Exposure to Toxic Substances

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Exposure to Toxic Substances

Irving Sunshine

WITH LEGAL ANNOTATIONS BY OLIVER SCHROEDER, JR.*

DEFINITION

A toxic substance, for the purpose of this discussion, is defined as any chemical absorbed by the body that produces a deleterious effect on an individual. This is a broad but apt concept. A similar definition occurs in many state laws.

FACTORS GOVERNING A DRUG'S ACTION

Before individual poisons and their effects are discussed, the factors governing a drug's action should be clarified.

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There must first be some contact with the chemical substance. Inhalation of noxious gases, mists or dusts, particularly in industry, is one type of exposure. Many home accidents are caused by the accidental ingestion of harmful materials. Industrial exposures due to ingestion also occur, particularly by those employees who eat or smoke while working in a contaminated atmosphere. A third type of exposure, parenteral, either intravenously, intramuscularly or subcutaneously, is a very common route of administration of drugs for therapeutic purposes. Occasionally civil litigation may result if the wrong drug is injected. Absorption of a harmful chemical agent through the skin represents still another mode of contact which causes many industrial problems.

Before a drug can act, it must be absorbed by the body. The rate of absorption is influenced by the physical form of the drug, its solubility and its concentration. Generally a gas will be absorbed more rapidly than either a liquid or solid, and a liquid, in turn, is better absorbed than a solid. Whatever its form, unless the chemical agent is soluble, it will not be absorbed by the body but harmlessly excreted. The concentration of a drug also influences its action. A 12 ounce bottle of beer contains as much ethyl alcohol as one ounce of whiskey. Under identical conditions an individual will tolerate the bottle of beer better than an ounce of whiskey. The same

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individual will tolerate either drink better after a hearty meal than on an empty stomach, since the food acts as a diluent and slows the absorption rate, and consequently, the deleterious effect of the alcohol.

The mode of action of a drug is also influenced by many subjective factors. A drug may have a given reaction on one person, and, occasionally, an entirely different reaction on another. Most people take aspirin with desirable therapeutic results. A few individuals, on the other hand, cannot "tolerate" this drug and have an undesirable reaction to it. The antibiotics are a more familiar example; one person "blows up" after an injection, but many others have no untoward effects. Thus individual allergy or idiosyncrasy to a drug is recognized. A person's age may also influence the action of some drugs. Youngsters, for example, cannot tolerate opiates as well as their parents. As a result of chronic exposure some people become accustomed to a given chemical such as nicotine, ethyl alcohol, arsenic or morphine to mention a few. They tolerate amounts that are hazardous to others. These people are not addicts; they merely require a larger dose to accomplish a therapeutic effect because their body has acclimated itself to the chemical. Finally, one's physical condition also influences reactions to drugs. A person with renal disease and concomitant faulty urinary excretion cannot tolerate drugs that would be relatively harmless to one who has good kidney function. An impaired liver, which decreases the ability to metabolize a drug, will make an individual more susceptible to a toxic reaction.

The following case illustrates an accident involving a patient with impaired liver function that might be the basis for civil litigation. A woman was admitted to the hospital with toxemia of pregnancy just prior to her term. Despite the fact that clinical laboratory tests indicated she had some impairment of liver function phenoarbital was prescribed for the patient. After several days she became comatose, due to barbiturate intoxication, during which period she delivered a stillborn baby. An autopsy was performed on the baby. The cause of death was determined to be barbiturate intoxication. The mother survived the incident and recovered.

The amount of drug that is absorbed in a given period of time also influences its mode of action. The ingestion of several tablets of bichloride of mercury will cause acute poisoning. When a large amount is taken, nausea and vomiting may occur, thus eliminating the greater portion of the poison. The absorbed residue may still be enough to cause severe damage to the kidney with resulting shutdown of urinary excretion. On the other hand, workers chronically exposed to mercury vapor for a prolonged period may evince a different set of symptoms, some gastro-intestinal, but mostly neurological.

Thus, an acute exposure may have one effect, while a prolonged exposure to much lower concentrations may produce chronic symptoms that are en-
tirely different from the acute effects. Civil litigation is generally concerned with chronic exposures, which usually result in an occupational disease. The extent of a chronic exposure is gauged by the maximum allowable concentration. This is the highest concentration of a given substance to which an individual can be exposed, with no adverse effects, throughout a working day for an indefinite period of time. Many agencies concerned with industrial hygiene have evaluated all the available data carefully and have promulgated a set of standard maximum allowable concentrations. These "Macs" are subject to periodic review as new facts are ascertained. Many states include "Macs" in their sanitary codes. It should be noted, however, that "Macs" serve only as guides. Each case must be decided on its own merits because each individual will react in his own peculiar way to a given exposure.

The duration of an exposure is significant. If the "Mac" is 100 ppm. (parts per million) for an eight hour day, it does not necessarily follow that a safe exposure could be 800 ppm. for one hour. The probability is that an acute attack would result from the latter exposure.

Enforcement of "Macs" is usually one of the functions of the department of health. This group seldom resorts to the letter of the law, but usually acts in an advisory capacity to secure voluntary cooperation from industry. In Ohio, for example, the Division of Industrial Hygiene in the Department of Health will evaluate the extent of an industrial hazard at the request of management, labor, or any other interested party, with no charge for the service. A similar agency is maintained by the City of Cleveland and will perform such services locally.

The excretion and metabolism of chemical agents also influences their action. The body has several means of eliminating foreign material. Insoluble substances will generally be eliminated through the colon and rectum as feces; soluble waste is eliminated through the kidneys, the sweat glands, the salivary glands, and in some cases, through the lungs (as expired gases). Soluble material is absorbed by the body and undergoes several changes before it is excreted. These changes are called metabolism. They may exert a therapeutical effect or a deleterious effect—poisoning. To avoid poisoning, steps must be taken to remove the source of the exposure or to alter the drug's metabolism. This may be more or less difficult. If, when a person is exposed to carbon monoxide, he is removed from this harmful atmosphere and oxygen is administered, the deleterious reaction is reversed. Arsenic attacks various enzyme systems, but if BAL (British

2 OHIO REV. CODE § 3701.34.
Anti Lewisite) is promptly administered, the arsenic metabolism is so altered that a toxic reaction does not occur.

**TYPES OF EXPOSURE**

Non-occupational exposure to toxic substances may come about through suicidal attempts, or through improper use of such household items as dry-cleaning solvents, paint solvents, insecticides, herbicides, disinfectants and drugs. Faulty home gas appliances may release carbon monoxide and cause an accident. Noxious residues resulting from fumigation of homes or ships are also dangerous if proper precautions are disregarded. The person who takes the contents of a misfilled prescription may be injured thereby and bring a civil suit against the druggist. This group of non-occupational exposures usually is typified by an acute reaction to a chemical agent.

Industrial exposures may result in an occupational disease. A clear and broad definition of an occupational disease given by one Court is, "a disease contracted in the usual and ordinary course of events which from the common experience of humanity, is known to be incident to a particular employment." An ailment does not become an occupational disease merely because it is contracted on the employer's premises. It must be one that is commonly recognized as inherent to a harmful exposure in a given industry.

It is important to note that an industrial hazard does not necessarily exist because an exposure is present. Only after the exposure is quantitatively determined can one evaluate whether or not it is a hazard.

An important distinction exists between accidental traumatic injuries and occupational disease injuries. The former are easily discovered, occur at a given place of employment and leave no question as to which employer is involved. An occupational disease injury may develop slowly, unknown to both employer and employee until a disability occurs. The exposure may have had its beginning in one establishment and then been aggravated by similar employment elsewhere. In Ohio certain occupational diseases are compensable when contracted by an employee during the course of employment in which such employee was engaged at any time with 12 months previous to his date of disability.

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3. Industrial Com. v. Roth, 98 Ohio St. 34, 38, 120 N.E. 172, 173 (1918).
4. Ohio Rev. Code § 4123.68 lists these diseases, among others:
   1. Lead poisoning: Any industrial process involving the use of lead or its preparation or compounds.
   2. Mercury poisoning: Any industrial process involving the use of mercury or its preparation or compounds.
Once an exposure, occupational or non-occupation, is suspected, it is essential to document the case. An immediate investigation to ascertain all the facts is very important. If an occupational disease is suspected, a survey of the hazard is helpful. Generally this may be obtained through the cooperation of a public office like the Department of Industrial Hygiene in Ohio. Industry, in Ohio, cannot refuse to permit this survey, which may be made at the request of any interested party. However, even if this survey indicates a harmful exposure exists, additional information is essential. A history of the exposure must be elucidated from the victim. This should

3. Phosphorus poisoning: Any industrial process involving the use of phosphorus or its preparations or compounds.
4. Arsenic poisoning: Any industrial process involving the use of arsenic or its preparations or compounds.
5. Poisoning by benzol or by nitro and amido-derivatives of benzol (dinitrobenzol, anilin and others): Any industrial process involving the use of benzol or nitro- or amido-derivatives compounds.
6. Poisoning by gasoline, benzine, naphtha, or other volatile petroleum products: Any industrial process involving the use of gasoline, benzine, naphtha, or other volatile petroleum products.
7. Poisoning by wood alcohol: Any industrial process involving the use of wood alcohol or its preparations.
8. Poisoning by carbon bisulphide: Any industrial process involving the use of carbon bisulphide or its preparations or compounds.
9. Infection or inflammation of the skin on contact surfaces due to oils, cutting compounds or lubricants, dust, liquids, fumes, gases or vapors: Any industrial process involving the handling or use of oils, cutting compounds or lubricants, involving contact with dust, liquids, fumes, gases or vapors.
10. Epithelioma cancer or ulceration of the skin or of the corneal surface of the eye due to carbon, pitch, tar or tarry compounds: Handling or industrial use of carbon, pitch or tarry compounds.
11. Carbon dioxide poisoning: Any process involving the evolution or resulting in the escape of carbon dioxide.
12. Brass or zinc poisoning: Any process involving the manufacture, founding or refining of brass or the melting or smelting of zinc.
13. Manganese dioxide poisoning: Any process involving the grinding or milling of manganese dioxide dust.
15. Chrome ulceration of the skin or nasal passages: Any industrial process involving the use of or direct contact with chromic acid or bichromates of ammonium, potassium or sodium or their preparations.
16. Potassium cyanide poisoning: Any industrial process involving the use of or direct contact with potassium cyanide.
17. Silicosis. [Silicosis shall mean a disease of the lungs caused by breathing silica dust (silicon dioxide) producing fibrous nodules, distributed through the lungs and demonstrated by X-ray examination or by autopsy.]


EXPOSURE TO TOXIC SUBSTANCES

include such details as the materials he handled, the use or non-use of protective devices, company policy regarding these safety devices, the adequacy of the ventilation system, previous occupational history, the duration of the exposure, and the physical manifestations of the detrimental effects of the exposure. A physician's clinical report resulting from a complete medical examination is necessary as soon after the discovery of the exposure as possible. Concurrent with this examination, all laboratory tests that may pertain to a given exposure should be ordered. Positive tests, of course, are significant; but negative tests must be properly evaluated. If improper samples were taken, or if the period of time between the end of the exposure and the time the samples were taken is too long, the laboratory results may be negative even though a real exposure was originally involved. Some substances are excreted or metabolized in 24-48 hours, whereas others may be detected several days after an exposure. Carbon monoxide, benzene and the chlorinated hydrocarbons are in the former category, whereas lead and arsenic typify the latter.

If samples are sent to a laboratory, it is important to follow several simple rules:

1. Take the sample in a clean container, preferably at the site of the exposure.
2. Seal the container and label it properly with the name of the victim, the time of the incident, and the laboratory tests requested.
3. Deliver or send the sealed container to the laboratory promptly.
4. Be certain the laboratory has qualified personnel to do the required test.

If death results from an exposure, an autopsy is a necessity to establish the cause of death.

There are no reliable statistics available on the incidence of non-fatal occupational or non-occupational poisonings. Many state agencies require that occupational disease be reported. However, these laws are seldom enforced and are more honored in the breach than in the observance. Many cases are not reported because doctors may not recognize that they originated as occupational diseases. Hospital record rooms contain the only available information about more serious non-occupational poisoning incidents, but these facts are not commonly available.

Compensation records give some data on the incidence of hazardous exposures, particularly for cases of longer than three to seven days duration or when death supervened. The Division of Industrial Hygiene of the United States Public Health Service has collected statistics on occupational

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*Trasko, The Work of State and Local Industrial Hygiene Agencies, 64 Pub. Health Reports, 482 (1949).*
diseases from industrial hygiene units in eight states for the 1947 calendar or 1948 fiscal year.

Table 1  Occupational Disease Reports Received by Industrial Hygiene Agencies in 18 States

<table>
<thead>
<tr>
<th>Causes</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poisoning due to</td>
<td></td>
</tr>
<tr>
<td>Metals (lead 257)</td>
<td>316</td>
</tr>
<tr>
<td>Solvents</td>
<td>48</td>
</tr>
<tr>
<td>Gases</td>
<td>134</td>
</tr>
<tr>
<td>Dusts</td>
<td>981</td>
</tr>
<tr>
<td>Pneumonconiosis (not specified)</td>
<td>274</td>
</tr>
<tr>
<td>Systemic poisoning (not specified)</td>
<td>1228</td>
</tr>
<tr>
<td>Chemical poisoning (not specified)</td>
<td>186</td>
</tr>
<tr>
<td></td>
<td>3,167</td>
</tr>
<tr>
<td>Dermatitis</td>
<td>18,288</td>
</tr>
<tr>
<td>Conjunctivitis</td>
<td>4,893</td>
</tr>
<tr>
<td>Pulmonary &amp; bronchial affections</td>
<td>1,616</td>
</tr>
<tr>
<td>Inflammatory Conditions due to repeated</td>
<td></td>
</tr>
<tr>
<td>motion, pressure or shock</td>
<td>2,380</td>
</tr>
<tr>
<td>Heat exhaustion</td>
<td>112</td>
</tr>
<tr>
<td>Nasal ulceration</td>
<td>137</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>719</td>
</tr>
<tr>
<td></td>
<td>31,312</td>
</tr>
</tbody>
</table>

These are the only available published data. Obviously, they are not complete. Those occupational diseases due to poisoning from dusts, systemic poisons, gases, metals and solvents will be discussed in this article.

Classification of Poisons

Customarily, poisons have been classified in many different ways, no one of which is really satisfactory to every one concerned with this problem. Arbitrarily, the toxic substances can be classified by the effects they produce. Thus we recognize irritants, asphyxiants, anesthetics, hypnotics, narcotics and protoplasmic poisons.

The irritants produce inflammation generally at the site of their entry to the body. Gaseous irritants such as chlorine, bromine, fluorine, hydrogen chloride, sulfur trioxide, chromic acid and formaldehyde affect the upper respiratory tract and/or the lungs. Oral irritants such as alkalis, acids, phenol, lysol, cresol, and bichloride of mercury will cause injury throughout the alimentary canal with concurrent vomiting and diarrhea. This latter group of irritants are generally called corrosives since they cause corrosion or destruction of tissue.
Asphyxiants produce harmful effects by improper oxygenation of tissues. This result may be due to displacement of the normal atmosphere by contaminants such as carbon dioxide, nitrogen, hydrocarbons, etc., which decrease the amount of available oxygen that reaches the lungs below the safe limit. A typical example is the case of a night watchman who fell in the doorway of a dry-ice (carbon dioxide) pit, struck his head and became unconscious. Unable to move, he died as a result of simple asphyxia while lying in that atmosphere. Chemical asphyxia results from the chemical reaction of a poison in the body which prevents the blood from transporting oxygen or prevents the tissues from accepting the vital oxygen even when the blood is fully oxygenated. Carbon monoxide, chemically, prevents hemoglobin from combining with oxygen. Hydrogen cyanide inhibits tissue oxidation by destroying those enzyme systems essential for oxidation. Nitrites, aniline and organic nitro compounds form methemoglobin, which reduces the oxygen-carrying capacity of the blood. The methemoglobin forming compounds cause a greyish-blue cast of the skin (cyanosis), which is typical of this type of anoxia.

Anesthetics cause paralysis of the central nervous system, as evidenced by loss of consciousness, of pain and of voluntary movements. In addition to the usual anesthetics, like ether, chloroform and cyclopropane, this group includes many organic hydrocarbons that are used as solvents or as natural gases for cooking and heating. Many home accidents come about from the use of these solvents for painting or cleaning without proper ventilation. In these latter cases death is usually due to asphyxia due to displacement of oxygen by solvent fumes rather than to the chemical effects of the solvent.

The hypnotics and narcotics, used to induce sleep, include the barbiturates, the opiates, chloral and paraldehyde. Their indiscriminate use has resulted in many accidental deaths. People under the influence of these drugs may be more prone to accidental death or natural disease. A driver under their influence has poor reflex actions and may thus precipitate a collision. Some people, under the influence of one of the hypnotics, may fall asleep outdoors and develop a serious pneumonia before awakening—if they awake.

A protoplasmic poison is one which destroys living tissue. The corrosives, mentioned previously, are essentially protoplasmic poisons. In general, the metals and their compounds are also in this group, as are the chlorinated hydrocarbons and phosphorus.

Other poisonous substances that should be mentioned, but which do not easily fall into one of the above classifications, are those that affect the blood-forming mechanism of the body (benzene) and those that affect the optic nerve (methyl alcohol and carbon bisulphide).
The classification system just described is open to criticism since a given poison seldom affects one organ only or has only one primary physiological effect on the body. Effects are usually multiple. The sequelae of acute exposure may refer to one organ and that of chronic exposure to another. Inhalation of a high concentration of benzene vapor produces intoxication and depression of the central nervous system, whereas chronic exposure leads to injury of the blood-forming organs.

**NOXIOUS AGENTS**

Space precludes a detailed discussion of all poisons. Consequently only typical examples of each of the classifications will be discussed.

The average individual thinks about what he eats and drinks; but he is seldom concerned about the air he breathes. Today—particularly in highly industrial areas—everyone is exposed to harmful gases. Some fundamental concepts will help to understand the mechanism of a gas' action in the body.

The mechanics of respiration are more easily understood if one is familiar with the anatomy involved. Air is inhaled and passes through the nose to the pharynx, which connects the nasal passageway with the trachea. The pharynx is a common pathway for both air and food. Between the pharynx and the trachea is the larynx, which contain a valve-like structure, the epiglottis. The latter is closed during the act of swallowing, thus pre-

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venting food from entering the trachea and the air passageway. The trachea is a tube, measuring one to two centimeters in diameter, which allows the air to reach the bronchi which lead to the right and left lobes of the lungs, then to the bronchioles and the terminal bronchioles. These tubes represent the upper respiratory tract. The lung unit or primary lobule, which is primarily responsible for respiration, consists of the respiratory bronchioles, the alveolar ducts, the alveolar sacs and the pulmonary alveoli.

The alveoli contain a large network of fine capillaries, and it is here that the diffusion of gases from alveolar air to blood and from blood to alveolar air occurs. The alveolar walls are so thin and the surface of the capillaries so large (eight square feet in the lungs of an average man) that diffusion of gases proceeds rapidly. Equilibrium between the gases in alveolar air and blood is established very quickly.

The normal function of respiration is to supply oxygen through the alveolar walls to the blood for distribution to the tissues and to remove from the blood carbon dioxide resulting from oxidation in the body. This oxidation is essential to life. The body is a very efficient chemical factory that converts chemical energy into mechanical work and heat. The tissues of the body can be compared to a furnace that burns everything to carbon dioxide very slowly, thus producing energy. This energy is stored by the tissues and is released as the body demands it.

The anesthetics and irritants are the gases most frequently encountered. The anesthetics seldom are a problem in civil litigation and therefore no detailed discussion is necessary. The irritants differ widely in chemical and physical properties. However, they all act in a similar fashion. The damage they cause is seldom due to chemical action, but is primarily due to the physiological response to their presence. Irritants, because of their different solubilities, act differently on various parts of the body. Extremely soluble irritants, such as ammonia, formaldehyde, sulphur trioxide or hydro-
gen chloride, tend to act on the first moist surface they contact. The upper respiratory tract is therefore affected. Conversely, irritants such as phosgene and nitric oxide are slightly soluble and therefore affect the lungs primarily. In both instances the resulting injury is the same—inflammation occurs at the site of the irritation. Mild irritation dilates the blood vessels, causing redness, and increases the flow of mucus. In more severe cases plasma exudes from the engorged blood vessels and fills the respiratory passage. It may cause swelling and separation of tissues which may slough off and leave oozing surfaces. Normal barriers to harmful bacteria are then removed and infection may set in, resulting ultimately in pneumonia. There is an additional complication when inflammation occurs in the lungs. The alveoli and the alveolar sacs fill with the exudate. A person then literally drowns in his own fluids since normal gaseous exchange is no longer possible. This inflammation may also put a severe strain on the heart since the normal flow of blood through the lung is obstructed. Heart damage, therefore, may also be a complicating factor after an irritant exposure. Thus, in all exposures to irritant gases the reaction is similar, but the symptoms that result are different, depending on the location in the respiratory tract of the irritant action.

Exposure to irritants may come from many industrial sources that use the irritant chemicals in their industrial processes. Other exposure to irritants may result from the accidental combustion of the nitrogenous material in x-ray films, as occurred after the disastrous Cleveland Clinic fire. The chlorinated hydrocarbons from fire extinguishers, when sprayed over hot surfaces, may decompose to form phosgene. Sulphur dioxide is used as a refrigerant and occasionally escapes and causes damage.

The question of whether or not exposure to irritating gases predisposes an individual to tuberculosis is often raised. The available scientific information indicates this is not so. The law, however, has sometimes recognized the relationship between exposure and tuberculosis.11

Asphyxiants have been previously discussed. They cause damage to tissues because of oxygen deficiency. If, after an exposure, the resulting anoxia is relieved promptly, the sequelae are relatively innocuous. If, however, prolonged exposure occurs, from which recovery is slowly made, then organic changes of a degenerative type may develop in the nervous tissues with concomitant disability. These changes may include paralysis, amnesias and other nervous derangements. This is the most serious consequence of prolonged anoxia other than death itself.

Carbon monoxide,12 the most insidious common poison, is responsible

12 See Drinker, Carbon Monoxide Asphyxia (1938); Beck, Chronic Carbon Monoxide Anoxemia: Clinical Syndromes, 30 South Med. J. 824 (1937); Beck, Clinical
for about fifty per cent of all poisoning fatalities. Because it is so accessible it is a favorite means of suicide. Many home and industrial accidental

**Chemical Agents Found in Fatalities**

**Cuyahoga County**

(797 Cases, 1943 through 1953)

<table>
<thead>
<tr>
<th>Chemical Agent</th>
<th>Number</th>
<th>Percent of All Poisoning</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbon Monoxide</td>
<td>374</td>
<td>47</td>
</tr>
<tr>
<td>Barbiturates</td>
<td>156</td>
<td>20</td>
</tr>
<tr>
<td>Heavy Metals</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Mercury, Arsenic, Lead)</td>
<td>81</td>
<td>10</td>
</tr>
<tr>
<td>Methyl Alcohol</td>
<td>27</td>
<td>3.4</td>
</tr>
<tr>
<td>Alkaloids</td>
<td>18</td>
<td>2.2</td>
</tr>
<tr>
<td>Chlorinated Hydrocarbons</td>
<td>16</td>
<td>2.0</td>
</tr>
<tr>
<td>Salicylates</td>
<td>14</td>
<td>1.7</td>
</tr>
<tr>
<td>Phenol-Lysol</td>
<td>12</td>
<td>1.5</td>
</tr>
<tr>
<td>Cyanide</td>
<td>12</td>
<td>1.5</td>
</tr>
<tr>
<td>Fluorides</td>
<td>11</td>
<td>1.3</td>
</tr>
<tr>
<td>Phosphorous</td>
<td>10</td>
<td>1.2</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>66</td>
<td>8.2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>797</td>
<td><strong>100%</strong></td>
</tr>
</tbody>
</table>

Deaths are due to carbon monoxide. Whenever combustion occurs, either in a heating unit, a cooking range, an automobile engine, or an industrial process, carbon monoxide is released. Since it is odorless and colorless, it is not detectable by the exposed individual. Carbon monoxide is not metabolized. It has an affinity for hemoglobin 210 times that of oxygen and is therefore preferentially absorbed by the blood, despite the simultaneous presence of oxygen. Unless the source of the carbon monoxide hazard is eliminated, it will tend to accumulate in the blood of an exposed individual. If the carbon monoxide source is removed, or if the patient is removed from the contaminated atmosphere, the carbon monoxide will be excreted.

The toxicity of carbon monoxide is a function of the time of exposure and the concentration of carbon monoxide. The formula $T \times C = K$ expresses this relationship. ($T =$ time in hours and $C =$ concentration of the gas expressed in parts per million [ppm]. One per cent of a gas equals 10,000 ppm.)


If $K = 300$ there is no perceptible effect.  
If $K = 600$ there is just perceptible effect.  
if $K = 900$ there is headache and nausea.  
If $K = 1500$ it is dangerous.

The toxicity of carbon monoxide is illustrated by the man who enters his one-car garage to "warm up" the auto on a cold winter day. Motor exhaust contains approximately seven per cent carbon monoxide. If the garage door is closed "to keep the heat in," a concentration of carbon monoxide will accumulate in five minutes that is sufficient to cause death in another fifteen. The toxic effect will vary with the degree of activity: the less active a person is, the more carbon monoxide he may tolerate since his oxygen demand will be lower. The symptoms evolved in a carbon monoxide incident are a function of the carboxyhemoglobin saturation of the victim's blood. Headaches, irritability, and fatigue from moderate exertion characterize carboxyhemoglobin saturation up to 25%. The characteristic irritability ascribed to chain smokers, traffic policemen and taxicab drivers may be due, in part, to carbon monoxide exposure. Fatalities have been reported from thirty per cent to eighty per cent saturation, usually occurring at values of forty-five per cent or higher.

When laboratory data are required to corroborate an exposure to carbon monoxide, the sample must be taken at the site of exposure, preferably during the time of exposure. Sixty per cent of the original carboxyhemoglobin concentration is lost in the first hour after removal from the exposure. Consequently, if an exposed person leaves the site of exposure and goes to a doctor's office to give a sample of blood for analysis, the carboxyhemoglobin content will decrease markedly and may no longer be detectable. Chronic exposures to carbon monoxide cannot exist, for as soon as one leaves the exposure, the carbon monoxide is effectively removed from the body by normal breathing. The most serious, but relatively infrequent, sequelae to carbon monoxide poisoning result from prolonged exposure to high concentration that leads to unconsciousness. The injuries that result are due to anoxia and may cause permanent degenerative changes to the brain and nervous tissue and concomitant disability if the period of unconsciousness is lengthy.

Another chemical asphyxiant, hydrogen cyanide, is frequently used as a fumigant. Its salts are used by jewelers and in the electroplating industry. Cyanides are fast-acting fatal chemical agents, producing chemical asphyxia within minutes after exposure. Their mechanism of action is interesting.

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Completely oxygenated blood reaches the tissues, but they cannot utilize this oxygen because the cyanide ion attacks the enzyme system that effects tissue oxygenation. Asphyxia results despite the availability of oxygen. Suicides account for most deaths due to cyanide. Accidental cyanide poisoning may result from fumigation residues which saturate upholstered furniture, bedding and draperies. If these articles are not properly aerated before use to remove the residual hydrogen cyanide gas, then persons prematurely entering the fumigated premises, unaware of the hazards, may be injured.

Occupational disease brought about by exposure to metals is much more common than available statistics indicate. Throughout industry, exposures exist, and some are hazardous. If a serious exposure is discovered by periodic medical and chemical examinations, the exposed individual is treated and then placed in a less hazardous position. There are, however, many small industrial plants that cannot afford the expense of a competent industrial physician. It is not unusual to find a periodic labor turnover in these small plants. Some of the employees develop an occupational disease that is seldom recognized or treated. Quitting the job removes them from the exposure and they obtain relief. However, this is still costly to the industry because it must constantly obtain new personnel.

Non-occupational exposures to metals and their compounds are relatively few. There are some exposures to lead — particularly in children who develop a habit of eating dried paint residues. Suicides resort to mercury bichloride, arsenic compounds and fluorides. The metals represent about ten to fifteen per cent of all fatalities due to poisons. Some metals are not harmful, even if ingested or inhaled. The hazard may be due to the concomitant solvent fumes. Occupational disease usually results from the accumulation of a metal in the body after chronic exposure. Seldom does an acute exposure develop in industry. Industrial exposures generally result in a dermatitis, a respiratory irritation, a gastro-intestinal or a neurological disturbance.

Of all metallic substances, lead is one of the more common industrial hazards. An exposure may occur wherever lead or lead alloys are processed or wherever lead or its compounds are "burned." Other industrial exposures to lead occur in the storage battery industry, in the paint industry, and in the preparation and handling of lead tetra-ethyl. Non-occupational hazards of lead to children have already been mentioned. Lead is usually introduced...
into the body by inhalation of vapors, occasionally by absorption through the skin as in the case of handling concentrated lead tetra-ethyl, and infrequently by ingestion. Good sanitation prevents the last. Lead tetra-ethyl only poses a problem in its manufacture. Gasoline handlers dispensing dilute solutions of this substance have no untoward reaction to it.

Inhalation of lead dusts is a frequent industrial exposure. The exposed person's blood obtains the lead from the inhaled dust passing through the lungs and transports it through the body. Some is excreted in the urine, and feces and some is retained, primarily in the long bones. One of the more common toxic effects of lead in industrial workers is so-called "lead colic," complicated by constipation and nausea; the blood may be affected and anemia and other blood changes may occur. More severe exposure may affect the nervous system, including "paralysis" of some muscles, so-called "wrist drop" and, in very severe cases, the brain may be irreversibly damaged.

The diagnosis of lead intoxication should be based on a history of exposure, the patient's clinical picture and corroborative laboratory data. This laboratory data should include a complete blood count, x-ray examination of the long bones, a quantitative blood and urine lead-determination and a urine copoporphrin test. One of these tests is insufficient evidence for the diagnosis of lead intoxication; other diseases may give a similar result. Only by evaluating all these tests can an accurate diagnosis be made. Positive blood and urine values indicate a person was exposed to lead, but not necessarily that he is poisoned by lead. When the blood-lead level is greater than 0.10 mg/100 ml of blood, and the urine-lead level is greater than 0.15 mg/litre, a probable lead intoxication is present. In order to obtain reliable chemical data, extreme care must be used in taking the samples. Very small quantities of lead are found even in intoxicated cases. If samples are improperly taken, they may have enough contamination from ordinary glassware to invalidate the results. The glassware containing the samples must be prepared and cleaned by the laboratory that makes the test to insure that they are lead-free.

Space precludes a detailed discussion of the other metals. More complete information will be found in the bibliography. The following brief discussion presents only a few salient comments on several metals.

Beryllium is a relative newcomer to the industrial scene. Ignorance of its toxicology led to poor handling during its early industrial use. Subsequently, many toxic reactions were reported, including acute pneumonitis, 10

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pulmonary granulomatosis and skin ulcers. Non-occupational exposures have come from broken fluorescent lamps which used beryllium as a phosphor. This non-industrial exposure is now precluded since the lamp manufacturers voluntarily have eliminated the beryllium phosphor.

Mercury and arsenic have already been mentioned as sources of intentional non-occupational accidents. Occupational hazards also may exist wherever these substances are processed. Arsenic is a common impurity in many ores, and processing these ores may release injurious amounts of arsine. Twelve accidental poisonings due to arsine occurred recently when a group of workers in a metal reclaiming plant processed a batch of metal containing a sizeable amount of arsenic impurities. Mercury is used extensively in industry and many vapor hazards exist because of poor housecleaning. Pools of the metal accumulate in unseen areas and their vapors then cause trouble. Mercurials are used therapeutically and, occasionally, extended use of these compounds may cause a toxic reaction, particularly in individuals with poor kidney function.

The last group of the inorganic substances to be mentioned are the non-metallic dusts. These dusts may produce a pneumoconiosis. This term, when correctly used, implies any type of pulmonary reaction to dust without regard to the severity of the reaction or to the effect on respiratory function. It is a general term and covers all exposures to dusts, hazardous or benign. The specific type of pneumoconiosis is named after the causative agent; e.g., silicosis, anthracosis, asbestosis. Only those dusts that produce fibrosis of the lungs are important to this discussion; i.e., silica and asbestos. Of these only the small particles are dangerous. Those particles greater than ten microns (one micron is a millionth of a meter) settle out quickly. Those that enter the respiratory tract and are greater than three microns are filtered out in the upper respiratory tract. Only those less than three microns can penetrate to the alveoli and are dangerous. These small particles are responsible for the lung pathology that is associated with silicosis and asbestosis. Silica, which chemically is silicon dioxide and not a silicate, and asbestos are the chief causes of this hazard. Silicosis in a live
patient can be diagnosed accurately only if a history of prolonged exposure exists and an x-ray examination reveals the abnormal pathology of the lungs compatible with this disease. Exposure to this dust hazard may lower the exposed person’s resistance to tuberculosis. However, neither pulmonary carcinoma nor pneumonia are developed by silicotics as a primary consequence of their exposure.

The organic compounds are those primarily derived from distillation of petroleum (the aliphatics) or of coal (the aromatics). With the tremendous strides the chemical industry is making today, these compounds are constantly utilized and new ones are being synthesized. Little is known about the toxicology of many of the newer substances, and, as each is put into production, elaborate tests have to be made to insure safe handling by workers and to determine what constitutes a harmful exposure.

The aliphatic hydrocarbons such as methane, propane, butane, ethylene, gasoline and benzine act on the central nervous system producing anesthetic effects. They produce symptoms that are typified by those of ethyl alcohol: first, a feeling of exhilaration and well-being; next, stronger states of depression leading to impaired judgment and motor incoordination, progressively getting worse and leading to convulsions in some cases. This is followed by unconsciousness with gradual paralysis of the respiratory center, leading to coma and death. The following simple facts are true of all these aliphatic compounds:

1. Any excess is harmful.
2. Few of these gases or liquids are stored in the body. If free from exposure 16 hours daily, one generally cannot build up any extreme saturation from normal exposures.
3. A short, severe exposure results generally in short disability.
4. Prolonged daily exposure to large amounts may lead to chronic poisoning.
5. Odor and irritation to eyes, nose or throat are usually safe guides to hazardous exposures.

Exposures which produce mild anesthesia may predispose individuals to accidental injury because of their lack of motor coordination and impaired judgment.

Chloral and paraldehyde have already been mentioned as other members of this group that have caused accidental, non-occupational injuries.

Common solvents such as chloroform, carbon tetrachloride, ethylene

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dichloride and the chlorinated hydrocarbons, cause many hazards in both industry and home. When these solvents are used as cleaning materials, absorption through the skin, inhalation of vapors when working in a confined, poorly ventilated room, and inhalation from fabrics saturated with these solvents during the cleaning process— all contribute to the hazard. Exposure to these substances may not reveal any immediate effects, but these protoplastic poisons affect the liver and/or kidney. Thus, exposed persons have been known to walk away from the exposure only to succumb to the hepatotoxic effects three to thirteen days later. The chemical laboratory data are seldom helpful in these cases since the disability usually is not recognized until after the traces present in the blood have been excreted.

Of all the aromatic compounds, benzene (benzol) has been one of the greatest offenders because it is cheap, available and useful for many different applications. Industry is gradually being educated to the nature of this hazard, and slowly is replacing benzene with other less noxious materials, such as toluene and Stoddards solvent. The most common exposure is a chronic one. Benzene, under these chronic conditions, attacks the hematopoietic system, particularly the bone marrow, resulting in anemia and increased susceptibility to disease due to a decrease in white cells. To establish the diagnosis of benzene intoxication, as in previous cases mentioned, one must have a history of prolonged exposure, a clinical picture and laboratory data. The laboratory data is usually in the form of complete blood studies and a urinary sulfate ratio. The latter is a fair index of exposure. This test can only be performed within twenty-four hours of exposure; thereafter the urinary sulfae ratio returns to normal.

The other organic compounds are too legion for mention. Just to read the names would take hours. It will be sufficient to name a few in passing. The barbiturates cause many a suicide today. Their promiscuous use and availability contribute to this problem. The alkaloids, such as the opiates, the strychnine group, the belladonna or atropine group and nicotine, were a greater problem years ago than they are today. They are not easily procured and have been replaced, in some cases, by newer synthetics. Occasionally they cause non-occupational accidents, but seldom an occupational one. The number of organic insecticides, herbicides, pesticides and rodenticides that are constantly being synthesized is growing yearly. These pose serious problems to the relatively uneducated public in the matter of safe handling and storage. They also are an occupational hazard to those agricultural workers who use them as sprays.


Another type of organic poisoning that should be mentioned is food poisoning. This may result from ingestion of adulterated food containing poisonous substances or from foods such as mushrooms, herbs and roots which may themselves be poisons because of their alkaloidal content. The so-called "ptomaine poisonings" are not germane to this discussion because they are reactions to bacterial infection and are not due to chemical agents. Ptomaines are organic compounds that are relatively innocuous when taken internally. The food poisoning to which the ptomaines have misleadingly given their name is due to bacteria, either the staphylococci, salmonella or botulinas type.

Before this article is ended, one final note is important. This has only been a cursory review of very few of the common toxic substances, many of which were not even named. Occupational diseases constitute the bulk of exposures to toxic substances and have been given the greater emphasis. These exposures may cause serious injury, even death. However, proper precautions can and must be taken. Under these circumstances, industry can continue to grow with the assistance of a stable labor force of healthy workers. The only way this can be accomplished is by constant vigilance to detect hazards. Once these are uncovered, steps can be taken to remove or ameliorate them. In those few cases where death occurs, it is most important to have a post-mortem examination of the body, including complete autopsy and a toxicological examination of the organs. Only in this way can the true facts be ascertained.