was hanged on April 18, 1912 (Adam, 1913).

1.2.9. Henri Girard, "The First Scientific Murderer" (1912)

The case of Henri Girard is important in this collection because it represents one of the first known uses of biological agents to carry out a poisoning homicide. The financial manipulator Henri Girard, purportedly a rather dashing-looking Parisian, made it his practice to insure the lives of various acquaintances and have himself listed as their primary beneficiary. These people soon died under mysterious circumstances by his hand. Girard's poisonous weapons of choice were the natural toxins from the mushrooms of genus *Amanita*, as well as various pathogenic bacteria. Soon after the deaths of Girard's acquaintances Louis Pernotte and Madame Monin, the insurance companies became very suspicious, and an investigation ensued. Girard was taken into custody in 1912 but cheated the French court by taking one of his own toxic germ cultures (most likely typhoid), which he had secreted in his personal effects (Kershaw, 1955).

1.2.10. Arthur Warren Waite, DDS, “The Playboy Poisoner” (1916)

The first dentist in our collection, Dr. Arthur Warren Waite was a good-looking raconteur who most likely preferred playing tennis to practicing dentistry. He grew up in Grand Rapids, Michigan, and after graduating from dental school went to South Africa to practice. Waite eventually left South Africa after receiving suspicious accusations and returned to Michigan, where he wooed and married the daughter of John and Hannah Peck. John Peck was a millionaire pharmacist who owned a reputable drug company in the city. The grateful Pecks furnished the newlyweds with posh accommodations in New York City. There, Arthur spent much of his time dabbling in the area of bacteriology and also took on a Mrs. Horton as his mistress.

In January 1916, shortly after Hannah Peck arrived to visit the Waites in New York, she suddenly became ill and died. Her body was immediately cremated and returned to Michigan for burial. In March of the same year, John Peck also went to New York, to console his daughter and her husband over the death of his wife. He, too, soon became ill and died. However, before his body could be cremated, his son received an anonymous telegram, in Grand Rapids, stating “suspicion aroused, demand autopsy.” Surprisingly, the autopsy indicated that John Peck was loaded with arsenic, and an investigation ensued. The accusing finger eventually pointed to the playboy dentist, and the police took him in for interrogation. A search of his dwelling revealed numerous bacterial cultures, as well as texts dealing with toxicology. While under interrogation, Dr. Waite changed his story numerous times. First he stated that he had obtained arsenic for his father-in-law, who wanted to commit suicide to end his grief over the loss of his wife. Then Dr. Waite claimed his own body was inhabited by the spirit of an evil Egyptian priest who had instructed him to kill his in-laws in order to gain their wealth. Eventually, Dr. Waite felt that if he told what had actually happened the courts would find him insane, so he revealed the whole story of administering typhoid, pneumonia, and diphtheria organisms, as well as arsenic while the Peck’s were undergoing dental work by him. It did not take the members of the jury long to see through Dr. Waite’s manipulations, and they convicted him of the murders. Dr. Waite was electrocuted at Sing Sing Prison on May 24, 1917.

1.2.11. Murderers of Mike Malloy, “The Case of the Man Who Wouldn’t Die” (1933)

The case of Mike Malloy is a rare case of multiple individuals offending a single victim, which, in retrospect, is almost humorous in some respects. In January 1933, America was in the middle of the Great Depression. A group of

men in a speakeasy in New York City devised a plan to make some easy money: they would insure the life of someone and then murder him to collect on the policy. The group, consisting of Dan Kreisberg, a local grocer; Joseph “Red” Murphy, a bartender; Anthony “Tony” Marino, the bar owner; Anthony “Tough Tony” Bastone, a hit man; Francis Pasqua, an undertaker; Hersey “Harry” Green, a taxi driver; and Dr. Frank Manzella, an ex-alderman, chose as their patsy a well-known Irish alcoholic and skid-row resident named Mike Malloy (one tough old Irishman!), who happened to come through the door of the establishment. After the \$1788 in insurance policies were initiated, the group began to offer Malloy free drinks and food, which he thought was very generous coming from new friends. However, the plotters added horse liniment, turpentine, and sometimes antifreeze to his drinks, and the free food was filled with rat poison, carpet tacks, and other potentially harmful foreign bodies. Unfortunately for the group, Malloy seemed to ingest these substances with little harm. Becoming desperate, the men then got Malloy drunk, doused him with water, and threw him out into a park in the middle of a winter storm, hoping that the temperature would do the job. However, the next day, their victim returned for more of their hospitality!

The group then stood the drunken man up in the middle of the street and had Harry Green strike him with his taxi. Astoundingly, Malloy survived this encounter as well. Feeling that they were working too hard for their money, the men decided to carry out the murder once and for all. They took the drunken Malloy to his bedroom, ran a hose from the gaslight on the wall down his throat, and killed him with coal gas (carbon monoxide). They collected their insurance money, and all would have gone well if one of the group had not bragged about their “project” (a problem often seen when multiple offenders are involved in a crime). When the police were informed, the entire group of seven individuals was placed on trial for the murder. The outcome was not what the group of men had envisioned when they started out on their money-making scheme: four (Marino, Pasqua, Murphy, and Kreisberg) were electrocuted at Sing Sing, on June 8, 1934, for the crime. Green and Manzella received prison sentences. The seventh individual, Bastone, was shot in a dispute over the money (Read, 2005).

1.2.12. Reverend Frank Elias Sipple, “The Poisoning Pastor” (1939)

Sometimes even a minister can be a poisoner. In 1939, in Grand Rapids, Michigan, the Reverend Frank Sipple was a spiritual leader of the Southlawn Church of God, in the suburb of Wyoming. He decided to murder his daughter, Dorothy Ann, and gave her a capsule containing cyanide. The act was carried out one Sunday morning just before he left for the church to deliver

his weekly sermon. The medical examiners determined Dorothy Ann's death to be the result of a heart attack, missing the true cause of the girl's death, and she was buried without further investigation. The homicide remained undetected until 1946, when Pastor Sipple attacked one of his church elders with a lead pipe in a dispute over church politics. The gentleman who had been the target of the pastor's attack also claimed he thought the minister had once given him candies that had been tampered with. Under interrogation by the police, Sipple admitted not only to the attack on the parishioner, but also to the poisoning death of his daughter many years earlier. The suspect stated that his daughter was mentally disturbed and that he thought she would be better off dead than having to spend the rest of her life in a mental institution. There was some speculation that his daughter might have had some information on the untimely death of Sipple's first wife in Illinois and was possibly going to take the matter to the local authorities. The court found Reverend Sipple guilty of murder, and, in 1946, he was sentenced to life in prison. Ill with terminal cancer, he was released from prison in December 1959 to return to Grand Rapids, where he died just 14 weeks later.

1.2.13. Sadamichi Hirasawa, "The Poisoning Bank Robber" (1948)

On January 26, 1948, a mass killer with a most unique plan struck at the suburban Shiinamaki branch of the Teikoku Imperial Bank in Tokyo, Japan. Sadamichi Hirasawa, pretending to be a Dr. Jiro Yamaguchi, entered the bank's facility at closing time, telling the 14 bank employees that they must drink some medicine to prevent an outbreak of amoebic dysentery then rampant in the district. The employees obediently swallowed teacups full of a liquid heavily laced with the deadly poison potassium cyanide. Thirteen of the bank's employees died on the spot, at which time Hirasawa looted the bank of more than 180,000 yen (then about \$600) and vanished into the general population. In one of the largest manhunts in Japanese history, the police laboriously interviewed thousands of people who had received business cards from a man pretending to be a physician and, finally, pinpointed Hirasawa. Sadamichi was identified by the lone surviving bank employee, admitted his guilt, and was imprisoned for life. After spending 40 years on death row, he died in 1987, gaining international fame as the longest resident on death row anywhere in the world.

Recently there has been speculation that Sadamichi might have been innocent of the robbery, and that the crime was actually committed by a renegade member of the Japanese army's disbanded and very secret "Unit 731", which, during World War II, had carried out bacteriological research experiments on human prisoners of war in Manchuria. In Japan, a campaign contin-

ues to this day for the posthumous overturning of his conviction for this crime (Triplett, 1985).

1.2.14. Christa Ambros Lehmann, “The Poisonous Neighbor” (1954)

The Lehmann case is interesting because of the relatively common background of the poisoner and the poison she selected. In February 1954, in the town of Worms, Germany, Christa Lehmann purchased five chocolate truffle candies at a local shop and delivered four of them to her friends, keeping one candy as a special gift for a woman who had been objecting to Christa’s association with members of the woman’s family. The targeted victim, instead of eating the candy, placed it in a kitchen cupboard as a treat for her daughter to eat. When the daughter sampled the treat, she complained of the bitter taste and dropped it on the floor, where it was quickly consumed by the family dog. Within a short time, both the girl and the pet were dead, and the attending physician was somewhat puzzled by the common symptoms and sudden fatalities exhibited by the two victims. Eventually the cause of the deaths was traced to a relatively new chemical substance called E-605, which had been developed as a potent insecticide by the Germans during World War II. We now know this substance as parathion. It acts much like a nerve-gas agent, causing rapid alterations in a person’s autonomic nervous system and a characteristic set of symptoms, eventually leading to death. Suspicion fell immediately on Christa as the poisoner of the candy, and during police interrogation, she confessed to lacing it with E-605. While in police custody, she also admitted to killing her husband and her father-in-law with the same toxic compound. The court sentenced her to life in prison. Unfortunately, the discussion in the press of the poison and its potential soon led to a rash of E-605 suicides in Germany among individuals depressed over the state of the country after the war.

1.2.15. Arthur Kendrick Ford, “The Accidental Poisoner for Sex” (1954)

The case of Arthur Kendrick Ford illustrates that not all poisonings are murder in the first degree. Arthur Ford was infatuated with two female coworkers in his London chemical company and decided he needed some chemical assistance in gaining their sexual attentions. Having heard about the effects of Spanish Fly as an aphrodisiac, he obtained the natural form of cantharides from the firm’s stockroom, stating that he needed it to breed rabbits. On April 27, 1954, Arthur entered the company’s office and offered three of the secretaries coconut candies in which he had placed large doses of the powdered cantharides. Neither he nor the unfortunate women knew the

horrible physical torment they would soon endure. Cantharidin, which is derived from the ground-up bodies of a Mediterranean beetle, is a powerful blistering agent normally used in dermatology to burn off warts. The corrosive effect of this compound on the human anatomy is disastrous. After only a few hours, all three women were hospitalized in torment, two of them dying from the ill effects. When autopsies revealed the cause of the deaths, Ford broke down while being interviewed and confessed to his involvement. He was placed on trial, but because it was not his intent to murder, he was convicted of manslaughter and sentenced to only 5 years in prison.

1.2.16. Nannie Hazel (“Arsenic Annie”) Doss, “The Poisonous Romantic” (1952–1954)

Nannie Doss was a female serial killer, if there ever was one. By the time she was finally detected, she had successfully poisoned 11 victims: 5 husbands, 2 children, her mother, 2 sisters, and a nephew. Nannie, a housewife living in Tulsa, Oklahoma, first came to the attention of the local authorities in 1954 when a suspicious physician decided to perform an autopsy on her deceased fifth husband. The analysis revealed an amount of arsenic equal to 20 lethal doses. The exhumation and toxicological analyses of other members of her family who had died over the years also revealed the presence of arsenic. On interrogation, her crimes came to light, and she stated that she had done away with her husbands because she had found them dull. This was probably related to the fact that Nannie’s favorite reading material consisted of romance magazines and her domestic life had not measured up to her romantic fantasies. She said, “I was just searching for the perfect mate, the real romance of life.” Found guilty of the multiple murders, in 1955, she was sentenced to life in prison, where she died of leukemia in 1965 (Nash, 990, p. 1006).

1.2.17. Graham Frederick Young, “The Toxicomaniac” (1971)

Perhaps one of the most fascinating of the poisoner personalities is that of an Englishman named Graham Young; he was a “toxicomaniac,” or a person obsessed with poisons. The poisons gave him a feeling of power over other people, and he used poisons throughout his life to nefarious ends. When Graham was 11 years of age, his father gave him a chemistry set for his birthday, and from that time on he followed his obsession with chemistry and toxicology. He read incessantly about the crimes of the infamous poisoners and became very conversant with the subject of poisons. He once told his sister that he would become more famous than the well-known British poisoners Palmer, Pritchard, and Crippen. Graham’s stepmother died when he

was 14 years old, and no one suspected that he had played a key role in her death by administering an antimony-containing compound. Other members of his immediate family, as well as school friends, also became subjects for his toxicological experiments. In 1962, when one of his teachers accidentally found strange notes and drawings in Graham's school desk, the authorities were called in to investigate. In his room at home, they found enough various poisons to kill almost 300 people, along with an extensive reference library on poisons. He was remanded to Broadmoor Criminal Lunatic Asylum where, after only nine years, the asylum's psychologists deemed him rehabilitated and released him.

In 1971, Graham began working at a photographic optical firm in Bovington, England that specialized in the production of high-quality optical lenses. In the production of these lenses, the company utilized the deadly poisonous element thallium, which coincidentally happened to be one of Graham's favorite poisonous tools. One of his jobs at the facility was passing out the daily tea on breaks. A wave of illness soon spread throughout the company, and two of his coworkers died from a supposed viral nervous system illness. When the company doctor was called in to address the concerns of the employees over what had become known as the "Bovington Bug," Graham drew attention to himself by freely spouting his knowledge of toxicology, and what he felt the physicians had missed in their diagnosis of a viral cause. He said that they failed to see that the symptoms were much more consistent with thallium poisoning. A review of Graham's past soon revealed that he had been hospitalized for his poison mania, and a search warrant was obtained for his lodgings. In his room the investigators found a diary that revealed the names of the individuals he had selected for his toxicological experiments, and notations on the effects of the administered poisons over the course of their intoxications. While he was awaiting trial Graham boasted that he had committed the perfect crime in 1962, in the killing and subsequent cremation of his stepmother. In June 1972, Graham was found guilty of two murders, two attempted murders, and two charges of administering poisons and was sentenced to life in prison. In 1989, at age 42, Graham died in prison of a heart attack. He is still one of the cruelest yet forensically fascinating poisoners in history (Holden, 1974; Young, 1973).

1.2.18. Ronald Clark O'Bryan, "The Halloween Killer" (1974)

The public despises poisoners for their lack of sympathy toward their victim (or victims), and rightly so in the case of Ronald Clark O'Bryan, who killed his own child to obtain money from an insurance policy. On Halloween

1974, in the town of Pasadena, Texas, 8-year-old Timothy Marc O'Bryan died after ingesting Pixy Stix®, candy straws filled with fruit-flavored powder. Examination of the treats revealed that they also contained potassium cyanide. Contaminated treats were also found in the candy bags of Timothy's sister, and those of three other children from the neighborhood. Mr. O'Bryan, who had accompanied the children around the neighborhood on their trick-or-treat activities, stated that the poisoned candies had been given out at the home of a rather shadowy figure that he could not identify. Police investigation eventually revealed that the 30-year-old O'Bryan had made inquiries around his workplace concerning cyanide and had recently taken out a \$65,000 insurance policy on his son. The court found him guilty of the murder, and he was executed—ironically, by lethal injection—on March 31, 1984 (Sauke, 2003).

***1.2.19. Rev. James Warren Jones,
“The Minister Who Went Mad” (1978)***

Many people can easily remember the television news scenes of November 18, 1978, showing 913 people lying dead in the sun in a jungle compound in the South American country of Guyana. This case represents one of the greatest mass suicides (murders?) involving poison in recent history. The pivotal personality involved in this incident, Rev. James Warren Jones, did not administer the poison with his own hands, but he certainly was the instigating force in this terrible event. Jones, who founded a communal group known as the People's Temple, had taken his flock to the jungles of Guyana and founded a spiritual refuge known as Jonestown.

Jones's hold over his followers was a prime example of the famous quote by Lord Acton that “power tends to corrupt, and absolute power corrupts absolutely” (cited in Kaplan, 2002, p. 554). In Guyana, Jones eventually lost touch with reality, becoming extremely paranoid in his view of the outside world. The triggering event to the mass poisoning was the visit of California Congressman Leo Ryan to investigate allegations made by the families of some of his constituents about Jones's hold over their family members. Congressman Ryan, and many other members of his entourage, were shot and killed at Jonestown, by Jones's followers, and then Jim Jones ordered his followers to carry out the “White Night” suicide exercises that they had practiced so many times as a test of their faith for their pastoral leader. A large container of fruit drink containing cyanide and sedatives was soon concocted, and many of the people lined up and voluntarily drank the deadly creation. Some, however, were less than willing to die for Jones's cause; many of their bodies bore signs that the poison had been injected by force. Jones's body was also recov-

ered from the commune death scene, but the cause of his death was a bullet to the head. A review of this terrible tragedy in Guyana reminds one of the Euripides saying “whom the gods destroy, they first make mad.”

1.2.20. Murder of Georgi Ivanov Markov, “The Umbrella Assassination” (1978)

Murdering a victim by means of poison can also be a political act. The Markov case represents a most unique murder with poison, because of the means of administration. Georgi Markov was a Bulgarian defector living in London and working for the BBC broadcasting pro-Western propaganda back to his Communist-controlled homeland. While going to work on the morning of September 7, 1978, Markov felt a stabbing pain in his thigh, and a man in the crowd behind him suddenly dropped and then quickly picked up an umbrella. The unknown man apologized for bumping into him, then entered a taxicab and disappeared. Over the next several days, Markov became increasingly ill, and medical teams were unable to discover the cause of his symptoms and of the changes that were happening to his normal blood constituents. Within four days of the event, Markov was dead. An autopsy revealed a small bruise on his thigh, which, when excised, revealed a metallic sphere, about the size of the ball on the end of a ballpoint pen, with holes drilled into it. Although no poison could be detected in this metal object, the toxicologists generally agreed that the poison that induced Markov’s symptoms was most likely ricin, a highly toxic plant substance found in the castor bean (*Ricinus communis* L.). Georgi Markov’s assassin was never found, and after the fall of the Soviet Union, it was revealed that its “Laboratory 12” had developed the assassination weapon. Ricin was indeed used by the Bulgarian State Security organization Durzhavna Sigurnost in an umbrella mechanism for the means of injecting the poisoned sphere into the organization’s victims. The case, recently brought by Markov’s widow, is currently in the courts (Bereanau & Todorov).

1.2.21. Unknown Offender, “The Tylenol® Tamperer” (1982)

In October 1982, a series of incidents occurred in Chicago that were to change forever the manner in which over-the-counter (OTC) medication was to be sold in the United States. Seven people were to fall victim to a tamperer when they innocently took Extra Strength Tylenol® capsules that had been laced with cyanide. Of the seven victims (three from the same extended family), six died almost immediately, and one lived for 2 days before succumbing to the effects of the poison. It took some time before investigators were able to determine that the common factor in all the deaths was that all seven

victims had taken the pain reliever. As a result of this incident, the product's manufacturer, Johnson & Johnson, immediately recalled all packages of its analgesic product on a national level and reformulated both the capsule format and packaging to make them more tamper resistant. In its prompt attention to the problem, the company was able to save its credibility with the public and set a standard for other manufacturers for handling any similar future incidents. Although an extensive investigation ensued, sufficient evidence to warrant the arrest of an individual for this heinous crime was never obtained. As a result of this incident, tamper-resistant packaging has become a norm in the US marketplace.

1.2.22. Stella Maudine Nickell, "The Camouflaged Poisoner" (1986)

In poisoning cases, things are not always as they first appear. The case of Stella Nickell is a perfect example of what appeared to be a death resulting from a tampering incident but was actually an attempt to cover up a very carefully planned homicide.

In Auburn, Washington, Nickell's husband, Bruce, died of what was believed to be emphysema. The cause of his death was actually cyanide, which his wife had administered in an attempt to collect on a \$175,000 life insurance policy. Unfortunately for Stella Nickell, a natural cause of death did not pay as much as an accidental death. Out of this dilemma, she concocted a plan.

Not long after Bruce's death, a young woman named Susan Snow collapsed and died in the bathroom of her own home, after taking an OTC pain reliever. On autopsy, cyanide was detected in the unfortunate woman. An investigation of her movements just before her death led to the discovery of a bottle of pain reliever capsules in her medicine cabinet that had been tampered with, and cyanide was found as the tampering agent. A rapidly instituted recall by the product's manufacturer revealed several other bottles of tampered medication in different locations. The case took an interesting turn when Nickell called the authorities to report that she thought her husband had also been a victim of this tainted pain reliever. Exhumation revealed that Bruce had also died of cyanide poisoning, and two bottles of contaminated capsules were found in the Nickell home. Stella said that she had purchased these bottles at two different stores, but it did not take the authorities long to realize that they were talking either to the most unlucky purchaser in history, or to someone who might know more about this tampering incident than it first appeared. Evidence began to mount against Stella, and members of her family voiced suspicions. In addition, authorities found small green flecks of material in the cyanide, which was eventually identified as an algae destroyer used in home aquariums. The home of Stella Nickell contained many such aquariums. A

forensic investigation of books at the local library eventually revealed many of Stella's fingerprints on toxicology books dealing with cyanide and other toxic compounds. A jury found Stella guilty of two murders, and she was sentenced to two 90-year terms in prison. In June 2002, an appeal was made in federal court to throw out Stella's conviction owing to suppression of evidence, namely that Stella's daughter received a financial reward for her testimony from an entity affiliated with drug manufacturers, and that conflict of interest may have influenced her testimony (Olsen, 1993).

1.2.23. Donald Harvey, "The Angel of Death" (1983–1987)

"The Angel of Death" was a serial killer who struck at victims within the health care system over a period of four years, from 1983 to 1987. Donald Harvey, a nurse's aide, used multiple methods to bring the lives of many patients under his care to a rapid end. Some were smothered, and some he poisoned with arsenic, cyanide, or morphine. His crimes eventually came to light in Cincinnati, Ohio, when a pathologist was able to detect the odor of cyanide on one of the hospital victims on whom he legally had to perform an autopsy. Further exhumations and autopsies were performed on other patients who had died unexpectedly during a given time period, and traces of poison were found in many of the bodies. One of the common factors shared by these victims was that they were cared for by Donald Harvey. On interrogation, Harvey admitted to the killings and was placed on trial. Court psychiatrists determined that Harvey had a personality disorder that resulted in a compulsion to kill, but that he was not insane. After pleading guilty to 24 murders, Donald Harvey was sentenced to three consecutive life terms in prison. Thus was incarcerated one of the most prolific medical poisoners in the history of the United States.

1.2.24. George Trepal, "The Eccentric Genius" (1988)

George Trepal was probably one of the most intelligent poisoners ever encountered in the United States. His IQ qualified him for membership in Mensa, a select group of individuals with proven high intelligence representing probably only 2% of the general population.

The case began in 1988, in Bartow, Florida, when several members of the Carr family suddenly became ill. Something unknown was causing paralysis and slow destruction of their nervous systems. The condition was thought to be the result of a virus, and they were hospitalized and provided whatever supportive care was possible. Eventually the mother of the family, Peggy Carr, succumbed from her condition, and one of her sons was permanently disabled by the effects on his nervous system. Suspicion fell on the heavy-metal poi-

son thallium as the cause of the family's maladies, and a search began for a possible environmental source of the substance. The investigators finally discovered the element in individual screw-cap bottles of Coca-Cola Classic® from a package that the family had been consuming over a period of time. The question was, how did this very toxic substance wind up in this consumer product? Obviously, someone had tampered with the bottles. During the extensive investigation, the person who came to light was George Trepal, a neighbor who had openly voiced some displeasure with the members of the Carr family over various neighborhood issues. An undercover police investigation revealed that Trepal had had access to the family, a motive, and a great deal of knowledge about chemistry. A search of his home revealed a container with traces of thallium. Trepal was found guilty of the tampering murder and was sentenced to die for the crime. At the time of this writing, he is awaiting execution on death row in Florida (Good and Goreck, 1995).

**1.2.25. Michael James Swango, MD,
“Double-O-Swango—License to Kill” (1983-1997)**

Michael Swango, talented pianist, National Merit finalist, and class valedictorian, was a physician who had everything going for him, yet something forced him over the edge to become a serial poisoner. Between 1983 and 1997, Dr. Swango is thought to have killed many patients in many US states, as well as in Africa. Since childhood he had a fascination with violent death, often collecting newspaper clippings associated with such events. For his senior thesis he chose the Markov murder, and his favorite film was *Silence of the Lambs*. Even his medical school classmates gave him the nickname “Double-O-Swango” in reference to the James Bond character. He kept a collection of books on poisons and maintained recipe cards on poisonous substances. It is unknown how many people he killed using arsenic and drugs, because he traveled from one hospital to another across the United States. Unfortunately, the hospitals he left did not pass on their suspicions to the next hospital in which he was about to practice. Eventually he traveled to Zimbabwe, where more suspicious deaths occurred. Dr. Swango was taken into custody on his return to the United States and interrogated. He was later placed on trial for multiple murders. In 2000, he plead guilty to four murders and was given three life sentences without parole (Stewart, 1999).

1.2.26. Harold Frederick Shipman, MD, “Dr. Death” (1974-1998)

Dr. Harold Shipman will probably be remembered as one of the most heinous serial killers in British history. Certainly Shipman was responsible

for a high body count. It is thought that Shipman, a family doctor operating out of his office in Hyde, Manchester, England, may have killed at least 215 patients (mostly women) between 1974 and 1998 by utilizing opiate drugs. Suspicions were aroused after his final murder, because for the first time in any of the deaths he apparently had had a financial motive and had forged a will in his favor. Investigations soon revealed that he was associated with multiple sudden deaths in patients who before his visit to their home were not clearly at death's door. It is thought that his motive in most of the killings was his need to have power over others. In 2000, he was sentenced to life in prison for 15 murders. On January 13, 2004, on the day before his 58th birthday, he was found hanging in his prison cell—a successful suicide (Whittle and Ritchie, 2001).

1.2.27. Ryan Thomas Furlough, “The Poisoner for Love” (2003)

Poisoners do not necessarily have to be medical personnel, or very old, to carry out a crime. Ryan Furlough was an 18-year-old high school student, in Ellicott City, Maryland, who was in love with the girlfriend of his friend Benjamin Vassilev. In a letter, Ryan wrote, “There’s not a day that goes by that I don’t think of her. . . . I will never give up until I have the key to her heart.” Soon afterward, he concocted a plan to make him the only recipient of the girl’s affections. Using his parents’ credit card, he purchased cyanide from an Internet source. On January 2, 2003, he invited Benjamin to his house to play video games and offered him a can of soft drink laced with the deadly poison. Almost immediately after drinking it, Benjamin went into convulsions. Ryan called the paramedics, but when they arrived they were unaware of the real cause of the symptoms, and Ryan did not offer any helpful information. The victim died soon after arriving at the local hospital. An autopsy revealed the real cause of death and an investigation ensued. Ryan was convicted of the murder on May 17, 2003, and sentenced to life in prison with the possibility of parole.

1.3. CONCLUSION

This chapter is but a brief overview of only a few of the more infamous people who chose to use poison as their weapon to achieve nefarious ends. We can only imagine the hundreds of individuals who have also used such a weapon but whose crimes have gone undetected.

To read more about infamous poisoners, I recommend the concise work by Michael Farrell *Poisons and Poisoners*, cited in the Suggested Reading section.

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Chapter 2

Types of Poisons

“Poisons and medicines are oftentimes the same substance given with different intents.” —Peter Mere Latham

In this chapter I discuss the nuances of poison as a weapon. What is a poison? What advantages does this type of weapon offer over the more traditional types of death-inflicting instruments? Many substances have been used for homicidal purposes, and they can come from animals, plants, or minerals. See **Fig. 2-1** for just a few examples of substances that have been used by actual poisoners.

2.1. DEFINITIONS

At the outset it might seem simple enough to define what a poison is; however, legally it is not quite as simple as it first appears. In the courts it has oftentimes been difficult to agree on the definition of “poison.” *Scientific American* once humorously defined a poison as

“any substance in relatively small quantities that can cause death or illness in living organisms by chemical action. The qualification ‘by chemical action’ is necessary because it rules out such effects as those produced by a small quantity of lead entering the body at high velocity.”

Humor aside, in the courtroom a great deal of discussion will ensue concerning whether a substance in question is really a poison. Is aspirin a poison? Most would agree that it is a medicinal agent, because people take this substance to relieve pain and fever. However, in sufficient dosages aspirin can be an agent that results in death. So what is a poison? Although definitions may differ according to the laws of various states, to

From: *Forensic Science and Medicine: Criminal Poisoning, Second Edition*
By: J. H. Trestrail, III © Humana Press Inc., Totowa, NJ

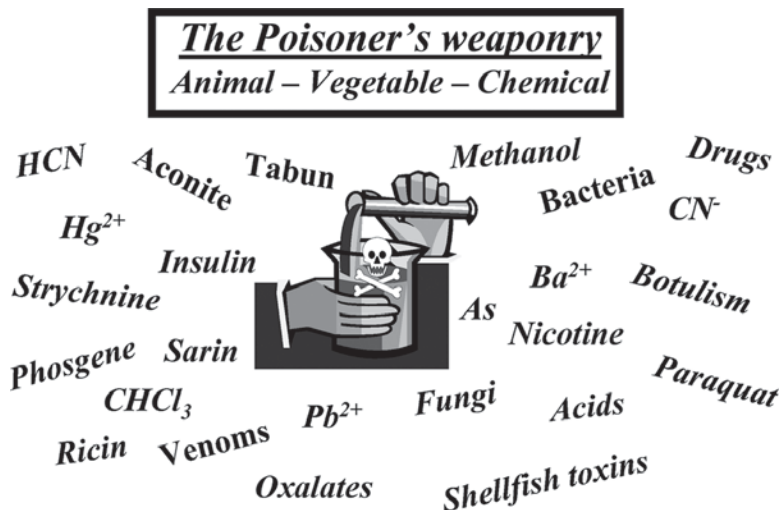


Figure 2-1

follow are some of the definitions of poison that have been cited in the legal literature:

“Poison: Any substance, either taken internally or applied extrnally, that is njurious to health or dangerous to life.” (Stedman’s Medical Dictionary, 27th ed., 2000, p. 1416)

“Poison: Any substance that, when relatively small amounts are injested, inhaled, or absorbed, or applied to, injected into, or developed within the body, has chemical action that causes damage to structure or disturbance of function, producing symptoms, illness, or death.” (Dorland’ Illustrated Medical Dictionary, 29th ed., 2000, p. 1422)

Probably the most astute concept of what constitutes the difference between a poisonous and a nonpoisonous substance was stated by the famous 16th-century alchemist Philippus Aureolus Theophrastus Bombastus von Hohenheim (Paracelsus) (1493–1541), when he wrote:

“What is there that is not poison, all things are poison and nothing (is) without poison. Solely the dose determines that a thing is not a poison.” (Deichman, Henschler, & Keil, 1986)

In this statement, Paracelsus was able to get to the very core of the argument of what determines, or defines, a poison, which is that anything can be a poison—it merely depends on what dose causes deleterious effects. Certainly the relaxation that might come from drinking a relatively small amount of an alcoholic beverage when compared to a death that may result from ethanol

intoxication (e.g., as experienced by a fraternity initiation) is merely dose-related outcome. The same principle applies to the medications that we routinely take to keep our bodies in healthy balance. Toxicology is nothing more than the subject of pharmacology (the study of the actions of drugs) pushed too far, into the “dark side” as it were. As the famous British toxicologist Alfred Swaine Taylor, MD (1806–1880) wrote:

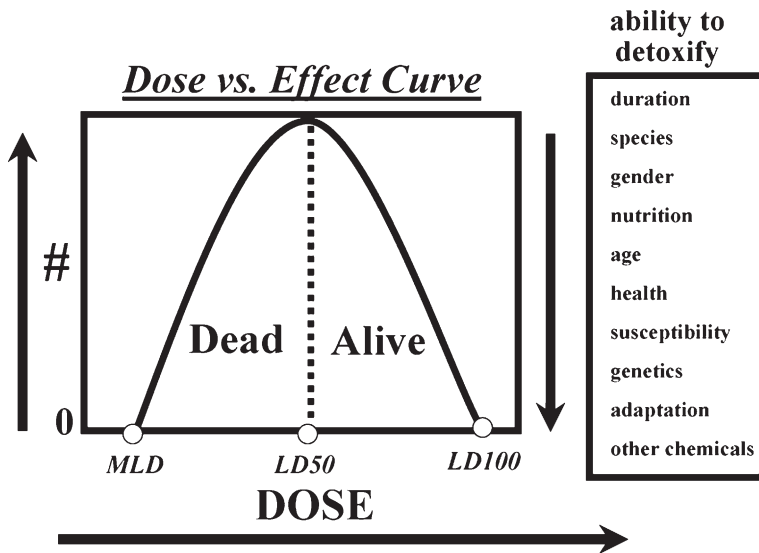
“A poison in a small dose is a medicine, and a medicine in a large dose is a poison.” (Taylor, 1859, p. 2)

When plotting out a dose vs effect curve, one will see the following points: the minimum lethal dose (MLD), which is the lowest dose that has been documented to have resulted in a fatality; the LD₅₀, which is the dose that results in a lethal outcome in 50% of a test population; and the LD₁₀₀, which is the dose that will kill 100% of a test population. People will die at different doses of poisons, owing to their ability to resist or detoxify the toxic substance. Many factors can alter the response of an individual to a dose of poison, including the duration of the exposure, the animal test species, the individual’s gender, nutritional status, age, health status, susceptibility to the material, genetic makeup, and ability to adapt; and the presence of other chemical substances already in the body. These factors help explain how some individuals can tolerate a dose of poison at a level high enough that it would kill other individuals (*see Fig. 2-2*).

One must never forget that although administering too much of a substance can kill, so can administering too little. For those individuals whose lives depend on medication, elimination of the necessary medication or a reduction in the dosage (subtherapeutic dosage) can bring about death. Drugs such as insulin, digoxin, anticoagulants, or anticancer drugs can bring about such a negative effect if the dosages are reduced. For the investigator, a patient’s death may appear to be the result of the patient’s noncompliance with the prescribed dosage, when in fact it is the result of foul play. An example of such a crime would be the case of the Kansas City pharmacist Robert R. Courtney, who, between 1992 and 2001, reduced the amount of anticancer drugs in the prescriptions he dispensed to his customers, in order to increase his profits. His scheme involved some 98,000 prescriptions and almost 4200 patients. For this despicable crime, Courtney was sentenced in 2002 to 30 years in prison with no parole.

2.2. CHARACTERISTICS OF “IDEAL” POISONS

There are certain characteristics to an “ideal” poison, and homicidal poisoners will select their murderous compounds to encompass as many of



these characteristics as possible. What follows are some of the characteristics of such an ideal homicidal poison:

- It should be odorless, tasteless, and colorless. This allows administration to the intended victim without any warning signs that the victim can detect by the normal bodily senses of smell, taste, and sight.
- It should be readily soluble, preferably in water. This allows easy administration in normal foods and drinks of which the victim may partake.
- It should have a delayed onset of action. This allows a time period in which the poisoner can attempt to create an alibi.
- It should be undetectable, and certainly the more exotic the poison the more likely that it will not be detected in more routine toxicological analyses.
- It should have a low-dose lethality, which means less of the toxic material needs to be administered in the dose. It is much easier to administer a pinch of a substance, rather than a pound.
- It should be easily obtained, but not traceable, so that it will leave no investigative trail that would lead to the poisoner.
- It should be chemically stable. This makes it easy to store without loss of potency.
- It should decompose/break down on death.
- It should be found as a naturally occurring substance in the environment of the burial place.

Ideal poison characteristics

- ☒ Tasteless
- ☒ Odorless
- ☒ Colorless
- ☒ Readily soluble
- ☒ Delayed onset of action
- ☒ Exotic
- ☒ Undetectable
- ☒ Low dose lethality
- ☒ Easily obtained (but not traceable)
- ☒ Mimics a natural disease state
- ☒ Chemically stable before administration
- ☒ Decomposes after death
- ☒ Found in the burial environment (*e.g.* Arsenic)

Figure 2-3

The heavy metals (*e.g.*, arsenic, antimony, mercury, lead) are elements or elemental compounds and are as toxic today as they were when first created millions of years ago. These compounds tend to remain detectable many years after a victim's burial, which is advantageous for law enforcement investigators.

Probably one of the greatest aspects desired in a poison is for its effects to mimic a natural disease state, because the poisoning will be missed. A death certificate would then bear an unrelated medical cause of death, and the victim would be buried without detection of an actual crime.

See **Fig. 2-3** for a summary of the ideal characteristics of poison discussed here.

2.3. CONTRASTS BETWEEN POISONS AND MORE TRADITIONAL WEAPONS

Certainly one of the main advantages of poison over the more traditional weapons is that it is invisible as a weapon. One might therefore consider it a "stealth" weapon. The unsuspecting victim never sees it coming and, therefore, cannot provide any form of reasonable defense to the attack.

In effect, a poison is a “chemical bomb.” It does not explode with an external violent force, but, nonetheless, like an explosive, it destroys the internal natural workings of a victim’s physiology. It could be reasonably hypothesized, therefore, that a poisoner would show some of the same characteristics one would find in the profile of a bomber. Bombers are usually Caucasian, male, underachievers, nonathletic, cowardly, neat and orderly, meticulous, loners, careful planners, and nonconfrontational (either physically or psychologically); possess average to above average intelligence; and have inadequate personality (Meloy & McEllistrem, 1998).

Usually no trauma or visible signs of a poison’s effects are found on a victim’s body, because the body kills itself physiologically via a chemical short circuit. This allows the poisoner to choose what symptoms he or she wishes the victim to exhibit, based on the chemical and toxicological nature of the poison selected. The victim has no protection as long as he or she eats, drinks, and breathes. Of course, if the poisoner could get the victim to stop these essential natural body processes, there would be no need to administer the poison. However, we must all carry out the processes of eating, drinking, and breathing, so we are all essentially vulnerable to the attack of a poisoner.

A poison is a silent weapon. There is no noise like that of a gun being fired; therefore, a poison could be considered the ultimate silencer. Unlike for a shooting, in which the shot could be heard and reported by someone, it would be impossible to find a witness who could state that he or she heard the sound of a cyanide molecule going off in the dead of night. In addition, in an attack carried out with a gun, there is always the chance that some innocent bystander could become injured or killed. However, poison allows the careful-planning poisoner to carry out very precise targeting on a victim while leaving nearby bystanders unharmed and unaware that the crime has even been attempted or perpetrated. Thus, a highly skilled poisoner could murder a single individual at a large banquet without being detected.

Certainly a poison allows the poisoner to overcome easily a physically or mentally stronger person by invading that person’s defense zone. The poisoner therefore may be physically or psychologically nonconfrontational. For example, a petite wife could easily overcome the brute strength of an abusive spouse, and a weaker-willed husband could overcome the abusive personality of a more domineering spouse.

If the first attempt to poison is unsuccessful, this type of weapon provides a chance for the poisoner to try again, because the victim is still unaware that an initial attempt at murder has even been made. In addition, unlike with more traditional weapons, the poisoner is unlikely to be disarmed of the poison and have the weapon used against him or her. Poison is also very easily

overlooked at the death (crime) scene, because it is often appears like any natural substance that is usually found in the environment. With this type of weapon, it is quite possible to make a murder look like suicide, or a suicide look like murder. Furthermore, for the poisoner who might be squeamish at the site of blood or gore, or who is concerned about having to clean up the scene of the murder, there is no such messy component to the crime of poisoning.

With this type of murder, there is a certain degree of depersonalization: in the mind of the poisoner, he or she merely sets the trap; it is the victim who actually springs it. This rationalization allows the poisoner to lessen the guilt that he or she may feel about being the one who actively carries out the deed (see **Fig. 2-4**).

2.4. HOW DO POISONS KILL?

Like a wrench thrown into a finely tuned engine of a car would disrupt its proper running, the chemical molecule, like a “chemical monkey wrench,” disrupts the proper running of the body’s biochemical processes. Literally thousands of these “chemical monkey wrenches” are available to the poisoner. Each poison can be carefully selected to disrupt a specific body process. **Fig. 2-5** shows some of the major classifications of poisons based on how they will do their dirty work on various organ systems.

Poisons can kill a victim in a number of ways, depending on the effects of the chemical substance on the body’s normal physiology.

2.4.1. Central Nervous System Effects

By altering the critical function of the body’s nervous system, a poison can cause central nervous system depression, resulting in coma; loss of the respiratory drive, resulting in respiratory arrest (the stoppage of breathing); and loss of the reflexes protecting the airway, resulting in a flaccid tongue obstructing the airway, or aspiration of gastric contents into the bronchial tree. The poison can also affect the heart with cardiovascular (circulatory) effects, including hypotension (low blood pressure) from decreased cardiac contractions, hypovolemia from loss of fluids, peripheral vascular collapse, or cardiac arrhythmias.

The body’s cells can die from a lack of oxygen necessary for normal cellular respirations, called cellular hypoxia (low oxygen at the cellular level), owing to a breakdown in the normal transport of oxygen. There can be seizures resulting from muscle hyperactivity, which results in hyperpyrexia (increase in body temperature); or kidney failure, resulting from destruction of muscle tissue (deposition of myoglobin in the kidneys). There can be brain

Poison advantages over other weapons

- ☠ invisible “*stealth*” weapon
 - overlooked at the death scene
- ☠ no visible signs of violence: wounds, bruises, *etc.*
- ☠ a chemical “*booby-trap*”
 - it causes *trauma* at the cellular level
- ☠ no protection: we all must *eat, drink, & breathe* to live
- ☠ no noise - no one hears it – no witnesses
- ☠ no gore for the squeamish
- ☠ precise targeting – but takes planning
- ☠ if you miss, try again - who would know?
- ☠ creates a sense of depersonalization
 - poisoner sets the “*toxic trap*”, but the VICTIM triggers it!
 - gun = OFFENDER active & VICTIM passive
 - poison = OFFENDER passive & VICTIM active
- ☠ can’t be disarmed and have weapon used against you
- ☠ for the “*weak*”, it is much easier to overcome a “*stronger*” person

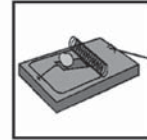


Figure 2-4

damage from lack of oxygen, which results in loss of the master control for the entire body engine. Death can be caused by pulmonary aspiration, which results in chemically induced pneumonia and destruction of the lungs.

Some poisons seem to pinpoint specifically a particular vital organ of the body. For example, the herbicide Paraquat destroys the lungs, the analgesic acetaminophen or the fungal amatoxins destroy the liver, and anti-freeze (ethylene glycol) or another fungal toxin called orellanine destroys the kidneys.

For some of the more common homicidal poisons, the investigating officer and analytical toxicologist can find poison profiles in the appendix to this

Some “Monkey Wrench” targets

- ☠ inflammation – irritant chemicals, acids
- ☠ enzyme inhibition – heavy metals (*As*, *Tl*), *HCN*
- ☠ receptor site interference (excite, or inhibit)
Atropine, Strychnine
- ☠ lethal synthesis – *Compound “1080”*
- ☠ necrosis – fungal *Amatoxins* (liver)
- ☠ neoplasia – carcinogens
- ☠ pharmacological effects – numerous compounds



Figure 2-5

volume. In each profile, the investigator will find discussions of the following important points about the poison:

- Form
- Common color
- Characteristic odors
- Solubility
- Taste
- Common sources
- Lethal dose
- Mechanism
- Possible methods of administration
- Time interval of onset of symptoms
- Symptoms resulting from acute exposure
- Symptoms resulting from chronic exposure
- Disease states mimicked by poisoning
- Notes relating to the victim
- Specimens from the victim to be obtained for analysis
- Analytical detection methods
- Known toxic levels
- Notes pertinent to analysis of the poison
- List of selected homicide cases in which particular poisons were used

What quantity of a poison would be necessary to produce a lethal outcome in a human victim? Many individuals are surprised to learn that a minute amount of a poison can result in death. To give the reader a hands-on feel for lethal amounts, let us use as a reference the weight of one US nickel (or two

The weight of coin examples
= 5.0 gm = 5,000 mg



POISON	Lethal Dose	# LDs in 5,000 mg
Thallium	1,000 mg	5
Compound "1080"	700 mg	7
Cyanide	200 mg	25
Arsenic	200 mg	25
Strychnine	100 mg	50
Nicotine	45 mg	111
Botulinus toxin	50 ng	100,000,000

Anthrax = 5.0 trillion spores = 500 million effective doses!

Figure 2-6

pennies). The average weight of these American coins is approx 5000 mg. If one had the same weight of some common homicidal poison, how many human lethal doses would this weight equal? See **Fig. 2-6** for examples of poisons and the number of lethal doses contained in this weight.

2.4.2. The "Molecular Firepower" of Poisons

Poisons as weapons contain a lot of "molecular firepower." When an individual aims a gun at another person and pulls the trigger, this in effect releases a single piece of lead that the offender hopes will produce a lethal effect on the intended target, by disrupting body tissue and vital organs, which will lead to the target's death. However, with a poison, one does not unleash a single bullet, but literally millions of "chemical bullets," to do their lethal business. How many chemical molecules are contained in a lethal dose? With an elementary knowledge of chemical principles, one can easily calculate the number of "killer molecules/atoms" contained in a lethal dose of any substance. This calculation is based on what is known as Avogadro's number, which states that there are $6.02214199 \times 10^{23}$ molecules, or atoms, per gram molecular weight of any substance. If one then takes the MLD of a poison, how many atoms or molecules are contained therein can be easily calculated (see **Fig. 2-7**). In scientific notation, 10^{18} is a number representing 1 billion billion. Thus, cyanide contains 4454 billion billion atoms in a lethal dose of



“Killer Molecules” in a Lethal Dose

POISON	Lethal Dose	Gram Molecular Weight	# Molecules
Arsenic	200 mg	74.92	$1,608 \times 10^{18}$
Botulinus toxin	50 ng	150,000	2×10^{11}
Compound “1080”	700 mg	100.03	$4,214 \times 10^{18}$
Cyanide	200 mg	27.04	$4,454 \times 10^{18}$
Nicotine	45 mg	162.23	167×10^{18}
Strychnine	100 mg	334.45	180×10^{18}
Thallium	1000 mg	204.37	$2,947 \times 10^{18}$

Figure 2-7

200 mg. This represents a great deal of chemical firepower for just a few cents’ worth of a substance. In fact, at a cost of \$14/lb (454 g) for cyanide, this amount of chemical represents 2270 human lethal doses, or 0.6 cents per lethal dose, or 1.7 lethal doses for a cost of 1 cent. In reality, then, a poison is much cheaper than a bullet. A 9-mm round costs approx 40 cents; for the same price, one can purchase almost 68 lethal doses of cyanide (*see Fig. 2-8*).

Over the last several years, a rather interesting number of murders have occurred in China with the use of a rodenticide called Dushuqiang, pronounced “doo-shoo-Chiang,” which is chemically known as tetramethylenedisulfotetramine. Having a human lethal dose of only 7–10 mg, in the weight of a nickel, this substance would contain about 740 lethal doses! The onset of symptoms—seizures and coma—after administration of this poison is 30 min to 3 h. There have been so many murders and tamperings in China with this material that the Chinese government has outlawed its production, sale, and possession. In 2003, the Chinese government seized 105 tons (92,281 kg) of this rodenticide, which amounted to a potential 13.6 billion lethal doses! Can we expect to see this rodenticide in the United States? In 2002, there was an accidental exposure in this country from this rodenticide, which had been illegally imported (“Poisoning,” 2003).

2.5. Elements of Poisoning Investigations

When examining poisoning cases, it is very helpful to look at the possible sources not only for the poison, but also the knowledge on how to use the poison.

Access to poison/knowledge:
Which comes first?

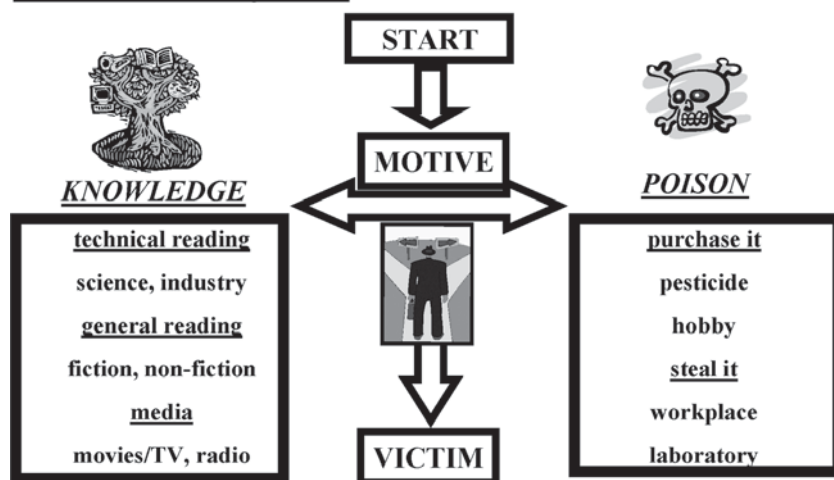


Figure 2-9

c. *The Poisoner's Handbook*, by Maxwell Hutchkinson, Loompanics Unlimited, Port Townsend, WA, 1988

On the Internet, any individual can easily find these manuals for sale, in which there is an enormous amount of information on poisons—their procurement, production, administration, and lethal dose—as well as how to avoid detection from crime committed using them. It is important to note, however, that protection of these types of reference manuals by the First Amendment to the US Constitution has now been condemned by the court decision *Rice vs. Paladin Press*, 87–1325. If criminal investigators find any of these materials during a search, their suspicions should be aroused immediately.

3. *Visual media*: Criminal poisonings are often examples of life imitating art. Investigators need to look for access to movies and television materials relating to poisons or crimes in which poisoning has played a part.
4. *The workplace*: Labels, manuals, material safety data sheets, or other materials dealing with chemicals should be investigated.
5. *Computers*: The amount of information that is available to computer users via the Internet or the World Wide Web is vast. Investigators should consider the possibility of a computer link for information on poisons, or crimes in which poisoning played a part.
6. *Word of mouth*: Although it is unusual for an offender to talk openly about poisons and their procurement, some cases have relied heavily on the testimony of individuals who have had conversations with the suspect on such matters.

2.5.3. Sources

Once the potential poisoner has knowledge about the poisonous weapon he or she has chosen, where does he or she go to obtain the material itself? Criminal investigators should consider some of these sources:

1. *Laboratories*: The suspect may have access to chemical substances found in laboratories in industrial, medical, or educational facilities.
2. *Hobbies*: The suspect may have hobbies that could provide poisonous substances, such as photography, jewelry manufacturing, or mineralogy.
3. *“Underground” catalogs*: Unbelievably, there are some individuals who collect poisons and poison bottles, like others collect guns or knives. There was at least one catalog available for such individuals, *JLF*, based in Indiana. This catalog of “poisonous nonconsumables,” which contained a long and detailed disclaimer, allowed one to purchase, e.g., toxic dried plants and fungi, and even stated in one issue that cobra venom would be “coming soon.” In 2002, the producer of this catalog was convicted of eight counts of violation of the Food, Drug, and Cosmetic Act and the Controlled Substances Act.
4. *Antique drug/chemical bottles*: Here is a source of poisons that is often overlooked by investigators. Surprisingly, one can purchase antique chemical bottles sometimes with their lethal contents still intact. These items can be obtained from flea markets, Internet auctions, or bottle collectors, and more than likely there is no paper trail of the purchase that leads to the offender.

2.5.3.1. SELLING ANTIQUE CHEMICALS CAN CAUSE MODERN TROUBLES

To reduce any potential for unwanted toxicological incidents, the sale and distribution of poisons, legend drugs, and hazardous materials is limited by law to licensed professionals. However, some of these items have found their way into the hands of individuals not licensed to possess or sell such materials. At antique shows and flea markets around the country, and over the Internet via auctions, dealers have been found selling antique drug and chemical bottles that still contain toxic contents, including arsenic, hemlock, mercuric chloride, phenobarbital (a controlled substance), sodium fluoride, and strychnine. How do these individuals come into possession of these drug and chemical containers that still hold hazardous material? When queried, most dealers have responded that their sources have been the stocks of old pharmacies and medical offices. They also have stated their belief that the container’s contents are now inert owing to the extreme age of the product, a belief that, for the most part, is clearly and dangerously erroneous. The compounds of arsenic, e.g., are as old as the earth itself and are as toxic today as when they were first formed billions of years ago. **Figs. 2-10** and **2-11** provide just a few examples of potentially lethal drugs and chemicals that were sold on an Internet auction site as antique poison bottles (contents included!).

Drug Poisons on the Internet

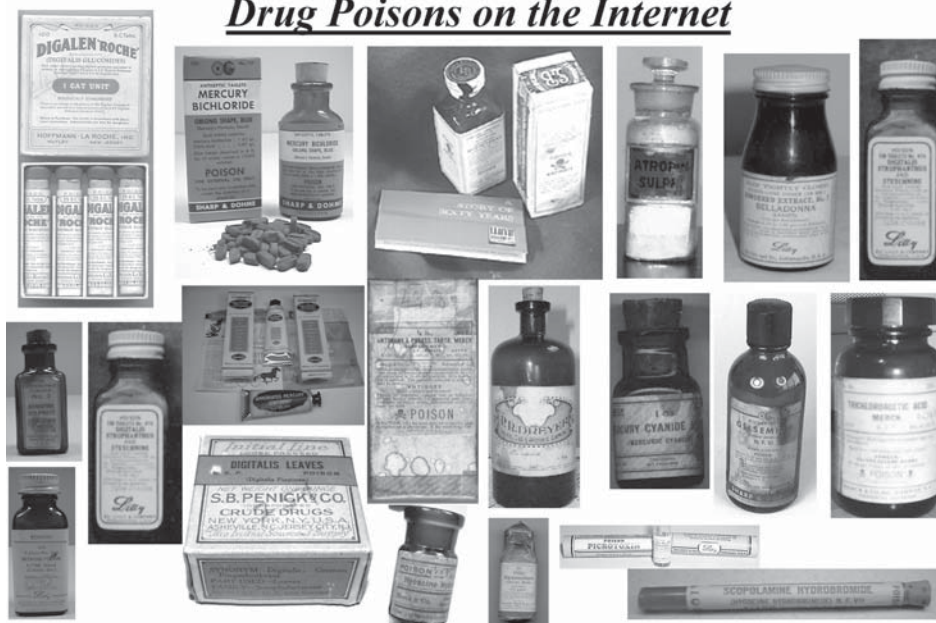


Figure 2-10

2.5.3.2. THE PROBLEMS THAT ANTIQUE POISONS PRESENT

A poison by any other name is still as dangerous, and the toxicological implications associated with the sale of antique hazardous chemicals focus on at least three major problem areas.

2.5.3.2.1. Antique Poisons in the Home

The presence of extremely hazardous antique poisonous substances in the home presents a clear and present danger for accidental and suicidal poisonings. If the collector of antique containers maintains a collection with their contents, then this presents an enormous risk in the home. One case in point involved the son of a pharmacist, who, while in a suicidal frame of mind, packed and swallowed capsules filled with sodium arsenite from his father's collection of antique bottles. The physiological impact of this heavy metal poison almost cost him his life, and the damage to his nervous system was such that even after months of hospitalization and rehabilitation the arsenic-induced peripheral neuropathies could not be totally reversed. The father will probably always regret that this poison was in his home, readily available to his emotionally distressed son.

Chemical Poisons on the Internet



Figure 2-11

2.5.3.2.2. Procurement of Poison With Homicidal or Tampering Intent

It is always of concern to society and to law enforcement personnel that the availability of poisons with a potential for homicidal or tampering use be carefully controlled. Thus, the sale of such substances must always be carefully documented in such a manner that proof of transfer of ownership is always maintained. By law, pharmacies are required to maintain a "poison register," to record the sale of any poisonous materials. These registers of sales include the purchase date, the name of the buyer, the buyer's address, the poison sold, the amount sold, and the intended use. Selling such poisonous substances without a paper trail could allow homicidal poisoners or tamperers to obtain chemical weapons with no traceable evidence.

2.5.3.2.3. Improper Disposal of Extremely Hazardous Substances

In accordance with Title III of the Superfund Amendments Reauthorization Act [P.L. 99-499], it is illegal for any individual or business to dispose of

“extremely hazardous substances” (EHS) unless done in a manner consistent with local, state, and federal guidelines. Most knowledgeable businesses will contract with a licensed toxic disposal firm to remove and properly dispose of their unwanted hazardous items. It is also important to note that even if the service of a disposal firm is used for hazardous substances, the original owner is still ultimately responsible for any environmental risk or contamination that may result from the materials. This responsibility cannot be transferred by sale or disposal.

It is important for pharmacists, physicians, and other health professionals to realize that selling their old chemical substances to an individual is not consistent with published guidelines, and that the health professional will ultimately be held legally liable for any toxicological problems that might arise. It is also illegal to dispose of these substances in domestic refuse, or to dump them in such a manner that could lead to contamination of soil or water environments.

2.5.3.3. THE SOLUTION

The solution to keeping toxic chemicals out of the hands of the lay public is quite simple. Antique bottles containing hazardous contents must never be sold. The hazardous contents must be carefully removed and disposed of in a manner that is consistent with local, state, and federal legal guidelines. Detailed records must be maintained that document proper disposal of any hazardous chemical substance. Health and law enforcement professionals must remain vigilant regarding the sale of these chemicals to unlicensed individuals and must clearly and emphatically state to the sellers the dangers of this practice. Any seller who refuses to withdraw these materials from sale should be immediately reported to the local office of the Food and Drug Administration, Consumer Products Safety Commission, and the Environmental Protection Agency.

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Chapter 3

Poisoners

“When you consider what chance women have to poison their husbands, it’s a wonder there isn’t more of it done.”—(Frank McKinney “Kin” Hubbard, http://en.thinkexist.com/quotations/when_you_consider_what_a_chance...)

As stated in the preface to this work, the poisoner has remained shrouded in mystery for centuries. Let us now examine what we know about the poisoner as an offender, and what we think we know about the poisoner.

3.1. TYPES OF POISONERS

One way to look at the motivation of a poisoner is to study how the victim is selected: some poisoners choose a specific individual, whereas others choose someone at random. The motives of these two types of poisoners are very different.

I have developed the following method of classification of poisoners based on victim specificity and degree of planning involved. There are two major groups: those who target a specific victim and those who choose a victim at random (*see Fig. 3-1*). Each group has two subgroups based on the speed with which the crime is planned and then carried out.

3.1.1. Type S: Specific Victim Is Targeted

Motives for the Type S group of poisoners include money, elimination, jealousy, revenge, and political ambition.

From: *Forensic Science and Medicine: Criminal Poisoning, Second Edition*
By: J. H. Trestrail, III © Humana Press Inc., Totowa, NJ

Poisoner types: by victim selection

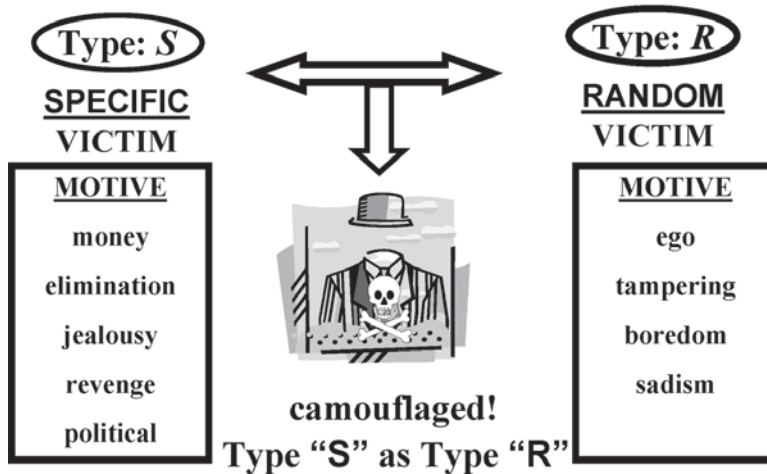


Figure 3-1

***3.1.1.1. SUBGROUP S: SLOWLY PLANNED POISONING
WITH A CAREFULLY SELECTED POISON***

An example of the Subgroup S type of poisoner would be a woman angry with her husband who goes to the library, reads about a particular poison, procures the chemical, and decides the best manner of administration to the victim. Type S/S = Specific/Slow.

3.1.1.2. SUBGROUP Q: A QUICKLY PLANNED POISONING

A crime committed by someone in Subgroup Q is done spontaneously with a poison selected as a weapon of opportunity. An example of this type of poisoner would be a woman angry with her husband who quickly takes a can of herbicide from storage and adds some to his food while preparing a meal. Type S/Q = Specific/Quick.

3.1.2. Type R: A Random Victim Is Targeted

Motives for the Type R group of poisoners include ego, the desire to tamper, boredom, and sadism.

***3.1.2.1. SUBGROUP S: A SLOWLY PLANNED POISONING
WITH A CAREFULLY SELECTED POISON***

An example of the Subgroup S type of poisoner would be a tamperer intent on industrial blackmail who adulterates a food/drug with a carefully

selected poisonous substance. This group would also include terrorists. Type R/S = Random/Slow.

3.1.2.2. SUBGROUP Q: A QUICKLY PLANNED POISONING

A crime committed by someone in the Subgroup Q is spontaneously done with a poison selected as a weapon of opportunity. An example of this type of poisoner would be an employee upset with his or her employer who quickly picks up a toxic substance and adulterates a batch of a consumer product (e.g., food, drug, cosmetic) to which the poisoner has access. Type R/Q = Random/Quick.

Although we may think we have been able to classify poisoners, we must remember to be aware of the “camouflaged” poisoner. In this situation, a poisoner gives the appearance of being a Type R but is in reality a Type S. An example of this type of poisoner would be an offender who poisons her spouse by tampering with his medication and then places similar tampered containers in a retail store to make the death of the victim appear to be random. There are various recorded cases of this type of poisoner at work. Among the more infamous cases, listed chronologically, are as follows:

- *Christiana Edmunds (Brighton, UK, 1871)*: Poisoned chocolates in a confectioner’s shop with strychnine, and one innocent child became a random victim. Her specific target was the wife of the man who thwarted her affection.
- *Ronald Clark O’Bryan (Pasadena, Texas, 1974)*: Poisoned Halloween candy with cyanide. His son, whom he killed to collect a life insurance premium, was the specific victim. He was also convicted of three attempts to murder other neighborhood children. Poisoning the other children had been part of his plan in order to make the crime appear to be the actions of a deranged tamperer.
- *Stella Maudine Nickell (Auburn, Washington, 1986)*: Poisoned Excedrin® capsules with cyanide to cover up the specific murder of her husband for insurance money. One random victim died.
- *Joseph Meling (Tumwater, Washington, 1991)*: Poisoned Sudafed 12-Hour® capsules with cyanide to cover up the specific murder attempt on his wife. One random victim died.
- *Paul Agutter (Athelstaneford, Scotland, 1994)*: Poisoned tonic water with atropine to cover up the specific murder attempt on his wife. Eight victims suffered from atropine intoxication as a result of his tampering, but there were no deaths in this case.

Investigators should remember, from these cases, always to question whether an apparent product tampering might actually be an attempt to cover up a specific homicide by throwing their investigation off the correct track.

3.2. MISCONCEPTIONS ABOUT POISONERS

It is important to correct some common myths about poisons and poisoners that exist in the minds of the general population. Among these myths is that most poisoners are female. In actuality, the majority of poisoners who have been detected have been male. The words “have been detected” are very important. This could lead one to speculate that females are more successful at escaping detection. Certainly they have a greater opportunity to invade the “security zone” of the victim, for it is typically the female who takes care of the sick, prepares the meals, and cleans the house.

Another common myth is that for every poison there is an emergency antidote. (It should also be emphasized here that the proper word for this type of drug is “antidote,” not “anecdote,” which is sometimes used erroneously.) In reality, there are currently only five drugs approved by the Food and Drug Administration that can make a life-or-death difference in the poisoning emergency. These antidotes are atropine (for organophosphate insecticides, nerve agents), Cyanide Antidote Package[®] (for cyanide intoxication), epinephrine (for allergic shock), naloxone (for opiates and narcotics), and oxygen (for carbon monoxide).

The last myth, and a question very commonly asked, is: Does a perfect undetectable poison exist? The answer to this question could be yes or no, depending on how one describes the word “undetectable.” The answer should be no if one considers that if there was such an undetectable substance how would one ever know it existed? If it has a name, someone must have detected it at least once to name it, and, therefore, anything with a name is theoretically detectable. However, the answer could be yes if one means by “undetectable” that the chance the poison would be routinely seen in a toxicological analysis is slim. If poisoning is suspected, and the analytical experts are given proper guidelines, almost every toxic chemical can be identified. However, if there are no guidelines to assist them, it is rather like looking for a molecular needle in a chemical haystack. If the substance is not one screened for in their normal substance panels, it will very likely go “undetected.” The problem is not the perfection of the poison, but the imperfection of the analytical process. Here it should also be emphasized that a “negative” toxicological screen does not mean that there is no poison in the specimen but, rather, that all the poisons routinely tested for in the analysis are absent. Other, more exotic poisonous compounds could remain undetected.

3.3. POISONER SCHEMATIC

For law enforcement to stand a better chance of solving poisoning homicides, it is important to understand why an individual would choose this type

of weapon over more traditional weapons such as a gun, knife, club, or rope. To understand the motive behind the choice to use poison, and to develop a “criminal investigative analysis” (sometimes referred to as a “psychological profile”) of this type of offender, the criminal investigation community must carry out a carefully planned study of convicted poisoners that looks for commonalities in their background and behavior. Such a study would be of immense value in guiding homicide investigators in their awesome task. Currently, I and my colleagues are in the early design stages of a plan to carry out this critical research on the psychopathy of the poisoner.

However, one can make some hypotheses about this type of individual by examining the personalities involved in the published cases of poisonings in which there have been convictions. There are commonalities among these types of poisoning offenders. Poisoners are for the most part cunning, avaricious, cowardly (physically or mentally nonconfrontational), childlike in their fantasy, and somewhat artistic (meaning that they can design the plan for the murder in as much detail as they would if they were writing the script for a play).

If one looks at the characteristics in the standard reference *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (American Psychological Association, 2000) in the section on personality disorders, clusters A, B, and C, one will find characteristics that seem to fit very well with our poisoner hypotheses. The poisoner’s personality especially correlates with the cluster B disorder known as narcissistic personality disorder, whose characteristics include the following a grandiose sense of self-importance (lies about achievements), a belief that the person is “special” and “unique,” a sense of entitlement (expects favorable priority treatment), a need to take advantage of others without regard to feelings, a lack of empathy (no acknowledgment of the needs of others), a feeling of envy toward others, an arrogant/haughty behavior or attitude, a preoccupation with fantasies of self-importance (fame, wealth, achievement), a requirement for excessive admiration, and an interpersonal exploitativeness (uses others to achieve his or her own end).

Why does the poison murderer select this weapon as the means of achieving his or her goal? One of the major reasons is that it provides a very good chance of getting away with the crime. Another reason is the fact that a poison allows completion of the assault without physical confrontation with the victim. The poisoner is truly an intelligent coward or, one could say, has the mind-set of an *enfant terrible* (incorrigible child) in the body of an adult. This is a very dangerous combination. If one also looks at many, if not most, of the male poisoners who have been tried and convicted, one will see that they tend to deal with conflict in a manner that is not physically confrontational. **Fig. 3-2** summarizes these characteristics.

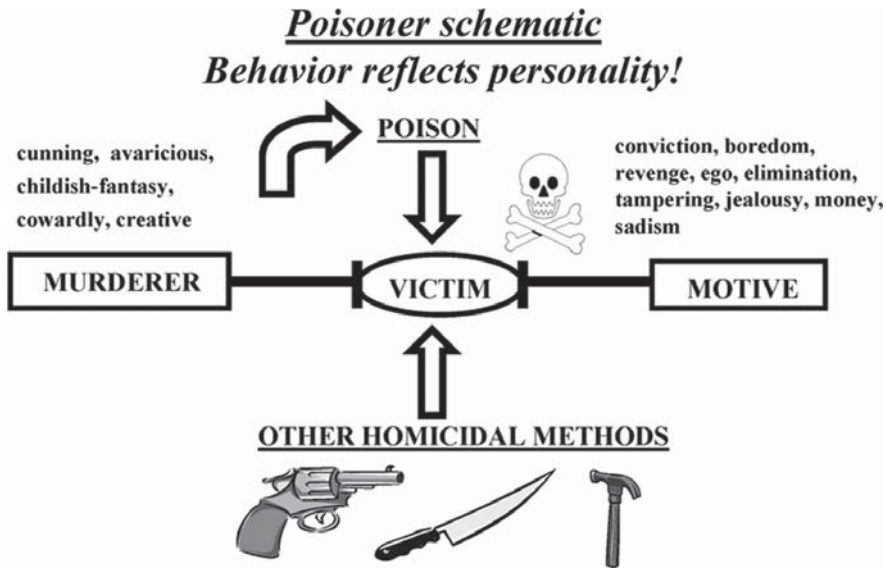


Figure 3-2

3.4. POISONER'S THOUGHT PROCESS

What goes through the poisoner's mind as he or she is planning his or her crime? Certainly all behavior is caused. The instigating force is the *motive*, or the force that moves the poisoner to the decision to eliminate that individual who stands between the poisoner and his or her goal. First, the poisoner says, "I want something!" and that is the *motive* to the crime. The poisoner's *intent* is to remove the obstacle that stands between the poisoner and his or her goal. Second, the poisoner realizes that he or she must devise a plan to access both the knowledge of the poison and the poison itself, which is the *means* to committing the crime. The poisoner also realizes that he or she must have knowledge of the victim's habits in order to have access to the target, or the *opportunity* to commit the crime. Finally, the poisoner believes that he or she can escape detection by ensuring that there are no witnesses to all the aspects of the crime, that he or she sufficiently distances himself or herself from the crime, and that no visible signs of the crime are left. Although the poisoner may be able to control most of these aspects of the crime, he or she cannot control the autopsy, analytical toxicology testing, and possible exhumation. **Fig. 3-3** summarizes these thought processes.

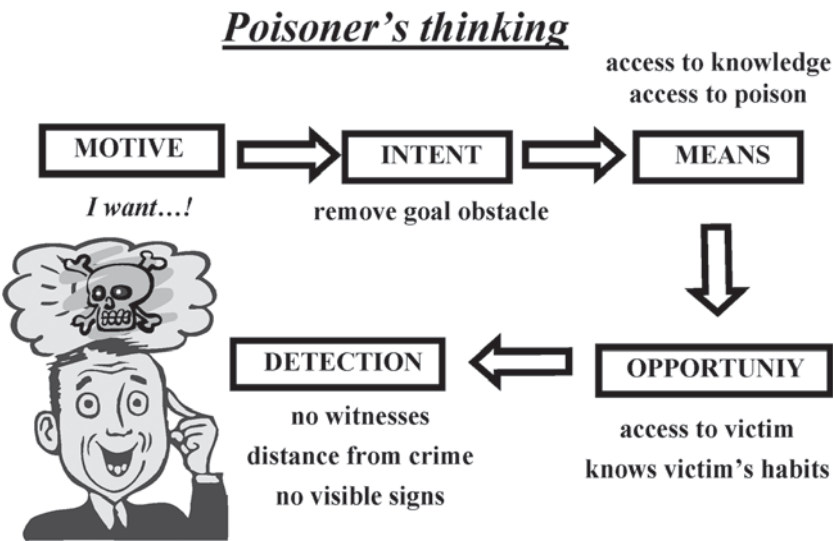


Figure 3-3

3.5. “*PSYCHOLOGICAL PROFILE*” OF POISONERS

In the personality of the poisoner, the investigator will probably find some of the following traits: an absolute defiance of legal authority; a refusal to accept any moral basis for life; a drive to kill in order to gain either emotionally or materially; an unfortunate married life for the offender; a childhood in which the poisoner was either spoiled by parents or reared in an unhappy home; a tendency to turn the victim into an object with no feelings; an abnormal life with wife, children, or home; a feeling that he or she has failed to make any kind of impression on life; a tendency to be a daydreamer and fantasist; a touch of an artistic temperament; possible connections with the medical world as a physician, nurse, pharmacist, dentist, other health care worker, or laboratory worker familiar with chemicals; possession of vanity in thinking that he or she cannot be discovered as being involved in the crime, because he or she carefully calculated the odds; a limited mind without sympathy; and a weak, cowardly, and avaricious temperament (Glaister, 1954; Rowland, 1960).

One sees in the childish personality of the poisoner an immature desire for one’s own way, and a dreamy, romantic disposition. Something seems to be hidden in the psyche of poisoners that keeps them permanently immature;

they never seem to grow up. They try to make the world obey their will by cheating it in minor ways, thereby stealing what it refuses to give them.

As far as poisoners' motives for murder, they are not much different from those of other homicides, in that they usually revolve around money (insurance); elimination of a goal blocker; jealousy (lover's triangle); revenge (make the person pay); sadism (make the person suffer); conviction (political motives, such as assassination or terrorism); boredom (wants to have fun by having a challenge of wits with law enforcement); and ego (belief in mental superiority).

3.6. *PUBLIC PERCEPTION OF POISONERS*

When one looks at how the general public views the poisoner, one can often see a sort of morbid fascination with this type of crime. In general, the public has a special hatred for the poisoner's sinister nature. The public generally believes that poison is a coward's weapon, because it is administered unemotionally and by stealth, often gradually over a long period of time. The poison is administered in full recognition of the victim's often prolonged suffering. The public reviles the poisoner for his lack of pity.

3.7. *THE TOXICOMANIAC*

A very rare mental condition is toxicomania. An individual who has toxicomania is obsessed with poisons, much like someone with pyromania is obsessed with fire. Toxicomaniacs relish the feeling of power provided to them by their weapon. A major example of this rather rare condition is the English case of Graham Frederick Young, discussed in Chapter 1, who from the age of 11 became obsessed with poisons, and eventually caused several deaths and many ill effects by experimenting on his relatives and coworkers with various poisons. He treated the victims not as humans, but as though they were rats in a toxicological study. The 1995 British movie *The Young Poisoner's Handbook*, a black comedy, was based on Young's life as a poisoner; it is unlikely, however, that most homicide investigators would find the film humorous.

3.8. *THE MEDICAL MURDERER*

Poisoners can also be found inside health care systems, although it is surprising that individuals who have sworn to their professional standards (e.g., the Hippocratic Oath) could coldheartedly kill their patients. Their murder is often covered by the fact that a sudden death in an already gravely ill or

elderly individual is not unlikely. And yet, there are numerous cases of physicians and nurses who have turned into serial killers for a number of different reasons. For some it was to create the medical crisis during which they could be seen as a hero trying to save the patient (e.g., Genene Jones, RN). For others it was to exercise “power” over life and death (e.g., Michael Swango, MD). And there are those who were motivated to kill for financial profit (e.g., Harold Shipman, MD).

3.9. *THE JUVENILE TAMPERER AS POISONER*

Of great concern is the number of juveniles who have been involved in recent tampering incidents. An analysis of the 1026 cases in my “Poisoner Database” revealed that between 1838 and 2005, there were 41 cases in which the offender was under the age of majority, with 27 (66%) of the cases having occurred since the year 2000. The intended victims of these incidents were as follows: 17 (42%) teachers, 5 (12%) classmates, 4 (10%) family members, 3 (7%) employees, 3 (7%) other, and 9 (22%) unidentified. Examples of these acts are adding a school cleaning substance to the teacher’s coffee or thermos, baking products made with chocolate laxatives, and adding mouse poison to a classmate’s lunch items.

Most of these incidents were probably intended as a prank, and owing to naiveté the offender most often used a substance that would not be of toxicological significance in a single acute dose (e.g., cleaning substances, or rodenticide containing the anticoagulant warfarin or the “superwarfarin” brodifacoum). However, such offenders do not realize the legal significance of their actions.

In October 13, 1983, the federal government passed the Federal Anti-Tampering Act [Public Law 98-127], which provides felony penalties for tampering with or threatening to tamper with any product covered by the Food, Drug, and Cosmetic Act. Offenders included in this legislation would be those who “with reckless disregard for the risk that another person will be placed in danger of death or bodily injury and under circumstances manifesting extreme indifference to such risk, tampers with any consumer product that affects interstate or foreign commerce, or the labeling of, or container for, any such product, or attempts to do so.” There are also state laws regarding the tampering of products, which will determine whether the action is to be regarded as a misdemeanor or felony. An example of such a state law might read like this:

Tampering with a consumer product in the second degree:

A person is guilty of tampering with a consumer product in the second degree when, having no right to do so nor any reasonable ground to believe that he

*has such right, and with intent to cause physical injury to another or with intent to instill in another a fear that he will cause such physical injury, he alters, adulterates, or otherwise contaminates a consumer product. Tampering with a consumer product in the **second degree** is a class A misdemeanor.*

Tampering with a consumer product in the first degree:

*A person is guilty of tampering with a consumer product in the first degree when, having no right to do so nor any reasonable ground to believe that he has such right, and with intent to cause physical injury to another or with intent to instill in another a fear that he will cause such physical injury, he alters, adulterates, or otherwise contaminates a consumer product and thereby creates a substantial risk of serious physical injury to one or more persons. Tampering with a consumer product in the **first degree** is a class E felony.*

A judge or jury would determine whether the crime would most appropriately fit into the degree of a misdemeanor or felony, based on the offender's "intent" and the "potential danger" to the victim represented by the poisonous exposure. Investigators and attorneys confronted with these types of cases of tampering should consult their state and local statutes to determine the most correct manner in which to proceed.

3.10. THE TERRORIST AS POISONER

Certainly since the tragic attack on 9/11, the world has become conscious of the role that terrorism has played in causing deaths worldwide. Terrorists' tools have mostly been explosives and guns, but poisons are also in their armory. There are numerous examples of radical groups who have been found to possess not only the knowledge of how to use poison, but also the actual poisons. What follows are just a few examples of these groups and the poisons that they used in their attacks:

- Order of the Rising Sun, Chicago, Illinois, 1972: typhoid.
- Red Army Faction, Paris, France, 1984: botulinus toxin
- Rajneesh Commune, The Dalles, Oregon, 1986: salmonella
- Minnesota Patriot's Council, 1992: ricin
- Aum Shinrikyo Cult, Tokyo, Japan, 1995: sarin nerve agent
- Anthrax Mail Tamperer, Washington, DC, 2001: anthrax

3.11. STATISTICAL ANALYSIS OF HOMICIDAL POISONINGS

In an attempt to shed some light on the poisoner, I have collected and analyzed 1026 documented known poisoning crimes in which the offender was convicted. This is a 51% increase from the 679 cases studied in the prior edition

Real Poisoners: poison used

n = 1,026 known cases

POISON USED	#	%
ARSENIC	265	26%
CYANIDE	83	8%
STRYCHNINE	63	6%
TOTAL	411	40%



Figure 3-4

of this work. What follows are the results of these analyses. As far as the geographic distribution of the cases analyzed, most were from the United States (404 [39%]) and the United Kingdom (255 [25%]), but numerous cases were from many of the world’s countries.

3.11.1. Most Common Poison Used

As can be seen in **Fig. 3-4**, the most commonly used poisons in order have been the “Big Three,” consisting of 265 (26%) cases of arsenic, 83 (8%) cases of cyanide, and 63 (6%) cases of strychnine. These three poisons were involved in 411 (40%) cases of the 1026 poisoning cases analyzed.

3.11.2. Poisoner’s Background

The analysis indicated that in 709 (69%) cases the vast majority of the offenders came from what could be called the general public, i.e., private citizens. **Fig. 3-5** presents the results.

3.11.3. Poisoner’s Gender

The majority of the known offenders were found to be male in 466 (45%) cases; the 400 females represented 39% of the cases. This analysis must be looked at with the caveats that in 16% (160) of the cases the gender of the offender was unknown, and that these cases represent only incidents that were detected. It could be, once again, that females were more successful at remaining undetected in the crime (*see Fig. 3-6*).

3.11.4. Number of Victims

In 420 (41%) of the cases, there were multiple victims, and these cases were divided into separate incidents. This indicates that in almost half of these

Real Poisoners: background*n = 1,026 known cases*

BACKGROUND	#	%
general public	709	69%
physician	59	6%
nurse	42	4%
juvenile	38	4%
political	32	3%
TOTAL	880	86%



Figure 3-5

Real Poisoners: gender*n = 1,026 known cases*

GENDER	#	%
male	466	45%
female	400	39%
?	160	16%
TOTAL	1,026	100%



Figure 3-6

poisoning crimes, the poisoning offender was a “serial poisoner.” To quote Schonberg’s Law, “Anybody who gets away with something will come back to get away with a little more” (Schonberg, 1972). Certainly no other means of homicide produces this percentage of offenders with multiple victims. If there are no consequences to a behavior, it becomes chronic, and whereas most serial killers attack strangers, most serial poisoners attack those they know (see **Fig. 3-7**).

3.11.5. Poisoner’s Motive

Examination of the recorded motives for the crimes revealed that they usually involved love or money. Using the crime classifications in the *Crime Classification Manual* by Douglas and Burgess (1992), it was found that 219

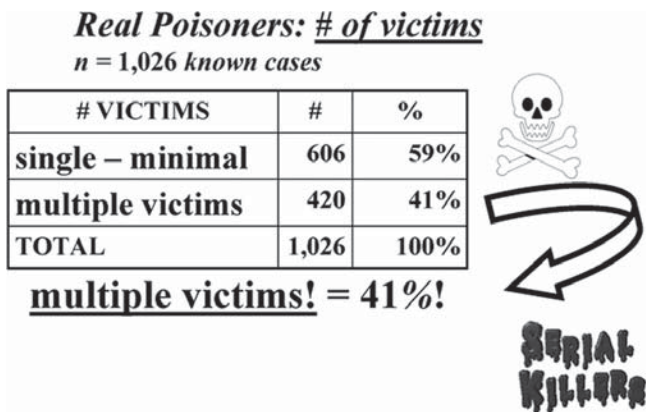


Figure 3-7

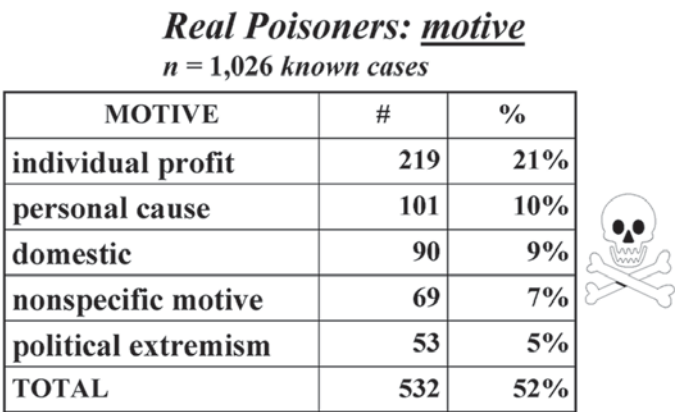


Figure 3-8

(21%) cases were motivated by individual profit, 101 (10%) cases by a personal cause, and 90 (9%) cases by a domestic reason (see Fig. 3-8).

3.11.6. Offender's Trial Outcome

In examining an enhanced set of 1074 cases of known poisonings, in 1026 (95%) of the cases, the suspect was convicted of the crime. Many of the remaining 48 (5%) cases were dismissed under a cloud of suspicion, but prosecutors were unable to prove the cases beyond a reasonable doubt (see Fig. 3-9).

Real Poisoners: Trial Outcome

n = 1,074 known cases

OUTCOME	#	%
guilty	1,026	95%
acquitted	48	5%
TOTAL	1,074	100%



Figure 3-9

Real Poisoners: gender of Multiple Offenders vs. # of Victims

n = 120 = (12% of 1,026)

GENDER Multiple Offender	% Victims	# Single victims	# Multiple victims	TOTAL Victims
male	46%	32	23	55
female	52%	34	28	62
? gender	2%	0	3	3
TOTAL	100%	66	54	120



Figure 3-10

3.11.7. Multiple Offenders on Victim(s)

In 906 (89%) of the cases, only a single offender was involved in the crime. Multiple offender cases, although rare, are usually easier to convict, because there is the possibility of the two or more participants providing evidence against one another (*see Fig. 3-10*). In the 120 cases involving multiple offenders, 55 (46%) were male, and 62 (52%) were female, with 3 (2%) of unknown gender.

3.11.8. Gender of Offender vs. Number of Victims

It has been speculated by some that if the female is less easily detected in her crime, she would have a greater opportunity to carry out her deeds on

Real Poisoners: gender
of Offender vs. multiple Victims

n = 420 known cases (41% of 1,026)

GENDER	#	%
male	191	46%
female	182	43%
?	47	11%
TOTAL	420	100%



Figure 3-11

multiple victims over a longer period of time. In an analysis of the subset of 420 known cases that involved multiple victims, for female offenders, 43% had multiple victims, yet for males it was 46%. This indicates that there is a slightly greater chance of males having multiple victims, but it might not be a significant difference between the genders (*see* **Fig. 3-11**).

As can be seen from this chapter’s discussion of the poisoner, much more needs to be determined about this type of criminal offender. Remember, one can be a famous poisoner or a successful poisoner, but not both! To be able to lift the veil of secrecy that surrounds poisoners, it will take a concentrated and coordinated effort on an international scale to examine commonalities and possible cultural differences in the use of poison as a weapon for murder.

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Chapter 4

Victims

“Most signs and symptoms associated with natural disease can be produced by some poison, and practically every sign and symptom observed in poisoning can be mimicked by those associated with natural diseases.”

—L. Adelson

Most often the victim of poisoning will appear rather natural in death. In effect, poisoning is murder in slow motion, because it may take a long period of time, depending on the dose and the poison that has been selected as the weapon. Two major factors that determine lethality of a substance are (1) concentration and (2) duration of exposure.

4.1. WHO GETS POISONED?

Poisoning murders can be classified into a number of groupings, depending on the motive for homicide. There can be a suicidally motivated parent who wishes to take the children with him or her. A good example of this type of killing is the case of Johanna Maria Magdalena (“Magda”) Goebbels and her husband, Joseph (Third Reich propaganda minister), who, in 1945, used cyanide to murder their six children in Hitler’s Berlin bunker, as the Allied forces approached and the end of the Third Reich was near. Another example is the case of the parents at “Jonestown,” Guyana who participated in the mass suicide there.

Another type of poisoning death is an unintentional homicidal poisoning (manslaughter). This type of poisoning might result from an accidental drug overdose, as in the death of comedian John Belushi. Another interesting incident is the 1954 British case of Arthur Ford, who in his wish to sexually arouse two of his female office coworkers accidentally killed them with his use of cantharides (Spanish Fly).

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A death may also result from administration of a harmful substance to a child in order to stop what is deemed improper behavior (e.g., bed wetting, nail biting, or not following parental instructions). Several manslaughter deaths from the administration of powdered black pepper (*Piper nigrum*) have been documented, and in one case this administration resulted in the aspiration death of a child (Cohle, Trestrail, Crahan, et al, 1988).

One of the more discussed abnormal psychological conditions is what has become known as *Munchausen Syndrome by Proxy*. This condition was named after Baron von Munchausen, a famous German teller of fabulously unbelievable tales. This syndrome is a phenomenon in which a mentally ill parent administers poison to his or her child in order to draw attention to himself or herself. Such a parent enjoys being the center of attention, and by using a child as the object of a medical emergency, the parent in effect becomes the center of attention by proxy. The offender gets some personal psychological reward from having doctors listen and begins to exaggerate the symptoms. This type of poisoner is usually a mother, who may show many of the following characteristics (Levin & Sheridan, 1995):

- Comes from a background where she was ignored and unrecognized
- Has a history of being abused herself
- May consider the relationship she had with her obstetrician the most intense, personal, and rewarding she had ever had and is attempting to transfer this role to the child's pediatrician
- May have some nursing training
- May have a history of weaving false tales of medical problems.

This type of poisoning event is considered a form of child abuse. In most cases, the father is usually oblivious to the activities or may be in sub-conscious collusion with the perpetrator.

The timely issue of euthanasia of the elderly and terminally ill by homicidal poisoning, in private homes as well as in nursing homes, came to national attention in the last decade. Dr. Jack Kevorkian's fight for doctor-assisted suicide brought this type of poisoning to the forefront of nightly news around the country. Its popularity is also evidenced by the publication of the euthanasia guidebook *Final Exit*, and the existence of the "Hemlock Society," which provides instructions for committing suicide to those wishing to end their own lives because of terminal illness.

The emphasis of *Criminal Poisoning* concentrates mainly on intentional homicidal poisoning, which is always considered first-degree murder because of the element of premeditation.

4.2. *INVESTIGATIVE CONSIDERATIONS*

What indices might the homicide investigator utilize to help him or her determine that a poisoning murder may have occurred? Investigators should look for friends or relatives who arouse suspicion, as well as suspicious circumstances that surround the death. Indicators might include the sudden death of the victim after eating, drinking, or going into the bathroom; or poison containers found near the deceased. In a suicide, the latter might well be the case, although it could be a murder masquerading as a suicide.

The antemortem (before death) clinical course might indicate that the deceased exhibited symptoms consistent with a poisoning, but it is easy for an investigator to be misled. One would think that an autopsy of the body would clearly reveal that the cause of death was other than natural, yet in many localities autopsies are not performed, owing to either the cost or availability of such a service, and the coroner or medical examiner normally lists on the death certificate his or her best judgment as to the cause of death.

4.3. *DISTINCTIVE PATHOLOGICAL FINDINGS*

Following are pertinent questions that the medical examiner should ask:

- Does the victim exhibit no morphological changes that can be attributed to direct chemical action by a toxic agent? Poisonous substances that might be considered in such cases include the following: acute central nervous system depressants (alcohols, ethers, sedatives, chloroform, hypnotics, and so forth), chemical asphyxiant gases (carbon monoxide, hydrogen cyanide), organophosphate insecticides (OPIs) (malathion or parathion), and alkaloidal compounds (strychnine, opiates).
- Are systemic lesions present without obvious injury at the site of entry? Poisonous substances that might be considered include arsine and nitrobenzene.
- Is an injury present at the site of entry that does not exhibit remote or systemic evidence of direct cell damage? Poisonous substances that might be considered include those that cause immediate cellular necrosis (corrosives) or gaseous irritants (chlorine, sulfur dioxide).
- Are local and systemic injuries present? Poisonous substances that might be considered include heavy metals such as mercuric chloride, arsenic, antimony, and lead.

4.4. *CLASSIC SYMPTOMS OF POISONING*

Some visible clues that should alert criminal investigators and health-care workers that a victim may have been poisoned are as follows:

- *Hair loss (alopecia)*: Often found as a result of chronic intoxication from the heavy metals (e.g., arsenic, antimony, and thallium). It is surprising how often this clue is overlooked.
- *Fever (hyperthermia)*: Results from activation of the body's defense systems.
- *Constricted pupils (miosis)*: Often found as a result of opiate compounds (e.g., morphine, codeine, heroin), OPIs, and so forth.
- *Dilated pupils (mydriasis)*: Can be found as a result of the solanaceous plant alkaloids, such as atropine, scopolamine, and hyoscyamine; as well as insulin; cocaine; nicotine; and so forth.
- *Odor*: Some poisons have characteristic odors that may be detectable on the victim. For example, arsenic smells like garlic, Vacor rodenticide smells like peanuts, and nitrobenzene smells like shoe polish.
- *Oral burns*: The mouth area or face may exhibit burns caused by caustics and corrosives compounds (acids and alkalis, such as sodium hydroxide [lye]).
- *Gastrointestinal effects (diarrhea)*: May be caused by many poisons, especially the heavy metals.
- *Change in skin color*: Cherry red skin color results from carbon monoxide and blue (cyanosis) from nitrites (methemoglobinemia).
- *Vomiting (emesis)*: Results from stomach irritation (arsenic, antimony, aconite, acids, alkalis, colchicine, cantharides, phosphorus, mercury, iodine, and so forth).
- *Injection tracks*: Can come from multiple administrations of poisons by the parenteral (injected) route.
- *Skin speckling*: This looks like raindrops hitting the surface of a dusty road. It can be caused by chronic doses of arsenic.
- *Stomach cramps*: A classic sign of chronic poisoning.
- *Brittle nails and Aldrich-Mees lines (white transverse lines across the nail beds)*: Heavy metals can change the nail structure. These lines should not be confused with leukonychia, which are white spots resulting from traumatic injury to the nail, or the lunula, which is the normal pale area at the nail root.
- *Convulsions*: Caused by strychnine, organophosphate compounds, camphor, cyanide, and so forth.
- *Coma*: Caused by opiates, hypnotics, sedatives, carbon monoxide, carbon dioxide, ethanol, phenols, and so forth.
- *Paralysis (general or partial)*: Results from alterations in the nervous system caused by botulism, cyanide, thallium, arsenic, and so forth.
- *Abrupt onset of symptoms*: Sudden appearance of symptoms in a previously healthy individual.

Table 4-1 summarizes these symptoms and the poisons that cause them.

With the correct determination that the victim has been the target of a poisoner's efforts, the focus turns to the crime scene. The role of the investigator is then to gather evidence that will eventually lead to the source of the poison, and the poisoner himself or herself.

Table 4-1
Common Poisons and Their Symptoms

Symptom	Poison
Hair loss	Thallium, other heavy metals
Fever	Multiple poisons
Constricted pupils (miosis)	Opiates, organophosphate pesticides
Dilated pupils (mydriasis)	Alkaloids, insulin
Garlic odor	Arsenic, antimony, etc.
Peanut odor	Vacor (rodenticide)
Bitter almonds odor	Cyanide
Shoe polish odor	Nitrobenzene
Oral burns	Corrosives (lye, acids)
Diarrhea	Heavy metals
Cherry red skin color	Carbon monoxide
Blue skin color (cyanosis)	Nitrites
Vomiting	Multiple irritant poisons
Injection tracks	Insulin, drugs of abuse
Skin speckling	Arsenic (chronic)
Stomach cramps	Multiple poisons
Nail changes (Aldrich-Mees lines, brittleness)	Heavy metals (arsenic)
Convulsions	Strychnine, cocaine, pesticides
Coma	Depressants, hypnotics
Paralysis	Botulism, heavy metals
Abrupt onset of symptoms	Multiple poisons

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Chapter 5

Crime Scene Investigation

“Murder is first conceived in the heart. But if it remains there—as it often does—it is no crime, though it may well be a sin. It is the acceptance of the idea of murder as a possible means of getting what one wants that is the decisive step. For most normal and sane people the idea is still-born. They smile to themselves and say ‘What am I thinking of?’ The thought passes, and is lost in the limbo of forgotten fantasy. But if the thought is not forgotten? If it recurs? If it is first half-accepted and then embraced? From this point we have a woman who intends to murder and we pass to the field of strategy and tactics.”—Gerald Sparrow

Poisoners are very concerned about the detection of their crime, and although they can control most incriminating evidence of their crime, they cannot control autopsies, analytical toxicology, and exhumations (see **Fig. 5-1**).

The prime directive for gathering evidence of poisoning at a death scene is to remember the proper chain of custody. Nothing can break a case assumed to be solid more easily than the defense being able to prove a reasonable possibility that evidence could have been tampered with before the trial.

The object that initiated the investigation was the victim, so it is with the victim that one begins searching for answers to important questions. What has become known as “victimology” is a study of the victim that it is hoped will reveal clues that can answer questions of *why* and *who*. Why was the person possibly a target? By answering this question, one begins to determine whether there was any individual who would have anything to gain either physically or mentally from the victim’s death. One needs to get a complete history of the victim, including a detailed financial background. Remember, *the victim himself or herself is the most important crime scene*. Interviews are

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Avoiding detection

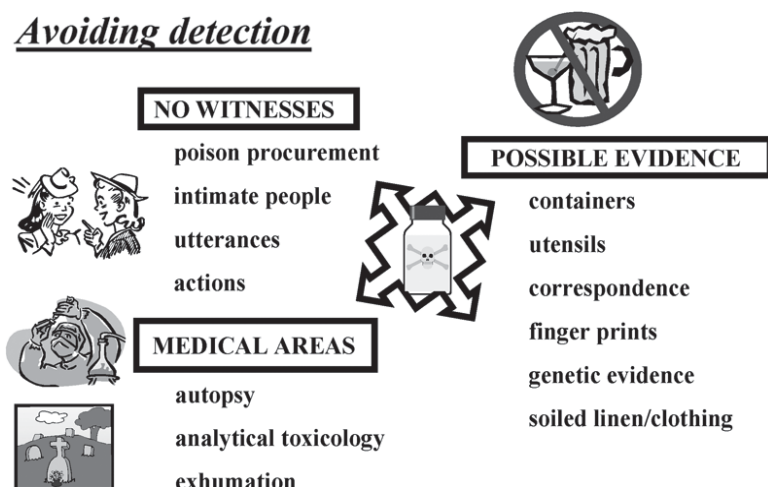


Figure 5-1

critical, for except for suicides, few poisoned victims die in isolation—they are surrounded by other people!

Regarding the crime scene, with poisoning multiple locations may have come into play during the planning and execution of the murder. Each location can yield important clues that must be included in the complete case investigation. Some of the locations and the items to look for to yield clues are as follows:

- Where the victim was *found* (vomited material, clothes containing poison residue).
- Where the poison was *administered* (medicine bottles, food/beverage containers).
- Where the poison was *disposed* of (storage areas, trash containers, sink traps, vacuum cleaner bags).
- Where the poison was *prepared* (tools with poison residues, utensils, clothes, containers).
- Where the poison was *procured* (stolen items, receipts of purchase, signature on a poison register, computer files).

The criminal investigator needs to look closely at the environment of the crime scene regarding place and time. Does the crime scene show that it might have been manipulated? In other words, is it too clean?

How many offenders might have been involved in this crime? With poisonings, the vast majority will involve a single offender on a single victim. This type of crime usually is not a group activity, although a few cases have

been documented in which multiple offenders have poisoned a victim or victims.

What matters is not the perfection of the poisoning crime, but the degree of imperfection of the detection process! The poisoner does not have to worry about disposal of the victim's body. The poisoner counts on the medical system doing it for him or her.

5.1. PHYSICAL EVIDENCE

The analysis of whether a crime scene appears organized or disorganized can often yield valuable information about the mind-set of the killer. The poisoner usually will exhibit some characteristics of both the organized and disorganized personality. The organized poisoner has a planned offense and usually leaves no weapon or evidence at the crime scene. By contrast, the disorganized poisoner leaves the evidence in plain view at the death scene.

The criminal investigator needs to look carefully at the death scene to answer the following important questions: Does the body disposition show the possibility of an unnatural death? Are there items intentionally left or strangely missing? Are there any unusual odors? Investigators must remember to be aware of the masking effect of tobacco smoke on unusual odors associated with some poisons (e.g., cyanide, solvents, and fumes) and not allow smoking at the crime scene. Finally, is there evidence of "staging" (purposeful alteration of the crime scene)? It must be kept in mind that with the poisoner the scene will be left mostly in a natural state, except that the vehicle for administering the poison may have been removed or cleaned. As the famous French forensic scientist Dr. Edmond Locard (1877-1966) taught, "Every contact leaves a trace." This is now known as Locard's Exchange Principle.

5.2. INVESTIGATIVE CONSIDERATIONS

If there was a first-aid responder, that person should be interviewed as to what the victim looked like before therapy was attempted. Was the victim cleaned up? If so, where are the materials that were used in the cleaning? They may contain valuable toxicological evidence. In addition, investigators need to keep the following in mind. First, they need to remember that what seems obvious may be totally wrong. Second, there should never be an unconscious selectivity of evidence; they must examine the evidence as a whole, and not only those parts that support their belief. Third, they should never attempt to manipulate evidence, or its interpretations, in an attempt to please law enforcement or attorneys.

5.3. *SEARCH WARRANTS*

If the poisoner does not think he or she will be suspected, the area of preparation may not be cleaned up, for it is the “den of security.” Why hide something one does not think an investigator will ever look for? It is important for investigators to stay within the “four corners” of the search warrant, meaning the corners of the pages of the warrant. It defines the only parameters of the allowed search. On the search warrant, the exact location and items that need to be investigated must be specified. Of course, to obtain a search warrant, there must be “probable cause,” as defined under the Fourth Amendment to the US Constitution. “Probably cause” is when known facts and circumstances, of a reasonably trustworthy nature, are sufficient to justify a person of reasonable caution or prudence in the belief that a crime has been or is being committed. Above all, it must be remembered that a poison can be found anywhere, so one must search areas such as wastebaskets, sink traps, garbage cans, and work locations. The poison most likely will be in small quantities (<200 g), not in a 50-lb barrel in plain sight.

There are related items to search for, and the criminal investigator should consider all of the following: publications on poisons, receipts for procurement of chemicals, medical publications, poison recipe cards, chemical catalogs, diaries/journals, and computer files and records.

Criminal investigators should also do the following when executing a search:

- Read over the search warrant and affidavit prior to executing it and become familiar with all the details of the proposed search.
- Remember to leave a copy of the search warrant at each location that has been searched. Leave a receipt, and note the date and time of the execution and securing of evidence.
- Maintain a complete and detailed inventory of seized properties.
- Do not consume any edible materials at the site, because they may be tainted, or one may be destroying important evidence.

5.4. *POISONING DEATHS COMPARED WITH OTHER TYPES OF VIOLENT DEATHS*

As a crime, poisoning exhibits some nuances usually different from the more violent and traumatic homicides. The offender is usually secretive, quiet, and covert and will not confide in others about the intention or execution of the murder. The victim will usually exhibit no external signs of violence. The crime almost always involves careful planning and cool deliberation (the murderer engineers the well-conceived opportunity). The poisoner is most

thoughtfully and seriously concerned with preventing discovery of the crime, and he or she is probably the most cunning of murderers. The offender usually possesses a high degree of skill as well as knowledge of the victim's routine and personal habits and usually acts alone; thus, there will be an absence of witnesses. The offender kills because he or she truly believes that he or she can get away with the crime. A poisoning murder is usually an intimate or "household" crime, with the principals usually united by close emotional ties (the marital bond is the most common).

Additional contrasts are that the victim of a poisoning is defenseless, with no protection, and, therefore, it is not likely that there will be any defensive kind of evidence. The offender has a high degree of knowledge of the lethal potentialities of the poison used, because he or she has researched the poison.

It has been stated that poisoning is the least used method of homicide, accounting for only 3–6% of known homicide cases (Adelson, 1974). Because of the complexity of poisoning homicide, it is one of the most difficult homicides to prove. Homicidal poisoning is almost always murder, never manslaughter (unless the offender is judged to be legally insane), because it involves premeditation, deliberation, and intent to kill.

5.5. INVESTIGATING A CRIMINAL POISONING

To better the chances of detecting a murder by poison, all death investigations should be handled as homicide cases until the facts prove otherwise.

To begin an investigation, it is necessary to gather some facts that may help determine whether the death might have been owing to a poisoning incident. If the victim is still alive, the investigator should ask questions that deal with symptoms, how he or she feels, and if the onset of these symptoms coincided with a common event. If the victim is dead, these same questions should be asked of any other individuals who had contact with the deceased. (The investigator should always remember that he or she may be interviewing the actual poisoner!) **Table 5-1** summarizes essential pieces of information that need to be gathered (Department of the Army, 1967).

If the criminal investigator determines that a poison is likely to have been involved in the death, then he or she must determine the possible source of the poisonous weapon. It is imperative to look for a paper trail that might show taking possession. If the poison was procured at a pharmacy, the investigator should ask to see the pharmacy's "poison register," which should indicate the date, purchaser's name and address, name of the substance, amount purchased, and intended use. Was there a commercial source, such as a chemical supply company, that might have purchasing records? Is there a possible

Table 5-1
Information to Obtain During a Poisoning Investigation

What was the location of the victim when symptoms first appeared?
What were the symptoms?
Did someone give the poison intentionally? (Who might have done it, and what was the motive?)
Did the victim administer the poison himself or herself (accidental or intentional)? If so, why?
Who called for help? (When and how?)
What did the victim do just prior to the appearance of symptoms?
What did the victim eat or drink prior to the appearance of symptoms?
Did the victim abuse any drugs or controlled substances (which could have been adulterated)?
Was food or medication requested or offered? (Who prepared it, and who served it?)
Did any other person consume the same items, and if so, how are they feeling?
Was the victim in the habit of consuming the substance in question?
Was the victim in the habit of consuming any type of alcohol not intended for drinking?
Did the victim eat or drink anything after the symptoms first appeared?
Did the victim take any medicine before the appearance of symptoms?
What was the victim's general health condition?
Was the victim recently unhappy, depressed, jealous, or angry?
Did the victim have money on his or her person prior to the appearance of symptoms?
What was the condition of the victim's estate, and did he or she owe large sums of money?
Who would inherit the victim's estate, and do they have immediate financial needs?
Did the victim have any recent difficulties with his or her employment?
Was anyone jealous of the victim because of his or her position? (Who might be promoted?)
Did the victim receive any threatening letters or other communications?
Did the victim receive any unsolicited gifts by mail for birthday or special holidays?
Was an unsolicited sample of a new product received in the mail?

Adapted from *Crimes Involving Poison*, Department of the Army Technical Bulletin TB PMG 21, Department of the Army, Washington, DC, 1967, pp. 11–13.

computer trail that shows purchase of the material over the Internet? Did the offender have any connection with an industrial or educational institution that would have allowed him or her to steal the material?

5.6. *PHYSICAL EVIDENCE*

The criminal investigator should take into evidence any and all of the following types of materials from the crime scene: remains of food and drink, drugs, medicines, chemicals, glasses, bottles, spoons, syringes, and soiled linen or clothing.

5.6.1. Product Tampering

There is always the possibility that the death was owing to a substance that had been tampered with (e.g., food, drug, cosmetic). According to the Federal Anti-Tampering Act, it is a felony to tamper with foods, drugs, devices, cosmetics, and other consumer products. There are certain governmental agencies that now or in the past have been involved in the investigation of product tampering. For example, prior to 1989, the Food and Drug Administration (FDA) maintained the Elemental Analysis Research Center, in Cincinnati, Ohio, and, in 1989, the FDA began the Forensic Chemistry Center, also located in Cincinnati.

When considering a product tampering, investigators must ask themselves these questions: Could tampering of the product have occurred during the manufacturing process, at the hands of an employee, or as a form of industrial sabotage? Could it have occurred during distribution of the product and then the product been returned to the shelf looking untouched (remember the “poisoner camouflage,” in which the offender has a specific victim in mind and tries to make the crime look like a random death). Could it have occurred after the point of purchase (which is when an individual seeking a financial settlement from the manufacturer makes a false report)?

5.6.2. Analytical Toxicology

We now come to the part of the investigation in which a specialized form of chemical qualitative and quantitative analysis takes place. This is usually done in a forensic toxicology laboratory. The criminal investigator needs to remember that these analytical tests do not routinely test for all chemical substances. Forensic toxicology laboratories normally have a set of specialized toxicology screens that they utilize. These general tests usually are drug screens, which look for commonly abused substances; heavy-metal screens, which look for substances such as arsenic, antimony, thallium, or lead; and volatile substance screens, which look for solvents such as chloroform or ether. There may also be general analytical screens, which detect cyanide, volatiles, strychnine, heavy metals, and drugs.

Remember that when a result comes back negative, it means only that none of the substances tested for were present in detectable quantities, not that the specimen was free of all chemical substances. It would be nice if someday, like *Star Trek*'s Dr. McCoy, we could pass a medical “Tricorder” over the body in question and thereby scan for more than a million different chemical entities. Unfortunately, such technology is too far in the future to be of help to us at present.

However, it is quite possible for the criminal investigator and pathologist to be of great assistance to the team running the toxicological analyses, by providing an indication of what substances are suspected. This help comes from the death scene investigation and any abnormalities found on autopsy. Remember, one cannot find what one is not looking for.

Typically the analytical toxicology laboratory will use one or more of the following methods in its qualitative and quantitative analyses:

- *Color tests*: Cheap, easy, and quick.
- *Immunoassays (radioimmunoassay [RIA])*: Utilizing antibody reactions.
- *Thin-layer chromatography, or TLC*: This method is based on the separation of substances based on their movement through a matrix by a defined solvent system. The unknowns are then compared with a known standard based on what is known as their Rf values.
- *Gas chromatography, or GC*.
- *Ultraviolet spectroscopy, or UV*.
- *Mass spectrometry, or MS*: A procedure akin to “fingerprinting” molecules.
- *Gas chromatography/mass spectrometry, or GC/MS*: Currently the most powerful method for confirmation of substance identification.

It is also important to remember that the analytical work can only indicate the presence and possible quantity of a poisonous material, not the reason for the exposure. It is up to the death investigator to determine the reason for the exposure. One observation that might be helpful is that there can be differences in the quantitative amount of the lethal material. In a homicide, usually just the right amount for a lethal dose is given, whereas in a suicide, usually a massive amount is taken.

To carry out a proper toxicological analysis, the analyst must have the proper specimens. In gathering specimens for testing, one must be absolutely certain that all specimen containers are clean and not contaminated, and that the proper chain of evidence has been maintained. Ideally, the specimens and amounts needed for analyses are as follows:

- Urine = all available.
- Gastric contents = all available.
- Blood = 25 mL (heart), 10 mL (peripheral).
- Brain = 100 g.
- Liver = 100 g (to look for metabolites).
- Kidney = 50 g.
- Bile = all available.
- Vitreous humor (from the eye) = all available. (This specimen’s levels usually lag behind the blood levels by ~1 to 2 h.)
- Hair and nails = hair (include roots), nail (one full specimen). (Hair usually grows ~0.5 in. [1.25 cm] per month and can be used for a segmental analysis.)

Desired specimens for tox analysis

- ☒ **urine = all available**
- ☒ **gastric contents = all available**
- ☒ **blood = 25 ml (heart) - 10 ml (peripheral)**
- ☒ **brain = 100 gm**
- ☒ **liver = 100 gm**
 - look for metabolites
- ☒ **kidney = 50 gm**
- ☒ **bile = all available**
- ☒ **vitreous humor = all available**
 - lag behind blood levels ~1-2 hrs
 - more resistant to decay
- ☒ **hair & nails = hairs (include roots), nail (1 full)**
 - hair grows (~0.5 inches = 1.25 cm)/month

Figure 5-2

Fig. 5-2 summarizes these points.

The results of the analyses must be interpreted with care. The following questions must always be answered:

- Was there the possibility of a laboratory error owing to contamination? Were acid-washed containers used?
- Is the laboratory capable? Is it certified?
- Is the result reproducible? Second opinions should be obtained by utilizing a different laboratory using the same analytical procedures.
- If the level is indicated as being high, who made that determination, and by what standard reference? The term “high” can be subject to interpretation by the individual conducting the analysis.

5.6.3. Analysis of Cremated Remains

Although it would seem, at first glance, that the ashes of a deceased person could be analyzed for toxic substances, this does present some very definite analytical and legal problems. For example, many chemical substances will be burned off at the high temperatures required for cremation, which would yield a false negative result. In addition, legally, one cannot prove that a sample is pure, because it could be contaminated with other cremated remains; therefore, the chain of evidence is broken. One would also not be able to prove that the

poison was in systemic circulation and therefore caused the death. Furthermore, one would not be able to prove in what organ the poison originated, and, therefore, there would be no basis for comparison (e.g., milligrams of arsenic per gram of liver). Although the analysis of cremated remains was used in the poisoning conviction of Graham Young in the United Kingdom in 1972, it seems that this type of evidence has too many pitfalls to warrant its use.

5.6.4. When Should the Criminal Investigator's Suspicion Be Aroused?

The astute criminal investigator might begin to wonder if it is ever possible to detect murder by poison, and if there is anything that might serve to warn an investigator of the possibility. Some things that might come up in an investigation that should send up a red flag are as follows:

- The death occurred in a normally healthy individual. Certainly a person can die without warning, but when this type of death occurs, a deeper look into the cause is called for, including an autopsy.
- An individual interfered with the victim receiving proper medical attention. This may lead one to wonder if that person does not want educated eyes and minds delving into the possible cause of the condition in question.
- There is no sign of violence to the body. This is always an indication that the death could have been the result of a poisoning misadventure.
- The affliction appeared as a natural disease yet failed to respond to normal treatment methods.
- An illness reoccurred in cycles; that is, the victim became ill at home, went to a medical facility and seemed to recover, then went home and became ill again, and so on. This would indicate that there is something in the home environment that is proving unhealthy for the individual. Could it be the chronic administration of heavy metals (e.g., arsenic) in the person's meals? There certainly have been recorded criminal cases in which this has happened, and the poisoner is often not caught in the initial stages of the homicide attempt.
- There are common mysterious symptoms in a common group of people. This could indicate that there has been a mass tampering, or that the supposed specific target was a off the mark of the poisoner.
- There is an individual who is anxious to dispose of food, drink, or medicine of which the victim partook. In this case, it is clear that the person is attempting to foil the investigation by destroying critical evidence.
- An individual prevented friends or relations from being sent for during the victim's illness. The criminal investigator should question what that person did not want others to witness.
- There is an insistence on no autopsy. The criminal investigator should clearly state that one will take place. Once again, the desire not to have educated minds look at the problem comes to the forefront.

When should your suspicion be aroused?

- ☠ a sudden death in a normally healthy person?
- ☠ any interference in getting medical attention?
- ☠ were friends/relatives NOT sent for during illness?
- ☠ a “natural disease” which fails to respond to treatment?
- ☠ death with no visible signs of violence?
- ☠ any history of cyclical illness? **BAD** → **BETTER** → **BAD**
- ☠ were there mysterious symptoms in a common group?
- ☠ anyone anxious to dispose of food/drink/medicine?
- ☠ any individual with a knowledge of poisons?
- ☠ any request for NO autopsy?
- ☠ any request for a rapid cremation?
- ☠ any cause of death offered by someone?
- ☠ any attempt to guide the investigation?



Figure 5-2

- There is an insistence on a rapid cremation. This could be construed as an attempt to burn the primary evidence of the crime and foil the investigation. The criminal investigator should clearly state that an investigation must take place before cremation can proceed.
- While grieving over the loss of a close family member or friend, a certain individual does not freely begin to offer an explanation for the cause of death. Neither will the person attempt to guide the investigation in any way. If the person does, it could very well be an attempt to divert investigators' attention from his or her crime, and investigators must be aware of this.
- An individual shows a familiarity with poisons and possesses literature about poisons. In this case, not just a red flag should go up, but a whole sky full of mental fireworks.

Fig. 5-3 summarizes these important points.

5.7. HOW DID THE POISON GET IN THE PATIENT?

Once it has been determined by autopsy that poison was present in the victim, the question arises: How did the poisoning occur?

It is possible that the cause was accidental. The poison could be present from a natural source, such as heavy-metal contamination of groundwater in the environment, or contamination of the body by leaching of heavy metals

prior to exhumation. Another natural source is food or drink, such as seafood, which causes temporary elevation of arsenic levels after consumption. Elevated levels of poison such as lead can result from occupational exposures, such as in jobs at shooting ranges, electroplating facilities, or smelters. There could also be a possible metabolic cause for the presence of the poison in the deceased, such as the elevated copper levels one sees with a condition known as Wilson's disease. Poisoning could be the result of filling a prescription with the wrong medication or providing incorrect instructions for a drug's use, or it could be the result of contamination in the home environment, such as carbon monoxide.

The poison could have been self-administered, as in the case of the accidental misuse of a product, the unforeseen result of substance abuse, or the intent of the victim to commit suicide.

The poison might have been administered by another person, perhaps from product tampering or with homicidal intent. For example, an individual is found with an extremely high level of insulin (hyperinsulinemia). Did it come from outside the body, or was it naturally produced internally? With this type of compound, it is easy to answer this question. Normally, when the beta-cells of the pancreas produce insulin, they produce one molecule of insulin plus one molecule of what is called C-peptide in a 1:1 ratio. Thus, if the victim's insulin level is high and the C-peptide level is also high, then the insulin source was from inside the body (e.g., from an insulinoma [insulin-producing tumor]). However, if the insulin level is high but the C-peptide level is not also proportionally high, then the source of insulin was from outside the body (injected in an accidental, suicidal, or homicidal event).

In all, the poisoning crime scene is one surrounded by mystery and invisible clues. But when a criminal investigator begins to focus on the possibilities, the mysterious fog begins to clear a little, and the face of the poisoner becomes more visible.

Our ability to detect poisons has greatly improved over the last 100 years, but our ability to suspect poisoning in the first place has not improved, and may have actually gotten worse.

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Chapter 6

The Forensic Autopsy

“Revolted by the odious crime of homicide, the chemist’s aim is to perfect the means of establishing proof of poisoning so that the heinous crime will be brought to light and proved to the magistrate who must punish the criminal.”—M. J. B. Orfila, 1817

6.1. THE AUTOPSY

During an autopsy, the forensic pathologist looks for certain clues that might indicate that a poison could have been involved in the death. These clues could include irritated tissues (from caustic and corrosive compounds); characteristic odors, such as the almond-like odor of cyanide; or Aldrich-Mees lines (white bands on the nails that indicate chronic exposure to heavy metals such as arsenic) (see **Fig. 6-1**).

6.2. POSTMORTEM REDISTRIBUTION—“NECRO-KINETICS”

The pathologist also reviews the results of any toxicological screens, to determine whether they are consistent with his or her pathological findings. Certain cautions in the interpretation of the analytical toxicology results should be observed.

The concentrations of substances revealed by an analytical test will vary, depending on the site of origin of the specimen as well as the length of time that has passed since the initial exposure. The reliability of any postmortem specimen is directly related to the conditions associated with the collection of that specimen and the storage environment. It has become increasingly clear that the blood concentration of many drugs is definitely dependent on the site of collection, and that blood concentration may be significantly higher, or

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Forensic autopsy signs – common poisons**☠ Arsenic**

- *Aldrich-Mees' lines*
- brown pigmentation (temples, eyelids, neck)
- hyperkeratosis (hands & soles of feet)
- gastric irritation

☠ Cyanide

- facial cyanosis
- froth on lips
- projectile vomiting
- red coloration (really uncommon)
- tissue corrosion from alkaline salts
- hardening of stomach wall

☠ Strychnine

- signs may be absent
- mild stomach congestion
- *myoglobin* from strong muscle contractions

☠ Thallium

- hair loss!
- *Aldrich-Mees' lines*
- liver shows fatty infiltration
- heart shows fatty degeneration = “*tabby cat*” striations
- *stomatitis*
- *pulmonary edema*
- gums (bluish line) [from 3-4 week chronic exposure]

Figure 6-1

sometimes lower, than at the time of death. If the pathologist removes blood merely from the left side of the heart or, worse yet, obtains a sample from the chest or abdominal cavity of the victim, this can yield results that may lead the investigator far astray from the actual meaningful and more accurate analytical results. Unfortunately, the ability to interpret the results of toxicological analyses has not kept pace with the great advancements that have been made in the detection limits of analytical instrumentation.

It is unfortunate that the literature available on postmortem levels in fatal intoxications typically consists only of case reports. It would be of extreme value to forensic scientists if an international database listing chemical sub-

stances that have been detected in bodies in relation to the time interval since death existed. This database should list the name of the substance, the type of specimen, the time interval since death that the specimen was obtained as well as analyzed, the determined level, and the type of analytical technique utilized. In other words, how long after death was it possible to prove the presence of a substance in the body? This information has major implications when considering the possible value of exhuming the body of a victim thought to have been poisoned.

It is well known that chemical substances redistribute in the body, a phenomenon often referred to as “anatomical site concentration” or “post-mortem redistribution.” This phenomenon could also well be called “necrokinetics,” or the movement of substances after death has occurred. Many studies have shown that the concentrations of certain drugs, such as propoxyphene and the tricyclic antidepressants, are increased in heart blood postmortem. Some researchers have proposed that drug concentrations obtained from liver specimens are much better indicators of toxicity (Hilberg, Rogde, & Morland, 1999; Jones & Pounder, 1987; Langford & Pounder, 1997).

Factors that can alter the movement of substances, and, therefore, their final concentrations in an analytical specimen, include acid-base changes in the body after death and the volume of distribution (V_d) of the substance in question. *Volume of distribution* is defined as that volume of fluid into which a drug appears to distribute to a concentration equal to that in plasma. Drugs with a low V_d will become less ionized as the pH (acidity) in the body decreases (i.e., becomes more acidic), and, therefore, their solubility in the surrounding tissues will increase. Examples of drugs that will shift with this change in acidity include salicylates, theophylline, and phenobarbital.

The ideal toxicological sample would be a peripheral sample obtained from a blood vessel that had been ligated shortly after death. Unfortunately, this ideal is seldom obtained in the case of homicidal poisoning.

6.3. ANALYTICAL GUIDELINES

The following guidelines must be kept in mind when carrying out a toxicological analysis:

- Postmortem concentrations are absolutely site dependent.
- Samples taken from the same site in the body will show different concentrations postmortem, depending on the time the sample was obtained.
- From a single postmortem measurement, no realistic calculation of the absorbed dose to create that level can really be made.

- When obtaining samples for analysis, a clean instrument must be used for each specimen, to avoid possible cross-contamination of specimens and erroneous results.
- Both the death scene investigator and the pathologist can provide crucial information to the toxicological analyst.
- Absolute chain of custody must be maintained on all specimens throughout the process from their procurement through their toxicological analyses.

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Chapter 7

Proving Poisoning

*“It is a capital mistake to theorize before one has data. Insensibly one begins to twist facts to suit theories, instead of theories to suit facts.”—
Sherlock Holmes, by Sir Arthur Conan Doyle*

Let us begin our discussion of the proof that a murder by poison has been committed by discussing the proper utilization of the services of an analytical toxicology laboratory, because it will play a key role in the detection of the crime.

First of all, a “shotgun” approach to detection will most likely not be successful. One cannot hand analytical toxicology personnel a specimen and say that poisoning is suspected and ask them to prove that a poisonous compound is present in the specimen. The analysts need some guidelines as to what compounds are suspected. These guidelines come from the criminal investigator’s analysis of the death scene, as well as the pathological findings that derived from the autopsy. The key here is that once there has been a death, a qualified medical examiner should be called in to the case immediately.

The investigator should also be aware that the concentration of compounds may differ depending on the site of origin of a blood specimen, in that cardiac (heart) blood may differ from peripheral (away from the central portion) blood in the quantitative analyses.

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7.1. KEY ELEMENTS TO BE PROVEN

The following elements are key to proving that someone has been poisoned:

- *Discovery*: This consists of legally proving that a crime was committed, and demonstrating beyond *reasonable doubt* that death was caused by poison, administered with malicious or evil intent to the deceased. Never forget the importance of the chain of evidence on all investigational specimens.
- *Motive*: This is critical because the investigator must clearly establish the instigating force behind the action. Why would anyone want to carry out such an act on the victim? This is where the close study of the victim (victimology) becomes central to the case.
- *Intent*: This constitutes the purpose or aim that an individual would have in commission of the act. Here the investigator will cover the desired outcome of the criminal act.
- *Access to the poison responsible for the death*: The criminal investigator must present such evidence as proof of sale of the poison, with such things as receipts or the signature on a poison register at the point of sale. Is there any original packaging, wrappers, or containers associated with the suspect? It may suffice to prove that a suspect has had access at a workplace, used toxins or poisons in his or her occupation, or had a hobby that involved the use of the poison in question.
- *Access to the victim*: Is there any proof that a suspect has knowledge of the victim's daily habits, could have had the opportunity to overcome any of the victim's normal defenses, and was able to administer the poison either directly or indirectly?
- *Death caused by poison*: There must be sufficient, sound evidence that would induce a reasonable person to come to this conclusion. Remember that in order to prove death by poison, the presence of the poison in the systemic circulation and/or body organs must be proven. The presence of the poison only in the gastrointestinal (GI) tract does not prove death by poisoning. The GI tract from the mouth to the anus is much like a garden hose, hollow and open at both ends, and therefore outside the topological framework of the body. Consequently, to have met its fatal potential, the poisonous compound must have been absorbed through the walls of the gut and entered the body's systemic circulation so that it could get to the site that caused the untoward effect.
- *Death homicidal*: This cannot be proven analytically or by autopsy but depends on the work of the criminal investigator at the crime scene, and examination of witnesses. This proof must categorically eliminate the possibility that the death resulted from an accident, intentional substance abuse, or an act of suicide.

In conclusion, to ensure the possibility of a conviction, it is imperative that proof of these investigational elements clearly leads to the conclusion

that the death was caused by poison, that the accused administered the poison to the deceased, that it is not possible or probable that any other person could have administered the substance, and that the accused was well aware of the poison's lethal effects on the victim.

7.2. STATISTICAL ANALYSES OF POISONINGS IN THE UNITED STATES

To my knowledge, no major epidemiological study has ever been done prior to the 1990s that reviews poisoning homicides in the United States. The first such study was carried out by Westveer, Trestrail, and Pinizotto (1996) and was published in 1996. These investigators decided to examine the data contained in the Uniform Crime Reports (UCR), maintained by the Department of Justice, from 1980 to 1989. These reports, submitted annually from police agencies across the United States, provide information on the victim and the offender in various crimes. This information includes the month; the year; the state; victim information (gender, age, and race); offender information (gender, age, and race); classification of the poisonous agent as drug, nondrug, or fume; the relationship between the victim and the offender; and a motive classification group. From 1980 to 1989, a total of 202,785 homicides were reported, and in this compilation, of all homicides, there were 292 poisoning cases of a single offender on a single victim. This total represents 14 poisoning cases per 10,000 homicides. The following information was determined from a statistical analysis of the UCR data:

Gender relationships:

- The number of male victims and female victims was equal.
- If the victim was a female, the offender was usually a male.
- If the victim was a male, the offender could have been either a male or a female.
- Twice as many offenders were male than female.

Racial relationships:

- The victim and offender were usually of the same race.
- The victims were mostly white.
- If the victim was white, the offender was usually a male.
- If the victim was black, the offender was either a male or a female.
- Twice as many black victims were male than female.
- The number of female white victims and male white victims was equal.
- For black or white offenders, there were twice as many males as females.

Age characteristics:

- The number of victims was highest in the age range of 25–29 yr.
- The number of offenders was highest in the age range of 20–34 yr.

Geographic relationships:

- Poisonings average 1.47 per million people per year.
- Poisonings were highest in the western region; the state of California had the highest number.

Other relationships:

- Probably one of the most startling revelations from this study was that the unknown offender rate for poisoning cases was 20–30 times higher than for nonpoisoning homicides. This is another indication that law enforcement, as well as other forensic scientists, need to sharpen their investigative skills in the area of murder by poison.
- More victims did not have a relationship with the offender's family. This is an unusual result, because it would seem that most homicidal poisonings would be a crime of the domestic environment.
- Nondrug poisons were used by 50% more males than females, and drug poisons were used by three times as many males as females.
- Poisoning rate by year or month was relatively constant. There was no month or year that showed a significant increase or decrease in the number of poisoning cases reported.

Westveer, Jarvis, and Jenson (2004) repeated the study just discussed. They examined the data for the period of 1990–1999, and the results were very similar. They did find that there were 346 poisonings in the 186,971 total homicides for the decade. The incidence of 18 poisonings per 10,000 homicides represents an increase of 29% over the value found for the prior decade (Westveer, Jarvis, and Jenson, 2004).

Future analyses should examine the data that represent poisoning cases in which a single offender poisoned multiple victims—the serial poisoner—and the relatively rare cases that represent multiple offenders on a single victim.

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Chapter 8

Poisoners in Court

“I have three rules. I never believe what the prosecutor or police say. I never believe what the media say, and I never believe what my client says.”

—Attorney Alan M. Dershowitz

The majority of the death scene investigations have been completed. Evidence has been gathered that points to a defendant as the probable perpetrator of a murder by means of poison. It is now time to take the evidence that proves method, motive, and opportunity to the jury. Let us take a look at some of the differences that might be encountered in the poisoning trial vs trials for murder by means of more traditional weapons.

To establish that the death was owing to poisoning, one must be able to prove the following (see **Fig. 8-1**):

- That chain of custody was maintained, to include who had possession of the evidence, and when possession was taken.
- That the poison was present based on analysis.
- That the poison was absorbed systemically and was in the body's circulation. (Remember that the gut is much like a garden hose.)

8.1. BATTERY BY POISON

One may commit a battery by causing injury through poisoning. Battery, of course, occurs when a person is injured in a dangerous situation intentionally created by the defendant.

A defendant is held culpable in a battery charge if he or she acts with either intent to injure or criminal negligence. “Aggravated battery” is punishable as a felony and results from actions taken with the intent to kill. In this

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Death from poisoning?

To establish poisoning death one must be able to prove the following:

- ☠ evidence chain-of-custody was maintained!
 - who had evidence possession?
 - when was evidence possession taken?
- ☠ presence of poison proven by analysis!
- ☠ poison absorbed systemically = circulating

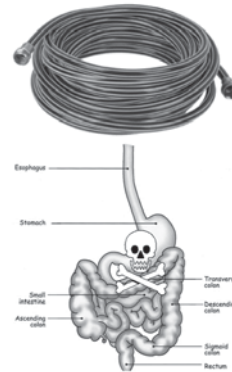


Figure 8-1

case, usually the defendant must have intended to cause the specific result; otherwise the crime is considered a “regular battery.”

8.2. STANDARD DEFENSE ARGUMENTS

When a poisoner goes on trial, obtaining a conviction will be far from simple. It is at this point that the results of the investigator’s careful and detailed work will become critical. In this section, I discuss some prosecution strategies and defense tactics that might come into play.

Unlike in trials for murder that has been carried out by means of a visibly detectable weapon (e.g., gun, knife, rope), in trials for murder by poison, one must not forget that the vast majority of the trial evidence will be indirect evidence, or circumstantial evidence. In the poisoning crime, there will be few, if any, witnesses.

The defendant will attempt to explain the facts being presented by the prosecutor. The poisoner’s best defense is the simplest answer that explains the facts. Some of the possible counterarguments that may be attempted by the defense team are discussed next.

8.2.1. Poisoning Not the Cause of Death

The defense will attempt to prove that the poison did not cause the death, that the victim died from another cause. For example, the defense might argue

that the cause of death was a subdural hematoma from a fall. At this point, the detailed combined work of a forensic pathologist and an analytical toxicologist will come into play.

8.2.2. Poisoning Not Homicidal

The defendant will attempt to downplay his or her involvement in the death by trying to convince the court that the victim caused his or her death by self-administering the poison, either with suicidal intent or as the fatal result of substance abuse.

8.2.3. No Homicidal Intent

It could be suggested that the substance was administered by the accused but not with the intent to murder. One is reminded of the Arthur Ford case in the United Kingdom, discussed in Chapter 1, in which the offender administered candy containing cantharides in order to sexually arouse two secretaries in his office. He was convicted of manslaughter, because the court agreed that his intent was not to kill. Another case would be the unfortunate death of comedian John Belushi from an overdose of drugs administered by another person.

8.2.4. Substance Not a Poison

Legal experts will often argue over the acceptable definition of a “poison.” Is it a drug, and not a poison? Remember Paracelsus’ definition of a poison: that it is solely related to dose. The major factors that determine the potential lethality of any substance are concentration and duration of exposure. This was clearly stated in 1915 by the German chemist Fritz Haber, who developed what was known as the “CT product.” His formula was $C \times T = \text{a constant}$. What this means is that the product of the concentration (C) of a poison and the survival time (T) of the victim is a constant value. For example, breathing a certain concentration of carbon monoxide for a specified amount of time will produce the same effects as breathing half the concentration for twice that time—the toxicological result should be a constant outcome.

8.2.5. The Accused Had a Reason to Have the Poison in His or Her Possession

As to why the accused had the poisonous substance in his or her possession, it may be argued that it was acceptably associated with his or her job (e.g., chemist) or hobby (e.g., photography), or that the substance was being used as a domestic pesticide or herbicide to rid the area of unwanted pests or plants.

8.3. PROBLEMS IN PROVING INTENTIONAL POISONING

The attorney for the prosecution is bound to find himself or herself beset with some unique problems in a poisoning trial. One of the major problems is that the majority of the evidence will be circumstantial (indirect). In the typical murder by poison, there are no witnesses to the act. Another problem is that there may not be an accepted legal definition of a poison. There is also bound to be a great deal of dispute over the scientific evidence, and much of the evidence is likely to be rebutted by the defense's technical experts.

The goal in obtaining a conviction is to prove that the death was caused by a poison, ideally with a combination of pathological and analytical evidence. It must be proven that the accused administered the poison because he or she had the access and opportunity, and that it is not possible or probable that any other person could have administered the substance. It is also imperative to prove that the accused was aware of the poison's lethality.

It would be wise for the prosecution to prepare its case while keeping in mind what I have named the "Conviction Pyramid" (see **Fig. 8-2**). This concept represents the four major points that must be proven to be connected in the case: the victim, the poison, the offender, and the motive.

The case usually begins with the discovery of the two points representing leg 1, Victim-Poison, but the proof must also encompass the more difficult legs of the Conviction Pyramid numbered 2, 3, and 4: Offender-Victim, Offender-Poison, and Motive/Intent-Offender, respectively. Unless each of these points is unquestionably linked, there may be great difficulty in obtaining a conviction.

Laying out the poisoning case comes down to trying to solve the following quasi-algebraic equation, in which **A** = the plaintiff, **B** = the victim, and **X** = the poison:

- **B** = Dead! = the VICTIM ("who")
- **X** is found in **B** = the CAUSE ("what")
- **A** had a reason for eliminating **B** = MOTIVE ("why")
- **A** obtained **X** = METHOD ("how")
- **A** had access to **B** = OPPORTUNITY ("when," "where")
- **A** poisoned **B** = ACCUSATION ("offender who")

Always remember the logic argument known as Occam's razor: the simplest solution to a problem is probably the correct one.

As one can see, the poisoning trial is beset with unique conviction pitfalls, but with proper investigational research, proper chain of evidence, and detailed planning, the chances of a conviction are greatly increased.

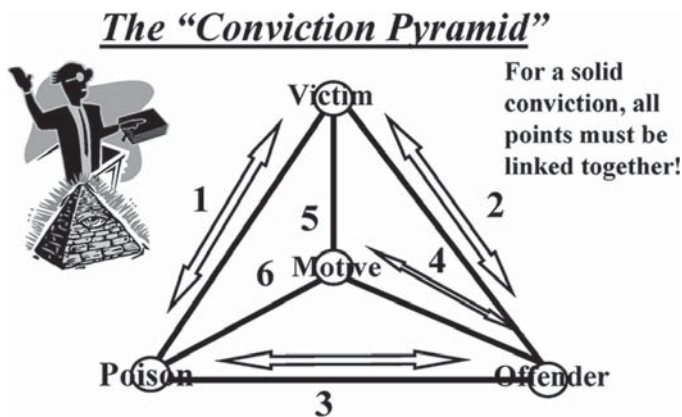


Figure 8-2

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Chapter 9

Poisoning in Fiction

MARTHA: "Well, dear, for a gallon of elderberry wine, I take one teaspoonful of arsenic, and add a half a teaspoonful of strychnine, and then just a pinch of cyanide."—*Arsenic and Old Lace*, Joseph Kesselring

It is often said that life can imitate art, and so it would behoove us to look at the use of poisons in fictional works, both written and visual. The scenario of an individual reading a novel or watching a film, and obtaining ideas that could lead to committing an actual murder, is not beyond the realm of possibility.

9.1. POISONS THAT HAVE BEEN USED IN BOOKS AND FILMS

In gathering information on how poisons have been used in fictional writing, I analyzed 187 texts. The types of poisons used varied slightly from those that have been used in actual cases of murder, but the primary ones did appear. In fiction, cyanide was used more often than arsenic. **Table 9-1** summarizes the poisons used in fictional writings.

It is also important to look at the visual media as well, because some movies can create ideas in the fertile mind of the poisoner. **Table 9-2** summarizes some of the films that have used poisons in their plots.

As part of the investigation of a criminal poisoning, it would be wise for the investigator to look at any fictional literature and visual media to which the suspect had access.

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Table 9-1
Poisons Used in the Literature (a Review of 187 Works)

Poison	No. of cases	%
Acid	1	0.5
Aconite	2	1.1
Air (by injection)	1	0.5
Akee	1	0.5
Antimony	1	0.5
Arrow poison	1	0.5
Arsenic	13	7.0
Atropine	5	2.7
Barbitone	3	1.3
Bowl cleaner	1	0.5
Carbon monoxide	3	1.6
Chloral	1	0.5
Chloral hydrate	2	1.1
Coal gas	2	1.1
Cocaine	2	1.1
Coniine	1	0.5
Curare	4	2.1
<i>Cyanea capillata</i>	1	0.5
Cyanide	25	13.4
“Devil’s Foot Root”	1	0.5
Digitalin	3	1.6
Digitalis	3	1.6
Digitoxin	1	0.5
Drugs	1	0.5
Fear: of poison death	2	1.1
Food poisoning	1	0.5
Formic acid	1	0.5
Fungus	1	0.5
Gelsemium	1	0.5
Hemlock	1	0.5
Henbane	1	0.5
Hexabarbital	1	0.5
Hyoscine	3	1.6
Indian hemp + datura	1	0.5
Jimson weed	2	1.1
L-Thyroxine	1	0.5
Microorganisms: cholera	1	0.5

Table 9-1 (Continued)
Poisons Used in the Literature (a Review of 187 Works)

Poison	No. of cases	%
Morphine	6	3.2
Multiple poisons	1	0.5
Muscarine	1	0.5
Mushrooms	15	8.0
Narcotic	1	0.5
Nicotine	6	3.2
Nitrobenzene	2	1.1
Oleander	2	1.1
Paint thinner	1	0.5
Phenylbutazone allergy	1	0.5
Phosphorus	1	0.5
Photographic developer	1	0.5
Physostigmine	2	1.1
Poisoned darts	1	0.5
Poison gas	1	0.5
Procaine	1	0.5
Purvisine (an alkaloid)	1	0.5
Ricin	2	1.1
Serenite (an invented poison)	1	0.5
Solanine	1	0.5
Streptomycin allergy	1	0.5
Strophanthin	5	2.7
Strychnine	6	3.2
Taxine	1	0.5
Tetra-ethyl-pyrophosphate	1	0.5
Tetrodotoxin	1	0.5
Thallium	2	1.1
Toxin	1	0.5
Trinitrin	1	0.5
Tuberculin	1	0.5
Unidentified native poison	2	1.1
Unknown poison	13	7.0
Venom: bee	2	1.1
Venom: snake	4	2.1
Virus	1	0.5
Warfarin	1	0.5
Total	187	100.0

Table 9-2
Poisons Used in Motion Pictures (a Review of 15 Works)

Film title	Date	Poison used
<i>Attack of the Mushroom People</i>	1964	Mushrooms
<i>Beguiled, The</i>	1971	Mushrooms
<i>Black Widow</i>	1987	Penicillin allergy + unknowns
<i>Court Jester, The</i>	1956	Unknown
<i>Dead Pool, The</i>	1988	Street drug
<i>D.O.A.</i>	1949	Iridium
<i>D.O.A.</i>	1988	Radium chloride
<i>Fer-de-Lance</i>	1974	Venom: snake
<i>Flesh and Fantasy</i>	1943	Aconite
<i>Goliath Awaits</i>	1981	Algae extract (Palmer's disease)
<i>Pope of Greenwich Village, The</i>	1984	Lye (sodium hydroxide)
<i>Serpent and the Rainbow, The</i>	1988	Tetrodotoxin
<i>Throw Mama from the Train</i>	1987	Lye (sodium hydroxide)
<i>Venom</i>	1982	Venom: snake
<i>Young Sherlock Holmes</i>	1985	Dart poison

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Chapter 10

Conclusion

“If all those buried in our cemeteries who were poisoned could raise their hands, we would probably be shocked by the numbers!”—John H. Trestrail III

As Sir Arthur Conan Doyle’s Sherlock Holmes stated to his partner, Dr. Watson, “The game is afoot,” so it is with investigators and the criminal poisoner. As homicide investigators, we must always remember that unless we remain ever vigilant, we will lose the game. Unless the possibility of poisoning is considered in the first place, the critical evidence of the crime will most likely be buried with the victim, and the poisoner will walk off into the sunset, with a feeling of superior intellect and smugness.

The *prime directive* for any criminal investigation is that *every death must be considered a homicide until the facts prove otherwise*. To this we must now add a new *subdirective* for the criminal investigation of homicidal poisonings: *Every death with no visible signs of trauma must be considered a poisoning until the facts prove otherwise*.

The investigative key is to put all the clues together, and where they overlap, one should be able to match the most probable offender. So let us review the basic categories of clues as they relate to poisoning homicides:

- **WHO** was the victim? Was the victim a specific or random target? Could it be a *camouflaged* poisoner hiding behind a tampering? Why would anyone want to kill this individual, as determined by their “victimology”? (see **Fig. 10-1**).
- **WHAT** was the poisoning weapon? Remember that whether it is a solid, liquid, or gas, they are just atoms and molecules, which carry out their biochemical destruction in the manner of a “chemical monkey wrench.” Never forget that it is imperative that the poison be proven to have been in the victim’s systemic circulation (see **Fig. 10-2**).

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Who?

study the victim = “victimology”

How? + Why? = Who



Figure 10-1

What?

one must prove the poison was systemic

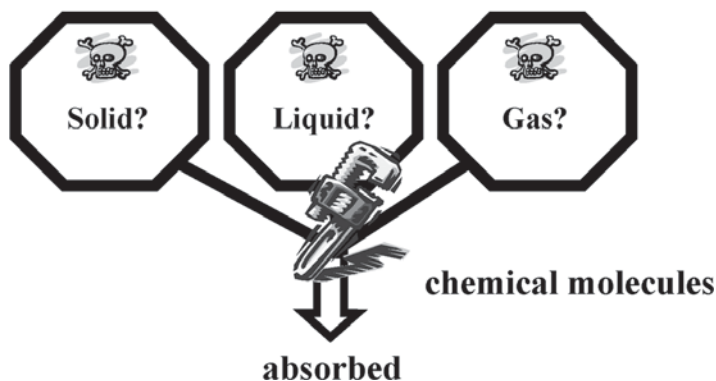


Figure 10-2

- **WHERE** did the crime take place? Remember that a poisoning may have multiple crime scenes (procurement, preparation, administration, disposal, and ultimately the death scene) (*see Fig. 10-3*).
- **WHEN** was the poison administered to the victim? Remember that the time from administration till death is dependent on the concentration and toxicity of the substance. With an acute dose one sees sudden onset. Carry out analyses on blood, urine, and gastric contents (BUG). Look for poisons that have a rapid action (e.g.,



Figure 10-3

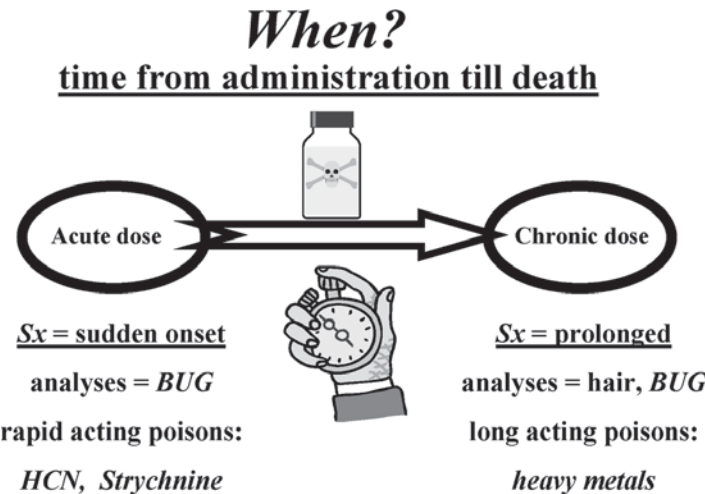


Figure 10-4

cyanide, strychnine). With a chronic dose situation, look for prolonged symptoms. Carry out analyses on the victim's hair looking for heavy metals (e.g., arsenic, antimony, lead, thallium) (see Fig. 10-4).



Figure 10-5

How?
look at all items the victim used routinely



Figure 10-6

- **WHY** was the victim chosen? Did the victim stand between a goal that was so important to the offender that elimination of this individual led to obtaining the goal? (see **Fig. 10-5**).
- **HOW** was the poison administered? Look at the items used routinely and solely by the victim (see **Fig. 10-6**).

Putting all your clues together

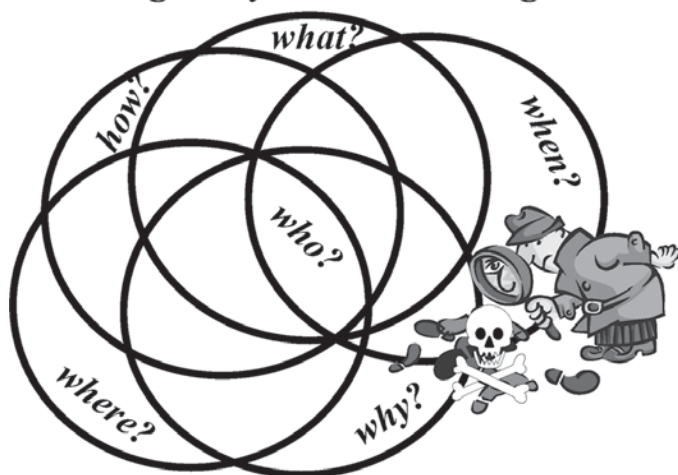


Figure 10-7

The bottom line!

“If one does NOT consider poison in the first place, one will NEVER detect it!”



Figure 10-8

The clues provided by the answers to these questions will point to an individual who is a likely offender (see **Fig. 10-7**). The bottom line is that if investigators do not consider poisoning they will never detect it.

The entire purpose of this important reference work can be summarized in the single graphic representation in **Fig. 10-8**. The results of our missing

Think about it!

***“If all those buried in our
cemeteries, who were poisoned,
could raise their hands, we would
probably be shocked by the
numbers!”***



Figure 10-9

deaths from poisoning over the years have resulted in a situation which is best depicted by **Fig. 10-9**. Think about it! And now on the following page, a concluding poetic challenge, from the poisoner to the newly educated “Toxic Avengers.”

“The Poisoner”

by

John H. Trestrail III, RPh, FAACT, DABAT

The Borgias, DeMedicis and all those past—
you may have thought you had seen the last.

But, we poisoners are still around today.

And if you miss my crime, I'll get away.

The body lies there neat and clean,
as the cause of death is seldom seen.

And the coroner may take time to pause—

“is this death due to a natural cause?”

An autopsy or tox screen may reveal death's why,
but I hope the case will just slip by.

My crime is quiet and well thought through.

For you're used to violence—can I fool you?

The event's rarity is on my side.

For I count on you burying my homicide.

And though I roam free round the nation,

I live in fear of an exhumation.

The clues I leave may be hard to find,

you see, to me, I have a superior mind.

My weapons are there before your eyes,
but they are so very small—of molecular size.

I don't think you'll have a notion,

for mine is murder in slow motion.

It gives me time to just slip by,

and create my perfect alibi.

Where to look for me isn't clear.

I may be far, or I may be near.

I could be a stranger, though it is quite rare,
for I'm probably related to the victim there.

I chose the place, the means, and time,
for poisoning is usually a household crime.

The knowledge gained by my living close,
made it so very easy to deliver the dose.

Seeing it as poisoning would be profound,
but I think you'll miss it as you look around.

I'm a different kind of killer as you can see.

I am a POISONER—can you catch me?

Appendix:

Some Common Homicidal Poisons

*ANTIFREEZE (METHANOL [CH₃-OH],
OR ETHYLENE GLYCOL [HO-CH₂-CH₂-OH])*

Form:

- Methanol (MeOH), also known as methyl alcohol or “wood alcohol,” is the simplest of the alcohols. For chronic alcoholics, this alcohol sometimes serves as a cheap substitute for ethanol (grain alcohol), as in the use of canned Sterno® as a source. Abuse of this toxic alcohol can have very dire consequences (e.g., blindness).
- Ethylene glycol is chemically known as 1,2-ethanediol. It is a slightly viscous liquid.

Color:

- Methanol: Colorless.
- Ethylene glycol: Colorless.

Odor:

- Methanol: Slight alcoholic odor.
- Ethylene glycol: Odorless.

Solubility:

- Methanol: Very water soluble.
- Ethylene glycol: Very water soluble. It can absorb twice its weight in water.

Taste:

- Methanol: A burning taste.
- Ethylene glycol: Has a sweet taste, which has often led to the accidental ingestion of this substance by household pets.

From: *Forensic Science and Medicine: Criminal Poisoning, Second Edition*
By: J. H. Trestrail, III © Humana Press Inc., Totowa, NJ

Source:

- **Methanol:** Is a common ingredient in windshield-washing solutions, duplicating fluids, and paint removers and is commonly found in gas-line antifreeze, which may be 95% (v/v) methanol.
- **Ethylene glycol:** Is commonly found in radiator antifreeze (in a concentration of ~95% [v/v]), and antifreeze products used in heating and cooling systems.

Lethal Dose:

- **Methanol:** The fatal dose is estimated to be 30–240 mL (20–150 g).
- **Ethylene glycol:** The approximate fatal dose of 95% ethylene glycol is estimated to be 1.5 mL/kg. For a person weighing 150 lb, this would be 102 mL (3.5 fluid ounces).

How It Kills:

- **Methanol:** The compound is oxidized in the body by the enzyme alcohol dehydrogenase into the more toxic compound formaldehyde (more commonly found in mortuary parlors), which in turn is further oxidized by aldehyde dehydrogenase to formic acid (the same compound that gives ants their sting).
- **Ethylene glycol:** The compound is oxidized in the body by the enzyme alcohol dehydrogenase into the more toxic compound oxalic acid (a substance that serves as the common ingredient in some rust removers). This metabolite then combines with circulating calcium in the blood to form characteristic “envelope-shaped” (dihydrate) crystals of calcium oxalate, which can be found in the kidneys and urine. Needle-like crystals (monohydrate) can also be formed, which can lead to kidney damage.

Poison Notes: None.

Victim of Antifreeze

Administered: Both antifreeze substances can be easily administered in beverages that are expected to have a sweet or alcoholic taste.

Symptom Onset Time Interval:

- **Methanol:** From a few hours to 30 h.
- **Ethylene glycol:** From 4 to 12 h.

Symptoms—Acute:

- **Methanol:** In the first few hours, the victim will appear inebriated and have gastritis. After a period of about 30 h, the victim will experience metabolic acidosis, visual disturbances, blindness, seizures, and coma, and death may occur. Patients have described the visual disturbances as being similar to

Antifreeze mechanisms

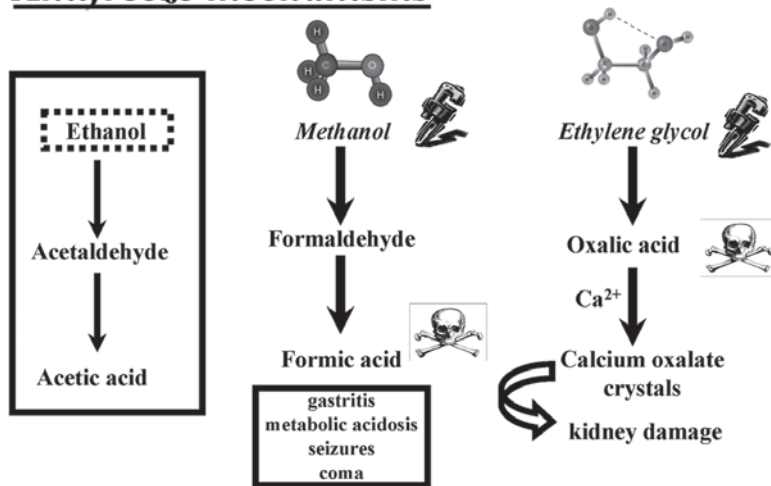


Figure App.-1

standing in a snowfield. The retinal toxicity is caused by the accumulation of formic acid.

- Ethylene glycol: In the first few hours, the victim will appear inebriated. Gastritis and vomiting may occur. After a period of 4–12 h, the patient will experience acidosis, hyperventilation, convulsions, coma, and cardiac conduction disturbances and arrhythmias. Kidney failure is common.

Symptoms—Chronic:

For both types of antifreeze, chronic administration is less likely. The symptoms, however, would be the same, with a more gradual onset.

Disease Confusion:

- Methanol: Uremia, diabetic ketoacidosis, and alcoholic ketoacidosis.
- Ethylene glycol: Diabetic ketoacidosis and lactic acidosis.

Victim Notes: None.

Detection of Antifreeze

Specimens: For both types of antifreeze, beverages, liquid medications, and blood; and in the deceased, vitreous humor (from the eye).

Method:

- Methanol: Gas chromatography (GC), the same technique as used for ethanol determination. An elevated anion gap acidosis in the patient supports

the diagnosis. NOTE: Enzymatic methods, sometimes used for ethanol determinations, do *not* readily detect methanol.

- Ethylene glycol: GC. NOTE: It has been observed that the concentration of ethylene glycol in postmortem tissue may rapidly decline during refrigeration if a colorimetric method for analysis is used. A high osmolar gap in the patient is often indicative of intoxication.

Toxic Levels (fatal cases): (1)

- Methanol

Units	Blood	Brain	Liver	Kidney	Urine
mg/L	400	?	?	?	?
Range (mg/L)	200–6300	?	?	?	?

- Ethylene glycol

Units	Blood	Brain	Liver	Kidney	Urine
g/L or g/kg	2.4	2.0	6.7	4.6	5.7
Range (g/L or g/kg)	0.3–4.3	0.3–3.9	0.2–15.1	0.2–11.3	0.6–10.8

Analysis Notes: None.

Selected Antifreeze Homicide Cases

Bobbie Jan Nichols (1992)
 Angelina Rodriguez (2000)
 Julia Lynn Turner (2001)
 Sandra Kay Baker (2003)

ARSENIC

Form: Metallic arsenic (As) is a steel-gray, brittle metal. Arsenic trichloride (AsCl_3) is an oily liquid. Arsenic trioxide (As_2O_3) is a crystalline solid and it can also exist as arsine gas (AsH_3). *Lewisite*, a gas used as a weapon in war, is a derivative of arsine.

Color: Metal, steel gray; salts, white.

Odor: Odorless, but arsenic can produce a garlicky odor to the breath.

Solubility: Arsenical salts are water soluble.

Taste: Almost tasteless.

Source: Pesticides, rodent poison, ant poison, homeopathic medications, weed killers, marine (copper arsenate) and other paints, ceramics, livestock feed.

Lethal Dose: Acute, 200 mg (As_2O_3); chronic, unknown.

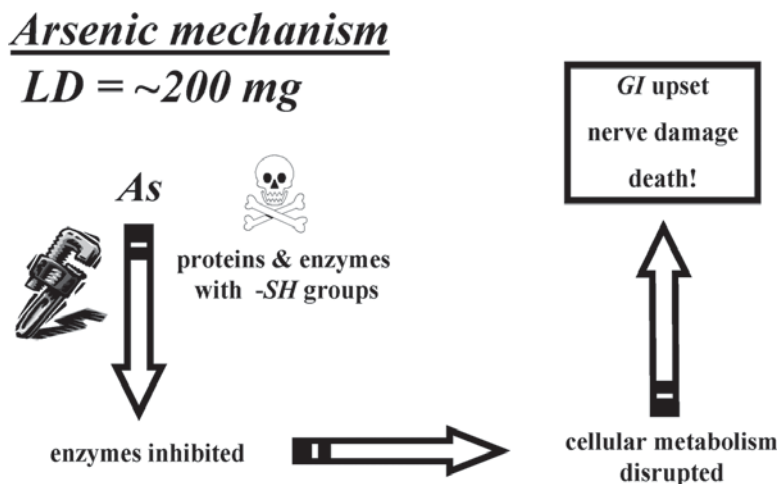


Figure App.-2

How It Kills: Arsenic is a general protoplasmic poison; it combines with sulfhydryl ($-\text{SH}$) groups on enzymes to inhibit their normal function. This inhibition results in disruption of normal metabolic pathways related to energy transfer.

(See Fig. App. 2.)

Poison Notes: The trivalent arsenic (As^{+3}) is more toxic than the pentavalent (As^{+5}) form. Arsenic is one of the oldest poisons used by humans.

Victim of Arsenic

Administered: Often administered to victim in food or drink.

Symptom Onset Time Interval: Hours to days.

Symptoms—Acute: Gastrointestinal (GI) (30 min to 2 h postexposure): vomiting, bloody diarrhea, severe abdominal pain, burning esophageal pain, metallic taste in the mouth. Later symptoms include jaundice, kidney failure, and peripheral neuropathies (destruction of the nervous system). Death from circulatory failure can occur within 24 h to 4 d.

Symptoms—Chronic: GI (diarrhea, abdominal pain), skin (hyperpigmentation of palms and soles), nervous system (symmetrical sensory neuropathy with numbness and loss of vibratory or positional sense, burning pain on the soles of the feet), other localized edema (face, ankles), sore throat, stomatitis (mouth inflammation), pruritis (itching), cough, tearing, salivation, garlic odor on breath, Aldrich-Mees lines (horizontal white lines that nor-

mally take 5 to 6 wk to appear after the exposed nail bed area grows), hair loss.

Disease Confusion: Gastroenteritis, neurological disease.

Victim Notes: In homicides the amount of arsenic could be administered in a single, large acute dose or in frequent, small chronic doses to make the symptoms appear like those of a progressing natural illness. In suicides, the amount of arsenic taken is usually large.

Detection of Arsenic

Specimens: Food, beverages, medications, blood, urine, gastric contents, hair, nails, autopsy organ specimens.

Method: Colorimetric, atomic absorption.

Toxic Levels: (1)

Blood	Urine	Gastric	Other
0.6–9.3 mg/L	3300 µg/L	Unknown	3 ppm (hair/nails) >1 µg/g dry weight

Analysis Notes: Arsenic can be detected in hair and bones many years after poisoning. Several hairs pulled out by the root should be sent for analysis with a clear indication of which end of the hair is the root. Segmented hair analysis using neutron activation gives an indication of arsenic exposure over the last several months.

Selected Arsenic Homicide Cases

Mary Blandy (1752)
 Madeline Smith (1857)
 Florence Maybrick (1889)
 Johann Hoch (1905)
 Henry Seddon (1911)
 Mabel Greenwood (1919)
 Herbert Armstrong (1921)
 Michael Swango (1985)
 Marie Hilley (1986)
 Blanche Moore (1988)

BOTULINUS TOXIN

Form: Usually in the liquid form from culture medium.

Color: Colorless.

Odor: Odorless.

Botulinus toxin mechanism

LD = ~50 ng

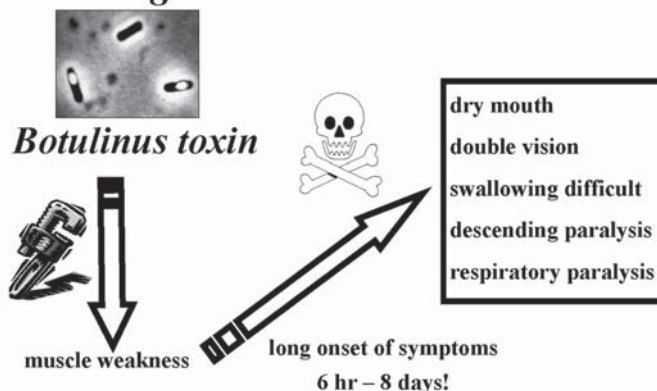


Figure App.-3

Solubility: Water soluble.

Taste: Tasteless.

Source: Produced by the bacteria *Clostridium botulinum*.

Lethal Dose: Acute, 50 ng; chronic, not applicable. Estimated to be as little as 0.1 mL of contaminated food. This substance is the most toxic known. The toxin is 7,000,000 times more lethal than cobra venom.

How It Kills: Botulinus toxin irreversibly binds to cholinergic nerve terminals and prevents the release of acetylcholine from the axon. Severe muscle weakness results, and subsequently death from respiratory failure.

(See Fig. App. 3.)

Poison Notes: The toxin can grow in home-canned food at pH > 4.5.

Victim of Botulinus Toxin

Administered: Can be administered in cool food or drink. Heating to a boiling temperature destroys the toxin within a few minutes.

Symptom Onset Time Interval: May be slow to onset (2 h to 14 d). Death may occur as early as 10 h after the symptoms first appear.

Symptoms—Acute: Dry, sore throat; dry mouth; dizziness; vomiting; stomach upset; difficulty in swallowing; difficulty in speaking; double vision; drooping eyelids; cranial nerve weakness; progressive symmetric descending paralysis; and respiratory arrest.

Symptoms—Chronic: Not applicable.

Disease Confusion: Viral illness, Guillain-Barré syndrome, stroke, tick paralysis, heavy-metal poisoning, adverse drug reactions, and many other conditions.

Victim Notes: None.

Detection of Botulinus Toxin

Specimens: Diagnosis is confirmed by determination of the toxin in serum, stool, or a wound; food; beverages; medications; blood; urine; and gastric contents. The test may be negative if the samples were collected late or the quantity of the toxin is small.

Method: Analysis is usually carried out by local health departments, or the Centers for Disease Control in Atlanta, Georgia.

Toxic Levels: (1)

Blood	Urine	Gastric	Other
Not applicable	Not applicable	Not applicable	Not applicable

Analysis Notes: None

Selected Botulinus Toxin Homicide Cases

No cases on record.

CYANIDE

Form: In liquid form, hydrogen cyanide (HCN) is also known as prussic acid. Pure hydrogen cyanide is a gas usually made by mixing an acid with cyanide salts. It is more commonly found in the form of sodium, potassium, or calcium salts, which are crystalline materials. Industrial cyanide can take the form of large nuggets called “cyanide eggs.”

Color: Sodium and potassium salts of HCN are white.

Odor: It is supposed to elicit the odor of almonds. However, 40–60% of the population cannot detect the odor because of a genetic inability—a form of “odor blindness.”

Solubility: Salts of cyanide are easily dissolved in aqueous liquids. Acidic liquids would cause the release of some HCN gas.

Taste: Salts of cyanide have a bitter (alkaline) taste and can have a mild corrosive action on tissues.

Source: Fumigants, insecticides, rodenticides, metal polishes (especially silver polish), electroplating solutions, metallurgy for the extraction of gold and silver from ore, photographic processing, jewelers, and chemical labora-

Cyanide mechanism

LD = ~200-300 mg KCN, or 270 ppm HCN

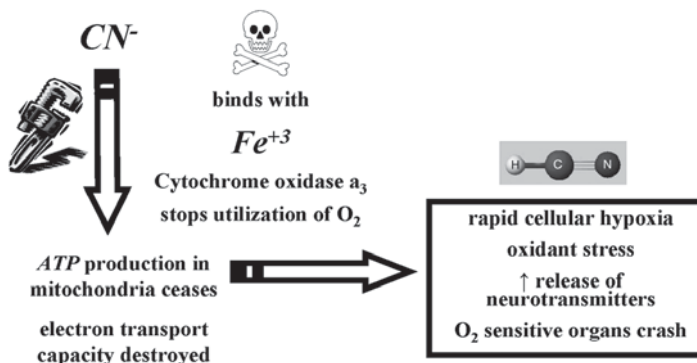


Figure App.-4

tories. Cyanide is contained as cyanogenic glycosides inside the pits and seeds of certain plants of the genus *Prunus* (e.g., cherry, peach, almond, cassava, and apricot). Cyanide can also be produced by the action of a flame on the synthetic plastic materials polyurethane and polyacrylonitrile. In addition, it is found as part of the intravenous antihypertensive drug molecule sodium nitroprusside (Nipride®). Cyanide is used in the most commonly employed method of synthesizing phencyclidine (PCP).

Lethal Dose: Acute, 270 ppm (air), 50 mg (HCN), 200–300 mg (NaCN or KCN); chronic, unknown in all forms.

How It Kills: Cyanide shuts down respiration at the cellular level by inactivating essential enzymes, resulting in metabolic asphyxiation. Critical effects are on those organs most sensitive to oxygen deprivation, the brain and heart.

(See Fig. App. 4.)

Poison Notes: When providing mouth-to-mouth respiration support to a victim, care must be taken to avoid contaminating oneself. A toxidrome (pattern of symptoms) has been established for cyanide intoxication and includes altered mental status, mydriasis (dilated pupils), abnormal respiratory pattern, low systolic blood pressure, increased heart rate, metabolic acidosis, and a large increase in blood lactate.

Victim of Cyanide

Administered: Often administered to victim in food or drink.

Symptom Onset Time Interval: Immediate (as little as 30 s). Few poisons are as rapidly lethal.

Symptoms—Acute: Headache, nausea, vomiting, difficulty breathing, and confusion. These initial symptoms are rapidly followed by seizures, coma, gasping respirations, and cardiovascular collapse.

Symptoms—Chronic: Not applicable.

Disease Confusion: Heart attack, acute asthmatic attack.

Victim Notes: An abrupt onset of profound symptoms after exposure is classic for cyanide exposure.

Detection of Cyanide

Specimens: Food, beverages, medications, blood, urine, gastric contents, and autopsy organ specimens.

Method: Colorimetric techniques.

Toxic Levels: (1)

Blood	Urine	Gastric	Other
12.4 mg/L	0.1 mg/L	Unknown	Unknown

Analysis Notes: Toxic levels of cyanide in tissues may diminish significantly after death by mechanisms that include evaporation, thiocyanate formation, and reaction with tissue components. The formation of cyanide in postmortem tissues with accumulation to toxicologically significant levels apparently from the conversion of thiocyanate to cyanide has been demonstrated but can be prevented by the addition of sodium fluoride. Blood specimens should be kept at temperatures from 20 to 4°F, to reduce losses.

Selected Cyanide Homicide Cases

- Theodosius Boughton (1781)
- Dr. Collidge (1847)
- Mrs. MacFarland (1911)
- Twigg/Elosser (1911)
- Jessie Costello (1933)

SODIUM FLUOROACETATE

Form: Looks like flour or baking soda. Sodium fluoroacetate (also known as sodium monofluoroacetate, Furatol, Ratbane 1080, or Compound 1080).

Compound “1080” mechanism

LD = ~700 mg

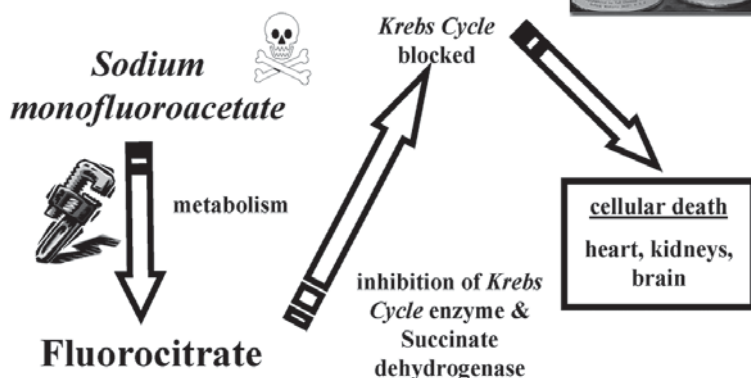


Figure App.-5

Another compound is Sodium fluoroacetamide (also known as Fluorakil, Fussol, Megarox, ancock, or Compound 1081). It is also the toxic constituent of the South African plant Giftblaar (*Dichapetalum cymosum*).

Color: White, crystalline compound.

Odor: Odorless.

Solubility: 1080 is very water soluble but has a low solubility in ethanol.

Taste: 1080 is mostly tasteless, but very dilute solutions may have a vinegar-like taste, owing to the acetate component.

Source: Pesticides, rodent poison, and predator (coyote) poison. **NOTE:** It is sold only to licensed pest-control operators and others qualified by training and experience in rodent-control procedures. It is usually mixed with black dye and added to grain baits.

Lethal Dose: acute, 2–10 mg/kg, 700 mg/150-lb victim; chronic, not applicable.

How It Kills: The salt’s metabolite, fluorocitrate, blocks normal enzyme mechanisms in the body, acting mainly on the heart and central nervous system.

(See Fig. App. 5.)

Poison Notes: As little as 1 mg of 1080 is sufficient to cause serious poisoning. This is one of the most toxic substances known, and there is no specific antidote.

Victim of Sodium Fluoroacetate

Administered: 1080 can be administered to the victim in food or drink. It can also be absorbed through broken skin.

Symptom Onset Time Interval: Symptoms are delayed while the body converts the compound into the more toxic metabolite. Compound 1080: from 30 min to several hours; Compound 1081: slower onset of symptoms.

Symptoms—Acute: Nausea, vomiting, diarrhea, agitation, confusion, seizures, lethargy, coma, respiratory arrest, and cardiac arrhythmias.

Symptoms—Chronic: Not applicable.

Disease Confusion: Gastroenteritis, viral infection, heart attack.

Victim Notes: None.

Detection of Sodium Fluoroacetate

Specimens: Blood, urine, and gastric contents.

Method: Specific gas chromatographic procedures for the identification of fluoroacetate in biological specimens have relied on flame ionization, electron capture, or mass spectrometric detection of a derivative. There has been a report for a method for high-pressure liquid chromatography on gastric contents.

Toxic Levels: (1)

Blood	Urine	Gastric	Other
Unknown	65 mg/L	12 mg/L	Liver: 58 mg/kg

Analysis Notes: None

Selected Sodium Fluoroacetate Homicide Cases

No cases on record.

STRYCHNINE

Form: Strychnine is an alkaloidal plant compound obtained from the tree *Strychnos nux-vomica*. The seeds are gray-green discs (1-in. in diameter, 0.25-in. thick at the rim) with a central depression and a satin-like appearance. Pure strychnine alkaloid is a white powder.

Color: White, crystalline powder.

Odor: None that is characteristic.

Solubility: Soluble in aqueous solutions.

Taste: Strychnine is extremely bitter, with a taste that is detectable at a dilution of 1:100,000. Strychnine could be administered in alcohol if the victim is accustomed to bitter drinks (e.g., tonic water). Strychnine can also be introduced into foods that normally have a sour or bitter taste.

Strychnine mechanism

LD = ~30-100 mg

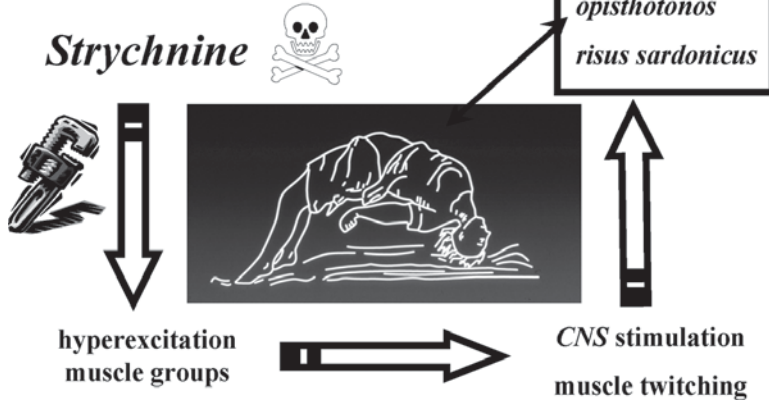


Figure App-6

Source: Rodenticides (concentrations >0.5% are currently distributed only to licensed exterminators). Strychnine is sometimes found as an adulterant in illicit drugs. At one time, strychnine was sold over the counter as an ingredient in a variety of stimulant tonics and laxatives.

Lethal Dose: Acute, 5–8 mg/kg, 30–300 mg (oral); chronic, unknown.

How It Kills: Respiratory arrest.

Poison Notes: None.

(See Fig. App. 6.)

Victim of Strychnine

Administered: Can be given in food, beverages, or medications.

Symptom Onset Time Interval: 15–30 min (oral route), 5 min (iv or nasal route).

Symptoms—Acute: Muscle stiffness and painful cramps, which precede generalized muscle contractions. The victim's body may take the form of an arch (with only the head and heels touching the floor)—this is called an “opisthotonic” convulsion. The face may be drawn into a forced smile or sardonic grin called “risus sardonicus.” The muscle contractions (spasms) are intermittent and can be easily triggered by emotional or physical stimuli (sound, touch, light). Death is usually owing to respiratory arrest.

Symptoms—Chronic: Not applicable.

Disease Confusion: Grand mal seizures, tetanus.

Victim Notes: Strychnine does not cause true seizures, and the victim is awake and painfully aware of the contractions.

Detection of Strychnine

Specimens: Food, beverages, medications, blood, urine, gastric contents, and autopsy organ specimens.

Method: Colorimetry, ultraviolet spectrophotometry, or GC.

Toxic Levels: (1)

Blood	Urine	Gastric	Other
21 µg/mL	9.1 µg/mL	61 µg/mL	Unknown

Analysis Notes: None

Selected Strychnine Homicide Cases

- Christina Edmunds (1871)
- Thomas Cream (1892)
- Jean-Pierre Vacquier (1924)
- Ethel Major (1934)
- Floyd Horton (1937)
- Patsy Wright (1987)

THALLIUM

Form: Thallium in elemental form is a soft metal. Thallium can also exist as metallic salts (acetate, carbonate, chloride, sulfate, and so on).

Color: Thallium salts are white crystalline powders.

Odor: Odorless.

Solubility: Thallium salts are readily soluble in water.

Taste: Tasteless.

Source: Thallium can be used as a rodenticide and an insecticide, but the use is restricted to licensed applicators (public use prohibited since 1965). Thallium salts are widely used in industry (manufacture of optical lenses, photoelectric cells, costume jewelry) and chemical analyses. It has no known biological function in the body.

Lethal Dose: Acute, 1 g, 12–15 mg/kg; chronic, unknown.

How It Kills: Thallium causes membrane depolarization by acting as a substitute for potassium in the sodium–potassium–adenosine triphosphatase pump. Like arsenic, it binds to enzymes containing (—SH) sulfhydryl groups.

(See Fig. App. 7.)

Thallium mechanism

LD = ~1 gm

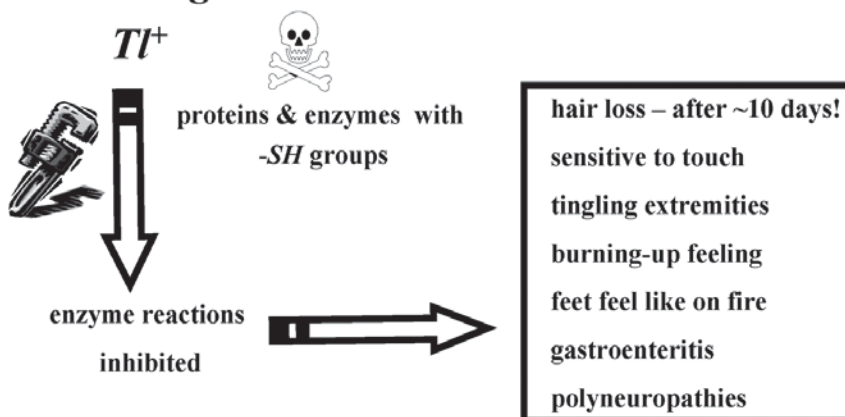


Figure App-7

Poison Notes: Thallium is one of the most lethal poisons and produces one of the highest incidences of long-term sequelae (mainly neurological).

Victim of Thallium

Administered: Can be given in food, drink, or medications.

Symptom Onset Time Interval: Thallium is rapidly absorbed through the skin, and mucous membranes of the mouth and GI tract. GI symptoms appear after a latent period of usually 12–24 h.

Symptoms—Acute: Abdominal pain, anorexia, nausea, vomiting, diarrhea, delirium, depressed respirations, seizure, coma, and death.

Symptoms—Chronic: Muscle weakness, atrophy, tingling and numbness in the extremities, peripheral neuropathy, painful legs, a feeling that feet are on fire, a burning feeling in the body, sensitivity to touch, Aldrich-Mees lines (3 to 4 wk post), and hair loss (a classic symptom; may appear after 2–4 wk).

Disease Confusion: Viral disease, Guillain-Barré syndrome.

Victim Notes: The victim is often misdiagnosed.

Detection of Thallium

Specimens: Food, beverages, medications, saliva, blood, urine, gastric contents, hair, and autopsy organ specimens.

Method: Flameless atomic absorption.

Toxic Levels:

Blood	Urine	Gastric	Other
0.5–11 mg/L	1.7–11 mg/L	Unknown	Unknown

Analysis Notes: Because thallium is not a normal body constituent, any concentration is considered significant. Blood and hair thallium levels are not reliable measures of exposure.

Selected Thallium Homicide Cases

- Martha Lowenstein Marek (1932)
- Caroline Grills (1947)
- Graham Frederick Young (1971)
- George Hanei (1976)
- George Trepal (1988)

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Bibliographies

This extensive bibliographic collection on forensic toxicology, poisoning murders, and poisons in general represents the results of more than 35 years of my research on the subject. The citations have been drawn from an in-depth review of the international literature. These bibliographies are included for two major purposes: (1) to serve as a catalog of available literature for those individuals wishing to study the subject of poisons and murder in even greater depth, and (2) to provide for criminal investigators a checklist of things to search for in the environment of the homicidal poisoner.

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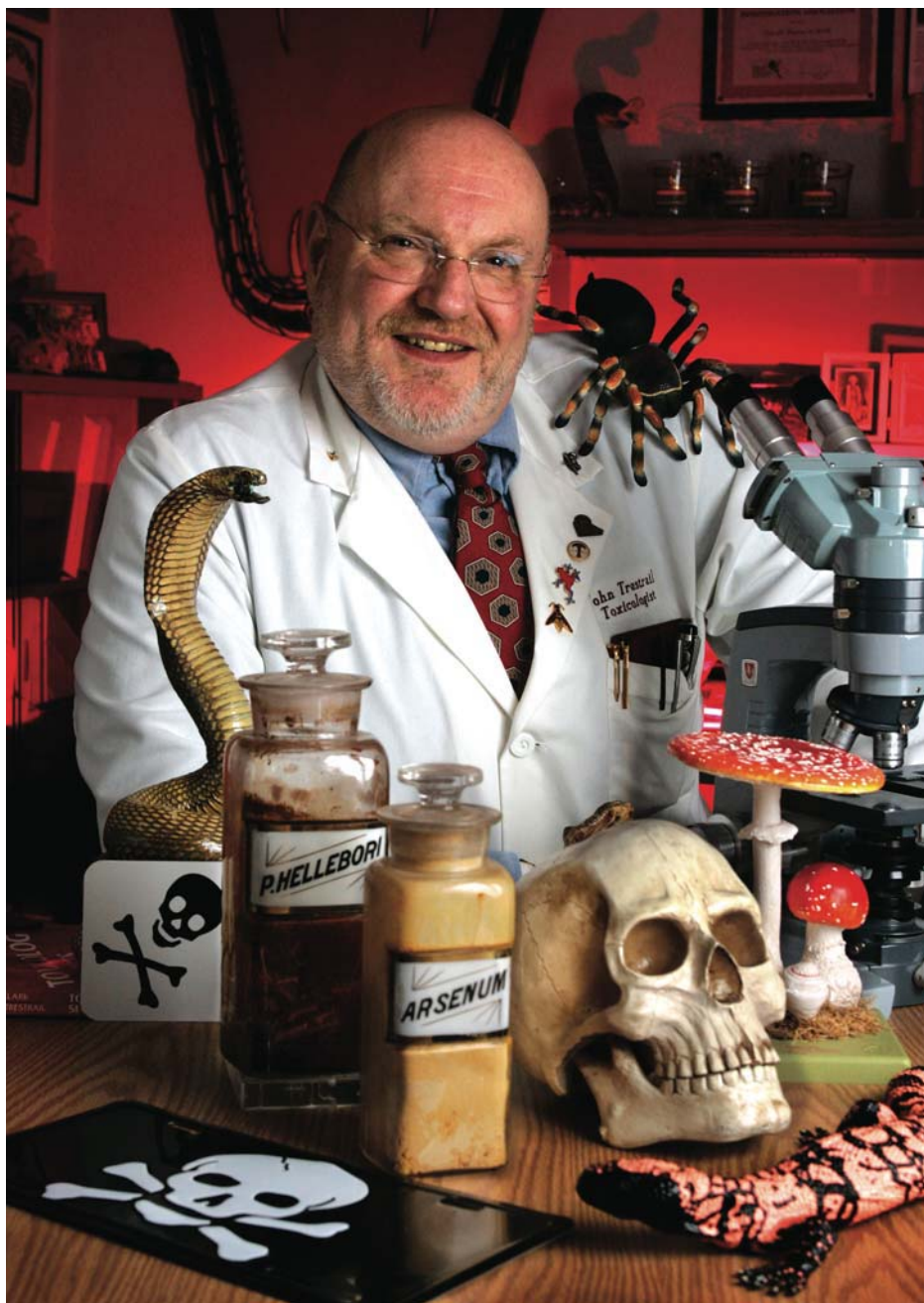


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