Diphenyl Poisoning in Fruit Paper Production

A New Health Hazard

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Production of diphenyl-impregnated fruit wrapping paper under poor hygienic conditions caused the death of one man and the poisoning of eight other workers. The diphenyl concentrations in the air had been much in excess of the present threshold limit value of 1 mg/cu m. The clinical picture of diphenyl poisoning is characterized by central and peripheral nervous damage and liver injury. The cause of death in the fatal case was acute yellow liver atrophy. In three of the eight men with poisoning, there was histological evidence of liver injury. Changes in the results of blood analysis or renal function did not occur. The prognosis of diphenyl poisoning is still unknown, but some patients showed deterioration when seen after one year.

The fungistatic agent diphenyl is an effective preservative for citrus fruits. It is usually impregnated into the wrapping paper; the air space between this and the fruit then becomes saturated with diphenyl vapor.[1] Impregnation is usually performed in the paper mill that manufactures the wrapping. Diphenyl is first dissolved in paraffin oil and then applied by a heated roller to the paper while it is still in the machine. The reels are then printed and cut into pieces, packed, and distributed to the consumers. All operations in which the agent itself or impregnated paper is handled expose the workers to various concentrations of diphenyl.

Few studies on the toxicology of diphenyl exist. The lack of data on human poisoning has created the impression that its toxicity is fairly low.[2] However, as early as 1947 Deichmann and his collaborators[3] were able to show that prolonged exposure to air containing diphenyl in concentrations of 5, 40, and 300 mg/cu m produced liver and kidney injury as well as bronchopulmonary lesions in mice and rats. Cutaneous application also gave rise to toxic effects.[3] The authors concluded that a concentration of 5 mg/cu m in the air should be considered dangerous for man subjected to prolonged exposure.[3] According to other authors, prolonged feeding (0.5% and 1.0% diphenyl in the food) may result in toxic symptoms, ie, inhibition of growth and kidney injury.[4]

The clinical picture of human diphenyl poisoning has so far not been described. The only data we have been able to find consist of a study from Strasbourg, France, describing transient nausea, vomiting, and bronchitis in "a certain number of workers" engaged in impregnating wrapping paper with diphenyl.[5] On the other hand, Weeks and Lentle[6] were not able to detect any difference between 47 workers exposed to organic reactor coolants (terphenyl and diphenyl) and 47 unexposed workers with regard to blood pressure, pulmonary function tests, serum creatinine and urinary protein values, and standard blood cell count. The air concentrations, however, were low (less than 1 mg/cu m).[6]

In the fall of 1969, a so-called oilman who had been intensively exposed to diphenyl for 11 years became ill and died. Since diphenyl poisoning seemed to be the most probable cause, a study of all exposed process workers in the paper mill was carried out. The results of this survey will be reported below.

Production Methods and Exposure

Diphenyl-impregnated wrapping paper for citrus fruits has been manufactured in Finland for about 15 years. In the process, diphenyl is dissolved in heated pure paraffin oil. This solution is prepared in a separate room, where a special worker (oilman) dispenses diphenyl from paper sacks into a mixing container. The mixture is heated to 90 C and pumped into a basin connected with the paper machine. The diphenyl-paraffin oil solution is then spread on silk paper with the use of a roller at the dry end of the paper machine at a temperature of 90 C. The production of such paper is discontinuous, but has increased over the past few years. At the end of the 1960's diphenyl paper was produced about one third of the year. Before a change in the type of paper, the diphenyl basin and containers as well as the pipelines and the equipment between them are emptied and cleaned. Cleaning was earlier done by trichloroethylene but since 1968 tetrachloroethylene has

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been used. The cleaning of equipment, which requires one hour, is done by the oil-man five to ten times a year.

In 1959, the workers had complained about the strong odor and of irritation of the throat and the eyes. The concentrations of diphenyl in the air were then between 4.4 and 128 mg/cu m (see Table 1). After these measurements only a simple exhaust hood was built over the paper machine and a ventilating fan with two exhaust ducts was installed in the oil-room. The next time measurements were performed was in January 1970 after the death of the oil-man. The results are shown in Table 1. As can be seen, the exposure of the oil-man had been intensive indeed. This job usually requires about 100 to 105 eight-hour shifts every year.

The concentrations were particularly high in the oil-room when diphenyl was added to the mixing container. Checks of the measuring container with the lid open also resulted in high exposure. In the oil-room, diphenyl occurs both as vapor and dust; in the paper machine hall, predominantly in vapor form. Skin contact is also likely to produce additional exposure. In the fatal case described below this route probably played an important role.

The oil-man's exposure to tetrachloroethylene while cleaning the equipment was insignificant considering the short exposure time and the results of the measurements made (Table 2). Nor could mineral oil vapor be a hazard because of its low vapor pressure (1.8 mm Hg at 100 C) and high boiling point (297 C).

Table 1.—Diphenyl Concentrations in Air of Paper Mill

Sampling Locations	Average Concentrations, mg/cu m	
	June 1959	January 1970
Paper machine hall		
In front of paper reel	17.9	7.2
Behind impregnating roller	128.0	64.0
Near paper machine	7.2	1.5
Near rolling machine	4.4	0.6
Oil-room		
Near measuring container	19.5	3.5
Near mixing container	. . .	15.5
During addition of diphenyl into mixing container	. . .	74.5
Above measuring container (lid open)	. . .	123.0

Table 2.—Tetrachloroethylene Concentrations During Cleaning of Diphenyl Containers and Pipelines

Sampling Locations	Average Concentrations, cc/cu m (ppm)	
	January 1970	November 1970
Paper machine hall		
In front of paper machine roll	9	30
Near operator of paper machine	30	14
Oil-room		
Near measuring container	75	34
Near mixing container	40	. . .
Breathing zone of oil-man during cleaning	32	35

Determination of Diphenyl in Air

Diphenyl in the air was determined both by direct ultraviolet light absorption spectrophotometry and by spectrofluorometry. The air samples were collected in ethyl alcohol (two flasks in series). The UV-absorption was measured at 248 nm. The samples were excited at wavelength 258 nm by spectrofluorometry and the emission was measured at 314 nm. The methods will be published in detail elsewhere.[7]

Material and Methods

A total of 31 men were engaged in the process and they are all included in the study. Six of them were oil-men, 13 were paper-machine workers, 7 were rolling-machine workers, 4 were handling residue mass, and one was a maintenance worker. Two additional workers were included in the study because of suspected poisoning: one stockkeeper (out of seven), and one female paper cutter (out of 20). Thus, the total number of examined workers was 33, one of whom was a woman.

The workers were first examined clinically and had a number of laboratory tests taken (blood cell count and measurements of thrombocytes, serum transaminases, urinary protein, and glucose). On the basis of anamnestic data, clinical findings, or pathological laboratory test results, eight men were admitted to the hospital for closer examination (see case histories), and 14 other men had a neurophysiological examination. Since the hospitalized men also were examined by these methods, 22 subjects in all were examined neurophysiologically.

The electroencephalogram was recorded for 30 minutes and included a three-minute photostimulation and a three-minute hyperventilation. The electroneuromyographic (ENMG) examinations comprised electromyography (EMG) of five to ten muscles, maximal conduction velocities (MCV) of the median, ulnar, peroneal, and posterior tibial nerves as well as measurement of the conduction velocity of the slower fibers in the ulnar nerves. The sensory thresholds were measured with the use of square wave pulses of 1 msec duration. A detailed description of these methods and our criteria for normal and abnormal, based upon measurements on 120 healthy persons, have been published earlier.[8]

The hospitalized patients received other examinations, including thin-needle liver biopsy, and in some instances lumbar puncture, pneumoencephalography, clinical neurological and otoneurological examinations, and psychological testing.[9] The most relevant methods of examination are referred to in connection with the case reports.

Results

Summary of Clinical Findings.—The most common complaints were headache, gastrointestinal symptoms (diffuse pain, nausea, indigestion), polyneuritic symptoms (numbness and aching of the limbs), and general fatigue. These symptoms were so common that diphenyl must be blamed for most of them even

Table 3.—Liver Function Tests of Eight Men Hospitalized for Suspected Diphenyl Poisoning

Case No.	Age (yr)	Exposure Time (yr)	Liver Biopsy	BSP Retention, %*	SGOT†	SGPT†
2	27	8	Normal	4	20	28
3	52	12	Incipient hepatic cirrhosis	17	100	120
4	27	6	Normal	3	50	61
5	55	16	Normal	2	24	27
6	43	13	Fatty metamorphosis	17	20	32
7	44	15	Normal	8	20	25
8	30	5	Normal	. . .	54	59
9	55	15	Fatty metamorphosis	10	54	64

*Forty-five minutes. †Wroblevsky-units.

though no control group was used for comparison. The distribution of the main symptoms was as follows: headache, 13 cases; gastrointestinal symptoms, 13; polyneuritic symptoms, 12; fatigue, 11; giddiness, 7; and symptom-free, 7.

The objective symptoms are mentioned in connection with the case reports. The laboratory tests yielded little information; however, of the 33 subjects studied, ten had elevated (more than 40 Wroblevsky [WR] units) serum glutamic oxaloacetic transaminase (SGOT) or serum glutamic pyruvic transaminase (SGPT) levels, or both.

Of the 22 men examined neurophysiologically, only three were without pathological findings. Four had borderline findings and the remaining 15 had one or more pathological test results. The distribution of neurophysiological findings was as follows: abnormal findings in EEG, three cases; abnormal findings in ENMG, five; abnormal findings in EEG and ENMG, seven; borderline findings, four; and no abnormal findings, three.

The frequency of specified neurophysiological findings in the 22 men exposed to diphenyl was the following: Of eight EEG abnormalities, there were six diffuse slow wave abnormalities and two spike and wave discharges (3 to 6 cycles per second). There were eight abnormal alpha distributions, four with other pathological findings and four without. There were nine instances of reduced nerve conduction velocity, three with MCV slowed and eight with slower fibers affected. Nine pathological EMG findings included three with reduced number of motor units and six with reduced number of motor units plus fibrillations. Elevated sensory threshold was found in five instances.

According to these results, diphenyl seems to exert a toxic action both on the brain and on the peripheral nervous system. The frequency of pathological findings was remarkably high even considering that the subjects were selected. A more detailed consideration of the neurophysiological findings will be published elsewhere.

Liver biopsy was performed on all hospitalized subjects. The results of this examination together with other indicators of liver function are shown in Table 3.

In five cases indications of liver injury were present. The biopsy showed hepatic cellular changes in three of them. These findings, together with the autopsy findings of case 1 (see below), suggest that the liver is one target for the toxic effects of diphenyl.

Report of Cases

Because there are no data published on human diphenyl poisoning, the nine cases studied more thoroughly will be presented in some detail. The first case is the fatality that initiated the survey; since very little of the toxicology of diphenyl was known to us at this stage, some relevant examinations were not made.

CASE 1.—The patient was a 32-year-old man, exposed for 11 years as an oil-man (see Table 1), who became ill in the summer of 1969. He had been completely healthy until then, did not use any drugs, and consumed alcohol moderately (about 500 gm expressed as absolute alcohol per month). His first symptoms were fatigue, abdominal pain, headache, and psychic disturbances (irritability, sleep disturbances, loss of memory). He visited two physicians during the fall, but no diagnosis was made. In November he had to stop working and was admitted to the hospital four days later. He was somnolent, icteric, and had severe ascites and massive edema in the legs (6.8 and 3.7 liters of ascites, respectively, were tapped on two occasions). Laboratory values included the following: hemoglobin, 11 gm/100 ml; leukocytes, normal; thrombocytes, 118.000/cu mm; urine protein, positive; plasma creatinine, normal; SGOT, 128, 180, and 94 Wr units; SGPT, 45, 100, and 116 Wr units; and total bilirubin, 17.3 mg/liter, of which 0.3 mg/liter was directly reacting. The prothrombin index (Quick) was first 24% and later 5%. The value for total serum protein was 60 gm/liter (30.3% albumin, 4% α_1-globulins, 3.4% α_2-globulins, 62.3% β- plus γ-globulins). The IgG and IgA were increased and the complement decreased. Sternal biopsy suggested hemolytic anemia. The EEG showed diffuse theta and delta activity compatible with brain damage.

The patient was treated with prednisolone (50 mg/day), parenteral menadione and thioctic acid (Thioctan), and neomycin (to prevent liver coma). In spite of all efforts, his condition grew progressively worse. On Dec 17, 1969, he had a large hematemesis. Two days later he died.

At autopsy, which was performed on the 11th day after death, the liver showed necrosis of most cells. The findings were partly compatible with acute yellow necrosis, but there were also cirrhotic areas. The kidneys showed severe nephrotic changes, and the heart muscle, degenerative changes. The brain was edematous and there was degeneration of the ganglion cells of the gray substance. The bone marrow was hyperactive, showing increased numbers of immature white and red blood cell precursors. There were only a few megacariocytes.

The patient was conscious until the last few days. During the four weeks in the hospital, his history was thoroughly investigated. There was no history of any liver disease or of severe infections. Alcohol seemed to be an unlikely cause for severe liver injury in such a young man. The fact that the patient had been working regularly until four days prior to hospitalization also spoke against alcoholism. He firmly denied any abuse, admitting only one bottle of vodka (500 ml) per week or

even less. The fact that he had been using tetrachloroethylene for washing the machines was given thorough attention; however, it seems unlikely that such a small exposure (about 10 to 75 ppm for one hour, five to ten times a year) could contribute to the fatal poisoning. He also firmly denied any abuse of this agent. Since no other plausible cause could be found, and since there were pathological changes in many different organs, the proof in favor of poisoning seems convincing. The most likely agent was diphenyl; this hypothesis was further supported by the long exposure to concentrations occasionally reaching even more than a hundred times the threshold limit value (TLV).

CASE 2.—A 27-year-old man had been exposed to diphenyl for ten years at the paper machine and as an oil-man. He complained of headache, weakness of the left arm, and abdominal pain. Liver functions were normal. The clinical neurological examination revealed motor dysfunction of the left hand and loss of superficial sensation in the areas of the ulnar nerve. The EEG and ENMG findings were normal.

CASE 3.—A 52-year-old man had been exposed for 12 years as a loader and a diphenyl stockkeeper. Skin absorption must have been considerable since he had been handling diphenyl with his bare hands. His alcohol consumption was about one bottle of wine a week. His main complaints were fatigue, abdominal pain, diarrhea, sweating, dysuria, impotence, and numbness of the legs. The transaminase levels were elevated (SGOT, 100 and SGPT, 120 Wr units), the sulfobromophthalein (Bromsulphalein, BSP) retention was 17% (normal upper limit, 8%), and the γ-globulins were increased. The liver biopsy showed parenchymal and portal tract fibrosis. The lobular architecture had deteriorated. The changes were indicative of incipient cirrhosis. The neurological examination revealed horizontal rhythmic nystagmus, hyperhidrosis, and impairment of all sensory qualities on the right side of the body. The sensory findings were compatible with a thalamic lesion. The ENMG findings were normal, and the EEG findings were borderline, showing unusual alpha distribution.

CASE 4.—A 27-year-old man had been working as an oil-man for six years. His health had been good, and his alcohol consumption was "slight." During the last two to three years he had developed increasing headache, fatigue, and numbness of the limbs. His memory was subjectively weakened, he slept badly, his potency was poor, and he was irritable. Occasionally he had diarrhea and dysuria. The only pathological liver function was a transient elevation of the transaminase levels (SGOT, 50 and SGPT, 61 Wr units). The clinical neurological examination revealed organic mental deterioration, hyperhidrosis, tremor, and neurogenic muscular atropy in the interosseus muscles. The cytological study of the cerebrospinal fluid revealed lipophages compatible with tissue destruction. The pneumoencephalogram was normal, however. The EEG was abnormal, showing mild to moderate diffuse slow wave abnormalities, particularly in the left posterior areas. The first ENMG was within normal limits. The acoustic and vestibular functions were disturbed. The psychological tests showed significant deterioration, especially for psychomotor functions. The examination was repeated seven months later; in the interim there had been no exposure to diphenyl. The condition of the patient had deteriorated. The EEG was as before, but the psychological tests were more abnormal. As a new feature, polyneuropathy was now diagnosed (slowing of nerve conduction by 20%, fibrillations, and diminished number of motor units in the EMG).

CASE 5.—A 55-year-old man had been working as a diphenyl-paper line operator for 16 years. His right forearm had been wounded in the war and was partially paretic. He had had low-back pain for 18 years. In 1969 he was operated upon because of a suspected prolapse of the presacral intervertebral disk but only an atrophic nerve root was found. During the last two years the patient had suffered from fatigue, headache, loss of memory, and pharyngeal irritation. Results of the liver tests were normal. The neurological examination showed organic mental deterioration and spontaneous rhythmical nystagmus. The otoneurological examination revealed perceptive hearing loss and disturbance of the vestibular function. The EEG showed slight diffuse slow wave abnormality. Nerve conduction was slowed by about 10% both in the peroneal and posterior tibial nerves, especially in the left leg. Fibrillations and a diminished number of motor units were noted in the EMG. A peculiar spontaneous activity, occurring in rhythmical series lasting from 10 to 15 seconds, was registered from the left anterior tibial muscle. The ENMG findings were not typical for mechanical nerve root compression, but were more compatible with polyneuropathy or partly with neuronopathy of anterior horn cells. The patient was considered to be incapable of working. Although there was evidence of poisoning, his orthopedic condition accounted for the major part of the disability.

CASE 6.—A 43-year-old man had been working as an oil-man for 14 years. He had been previously in good health and consumed no alcohol. For some years he had various symptoms of increasing intensity: abdominal pain, pharyngeal irritation, nosebleeds, numbness of the arms, mental irritation, sleep disturbances, impotence, and loss of memory. The BSP retention was pathological (13%; controlled, 17%; normal upper limit, 8%). Liver biopsy specimen showed distinct fatty metamorphosis and slight inflammatory changes. The finding was compatible with toxic changes, although nonspecific. Neurological examination revealed organic mental deterioration and signs of motor and sensory neuropathy. Electronystagmography disclosed moderate disturbance of the vestibular function. The EEG was normal. The conduction was slowed by 10% in some peripheral nerves. The EMG showed diminished number of motor units in several muscles.

CASE 7.—A 44-year-old man had first worked for 2 years as an oil-man and then for 13 years as a maintenance worker. Previously, he had been in excellent health; he used no alcohol. During the whole exposure time irritation of the nasal mucosa and the conjunctivas developed during work. For several years he experienced giddiness, fatigue, tremor, and pain in the forearms, back, and shoulders. Abdominal pain had been present for three years. In addition, he complained of loss of hearing, impotency, and mental alterations (depressive mood, irritability, loss of initiative). There were no signs of liver injury. The clinical neurological findings included dysfunctions of the autonomous nervous system and mixed peripheral neuropathy. There was a marked perceptive hearing loss, but normal vestibular function. The EEG showed mild diffuse slow wave abnormalities. The conduction velocity in the slower nerve fibers was reduced by 10%. The number of motor units in the EMG was diminished.

CASE 8.—A 30-year-old man had been preparing the diphenyl solution for five years, two to five months a year. The rest of the time he had been working at a paper machine, situated near the oil-room, but not used for manufacturing diphenyl paper. The diphenyl concentrations near this paper machine were not measured. As a child he had had mild hepatitis, but had made a full recovery. He used alcohol less than ten times a year. He had had headaches for several years and abdominal pain for some two years. He had lost consciousness twice while at work. The transaminases had been slightly increased during 1969 to 1970, but the liver was his-

tologically normal. Objective neurological findings included hyperhidrosis, spontaneous rhythmical nystagmus, and a central facial paresis on the right side. The EEG revealed spike and wave discharges during photostimulation, spontaneous curve normal. The conduction velocity of the slower nerve fibers was 10% less than the normal limit, but the MCVs were normal. Electronystagmography disclosed disturbed vestibular function bilaterally. A year later, the EEG showed mild diffuse slow wave abnormality in addition to the spike and wave complexes induced by photostimulation. All nerve conduction velocities were by this time normal.

CASE 9.—A 53-year-old man had been working at the diphenyl paper machine for 15 years. Apart from a low-back condition he had been in good health; he used no alcohol. Subjective symptoms had been present for several years: headache, numbness of the limbs, giddiness, and abdominal pain. The transaminases were slightly increased and the BSP retention was borderline (Table 3). The liver biopsy showed distinct centrilobular fatty metamorphosis, characteristic for toxic substances. On EEG, there were slight diffuse slow wave abnormalities and homogenization of the alpha activity. The ENMG showed neurogenic lesions of slight degree probably on the medullary level ranging from the cervical to the lumbar region. The patient was considered to be completely incapable of working.

Comment

This unfortunate story offers an example of how erroneous conclusions regarding the toxicity of an industrial chemical may lead to a false feeling of safety. Hygienic precautions had been inadequate and resulted in extremely high concentrations of diphenyl in the air. It is clear that with such careless handling, any chemical may be dangerous. However, a heavy responsibility falls on the producers, who as late as 1965 in their technical service bulletin stated that, "Laboratory and practical investigations have shown that diphenyl is completely harmless in quantities several times greater than that to which workers are exposed in the manufacture or use of diphenyl impregnated paper." Such a view is somewhat surprising considering the results of Deichmann et al,[3] and this statement no doubt contributed to the nonchalant handling of diphenyl in the paper mill.

Although Deichmann and his collaborators warned of the toxicity of diphenyl as early as 1947,[3] no formal safety recommendation was given until 1968. In that year, the American Conference of Governmental Industrial Hygienists set a TLV of 1 mg/cu m.[11] The documentation is defective, however, since the literature contains very few observations. Unfortunately our data, too, are difficult to interpret in terms of the safety of the present TLV, since exposure was much in excess of 1 mg/cu m. The only thing that can be stated is that high concentrations are anything but harmless to man. Such dangerous concentrations apparently can result from the production of impregnated fruit paper if hygienic measures are neglected.

The clinical picture of diphenyl poisoning is characterized both by central and peripheral neurological lesions. The exact type of brain lesion remains to be defined. Partial demyelination may be the most probable explanation for the peripheral findings, but this suggestion is still hypothetical. However, even these incomplete data clearly show that diphenyl is a neurotoxic agent. It is unfortunate that the nervous system has received so little attention in animal experiments.[3,4] More careful observations might have provided important clues as to the mechanism of nervous damage. Another main target of toxic action appears to be the liver. Of the nine patients examined more thoroughly, four had histological evidence of liver damage. The possible role of alcohol can, of course, never be excluded with full certainty, but the fact that all workers had had stable employment for several years, and none was known to abuse alcohol, strongly speaks against alcoholism as the cause of the liver damage found.

The prognosis of the poisoning is still unknown. At present there is no reason to be optimistic, since some patients' conditions seem to deteriorate in spite of complete avoidance of any contact with diphenyl. The deterioration concerns both central and peripheral nervous functions. Slight improvement has sometimes been observed, however. Thus the final concept of the prognosis must await the results of a longer observation period.

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