Two Episodes of Acute Illness in a Machine Shop

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Abstract: Following an explosion in a machine shop and temporary plant closure, on the day the plant returned to full operations a degreaser malfunctioned. Workers in the assembly room were exposed to trichloroethylene levels later estimated to have exceeded 220 ppm (OSHA PEL 100 ppm). The plant was evacuated and the degreaser taken out of operation. Blood testing for carbon monoxide (CO) on five employees found carboxyhemoglobin levels in excess of normal. The plant reopened the following morning. Over the next two weeks, 15 employees were seen by the plant nurses for similar complaints; although all returned to work, their carboxyhemoglobin levels, later found to be inaccurate, were reported by a local medical clinic to range from 13.7 to 20.0 percent. At the end of the second week, another outbreak of illness occurred, but carboxyhemoglobin, trichloroethylene, fluorocarbons, and methylene chloride were not elevated in all 17 persons tested; plant-wide monitoring for CO found no elevated levels. During the first outbreak of illness, cases were 2.26 times as likely to have entered the assembly room as noncases. During the second outbreak, cases were no more likely than noncases to have entered the assembly room. We believe the explosion, earlier toxic exposures and illness, and the misleading blood test results led to plant-wide anxiety which culminated in a collective stress reaction and the second outbreak. An open meeting with all employees, informing them of our findings, provided reassurance and no further episodes of illness occurred in this workforce. (*Am J Public Health* 1989; 79:1024–1028.)

Introduction

Disease is the result of the interaction between a host, an agent, and the environment. The host must be susceptible to the agent and the environment dictates if the agent and host contact each other. In this paper, we describe two outbreaks of illness in a manufacturing plant within 16 days. Whereas the workers' symptoms were similar on both days, the agents and environments differed. Although the outbreaks were not independent of one another, we believe they represented separate illnesses with unique causes.[1]

The 225,000 sq ft factory manufactures rotary compressors for small air conditioners. At the facility, internal compressor parts are machined to specifications using a variety of machine tools. Welding, brazing, painting, and soldering operations are used as appropriate during compressor assembly. The majority of the workers are unskilled and assigned to the same daily task with little or no variation in their day-to-day routine. A floor plan of the plant is presented in Figure 1.

Chronology of Events

A time line and chronology of events is presented in Figure 2.

On January 29, 1986, a regulator valve on the fuel line to the plant boiler malfunctioned resulting in a large explosion. Fortunately, this occurred while second shift workers were on break in a second floor lunchroom. No one was injured.

The plant reopened on February 3. First shift began at 7:30 am. Between 8:35 am and 12:30 pm, 10 employees became acutely ill and were taken to the local emergency room where they were evaluated at a local hospital for a presumed toxic inhalation. Their symptoms included headache, dizziness, disorientation, eye and throat irritation, nausea, vomiting, shortness of breath, and chest pain. Arterial blood samples, drawn for carboxyhemoglobin determination, were elevated in five workers (mean 14.0 percent, range 13.3–14.8 percent). All workers were treated and released. By early afternoon, first shift production was stopped; second shift workers were sent home as they reported for work.

State OSHA (Occupational Safety and Health Administration) inspectors and an industrial hygiene consultant hired by the company arrived that afternoon. Direct-reading mea-

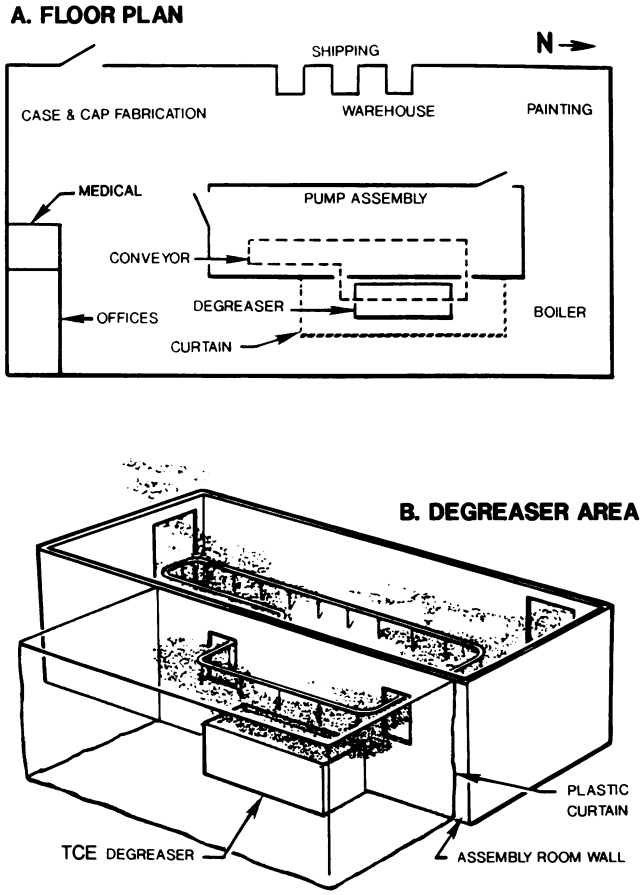

FIGURE 1—Floor Plan of a Machine Shop

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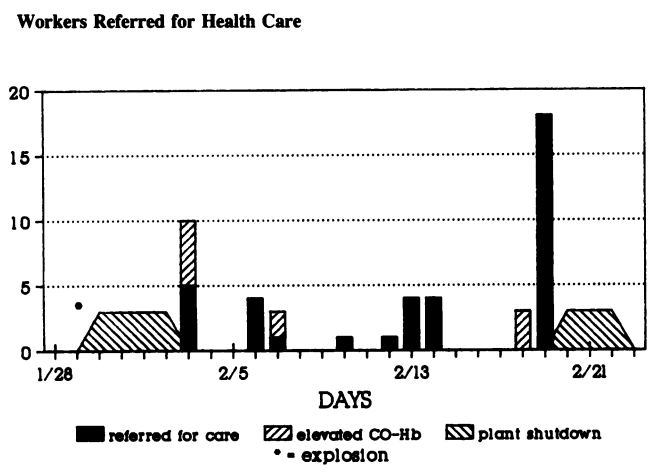


FIGURE 2—Time Line and Sequence of Events from Explosion January 28 through February 21

surements for carbonmonoxide (CO) were made throughout the plant but none exceeded the federal OSHA standard, the ACGIH threshold limit value (TLV), or the NIOSH (National Institute for Occupational Safety and Health) recommended standard.[2–5] Propane forklifts, open flames in the welding and brazing operations (located within the assembly room), and a portable heater were identified as CO sources. Two of seven forklifts, emitting excessive CO, were taken out of service, pending repair.

Workers interviewed the same day reported that the degreaser, started two hours before the beginning of first shift, had emitted trichloroethylene fumes into the assembly room. Following the explosion, the degreaser had been surrounded by polyurethane curtains to prevent contamination of compressor parts with residue from the explosion (Figure 1B). The curtains had enclosed not only the degreaser but also two openings in the assembly room wall through which parts were normally conveyed to and from the degreaser. On the morning of February 3, the degreaser malfunctioned. Apparently, the trichloroethylene level had fallen below the operating range and then overheated; excess fumes trapped within the polyurethane curtains escaped into the assembly room.

The degreaser was removed from operation but was restarted two days later to simulate the conditions of February 3. Measurements taken two hours after start up found trichloroethylene concentrations of 224 parts per million (ppm) in the assembly room. The OSHA permissible exposure limit (PEL) for trichloroethylene is 100 ppm as an 8-hour time-weighted average (TWA) and 200 ppm as a ceiling concentration.

Full production resumed the next day. Between February 4 and 18, 15 employees were seen in the plant infirmary with complaints of headache, eye and throat irritation, nausea, fatigue, and muscle aches. Twelve employees sought treatment at local medical facilities because of concern about continuing exposures at work. One clinic drew venous blood samples for carboxyhemoglobin determination from five workers. All were elevated, ranging from 13.7 to 20 percent. Three workers were told of their elevated carboxyhemoglobin levels while at work, during the morning of February 19. Local newspaper accounts of the February 3 mishap were published daily over the next four days. On February 13, the press had reported that another employee had been rushed to the hospital, "overcome by toxic fumes." All environmental samples for CO were well below the NIOSH recommended exposure level (REL) of 35 ppm.

On February 19, between 9 am and 11 am, workers again became acutely ill. Most were triaged from the single room plant infirmary where they could be found sitting or lying on either the floor, examining table, or stretchers. Two workers lost consciousness for a brief period. Fifteen workers were transferred by ambulance to the hospital. Emergency medical technicians carried stretchers through the plant production area as they left the facility. Four additional workers later requested a medical evaluation. Production at the plant was stopped.

At the hospital, 17 employees were tested for elevated carboxyhemoglobin levels, all tested normal (mean 2.3 percent, range 0.0–8.0 percent). Additional toxicologic screening for trichloroethylene, fluorocarbons, and methylene chloride was also negative.

State OSHA investigators inside the plant during the outbreak found CO levels less than 15 ppm.

NIOSH Investigation

NIOSH investigators arrived the next day. Our evaluation included an environmental survey and a review of the medical findings. With the exception of the trichloroethylene exposures on February 3, no environmental sources of toxic exposures likely to have caused acute illness were identified.

A review of medical records showed that treatment had consisted of monitoring vital signs, administering oxygen, and an occasional precautionary intervenous line. Although CO levels inside the plant were low, all carboxyhemoglobin levels in employees seen at the clinic from February 4–18 were elevated. At the same time, no carboxyhemoglobin levels were elevated at the hospital after February 3. One of us (PRK) submitted samples of his blood for carboxyhemoglobin determination on February 20. Results from the hospital were 0.03 percent, while the clinic results were 16.0 percent. Two additional samples, collected at the clinic from employees on February 21 (two days after the plant was closed) were also elevated, with carboxyhemoglobin levels of 16 percent and 20 percent. The inaccuracy of the clinic carboxyhemoglobin values were later confirmed by the regional laboratory director.

During meetings with the union and management, the decision was made to re-open the plant. An open meeting was held for all employees on February 23 informing them of our findings. NIOSH investigators reassured workers that the plant environment was safe and would continue to be monitored. Plant operation began immediately following the morning meeting.

Questionnaire

On February 25, a questionnaire was administered to 325 employees (279 first shift and 46 second shift workers). Second shift workers and eight others who had not worked on February 3 and February 19 or did not adequately complete the questionnaire were excluded. Employees ranged in age from 18 to 62 years with a mean of 33 years; 67 percent were

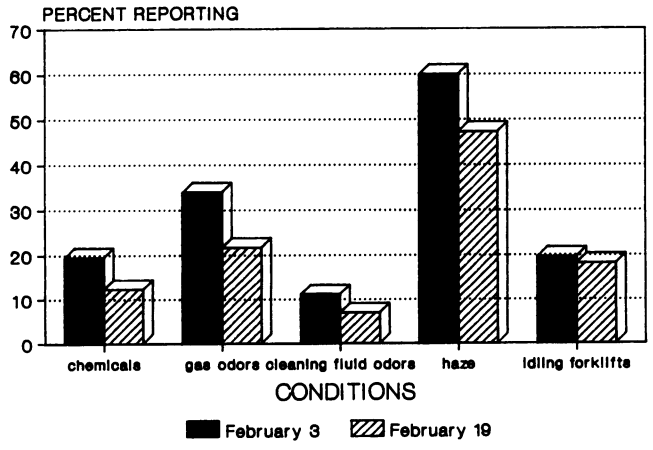
male. Almost 30 percent of the employees had not finished high school, 42 percent were high school graduates, 21 percent had some college or vocational training, and 7 percent were college graduates.

Figure 3 illustrates the proportion of employees who reported the presence of certain conditions in the plant. The presence of chemicals, odors of gasoline and cleaning fluid, and visible haze was reported by a greater percentage of workers on February 3 than on February 19.

Headache was the most common symptom on both February 3 and 19, with greater than 20 percent of the workforce reporting it. Other reported symptoms included sleepiness, sore throat, lightheadedness, nausea, and weakness. The percentage of employees reporting each symptom was similar on the two days. In order to define a case, we used the occurrence of headache plus a varied number of additional symptoms. If the number of symptoms experienced by an individual had been a function of exposure, we would expect that workers with an increased number of symptoms had received an increased exposure. This would result in a case definition that became more specific for exposure as it became more restrictive.

Employees were asked to recall their time spent in the assembly room on February 3 and February 19. Eighty-seven of 240 and 79 of 245 employees entered the assembly room on February 3 and February 19, respectively. On February 3, entry into the assembly room was associated with illness; the relative risk ranged from 1.33 to 3.10, increasing as the case definition became more restrictive (Table 1). No association between illness and entry into the assembly room was observed on February 19. Time spent in the assembly room was also associated with illness on February 3, but not on February 19 (Table 2).

On both days, women were more likely than men to become ill. On February 19, women throughout the plant had a two-fold increased risk of illness (RR = 1.99, 95% CI = 0.93, 4.23). In contrast, on February 3, women entering the assembly room were at greatest risk (RR = 3.15, 95% CI = 1.06, 9.37) while the RR for women versus men who did not enter the assembly room on February 3 was much lower (RR = 1.24, 95% CI = 0.35, 4.43).



Reported by 1st shift workers

FIGURE 3—Workers' Perceptions of Conditions Inside a Machine Shop during Two Episodes of Acute Illness.

Discussion

Our findings suggest that the cause of illness on February 3 differed from that on February 19. The illness of February 3 appears to have been the result of the toxic effects of CO complicated by exposure to trichloroethylene. The illness of February 19, however, more likely represents a collective stress reaction. The combination of the boiler explosion on January 29, toxic exposures on February 3, reports in the local press, and the misleading laboratory results reported to employees on February 19 led to plant-wide anxiety, which precipitated the acute outbreak of illness on February 19.

On February 3, presence in the assembly room was associated with illness and this association was strengthened by both an increase in the number of symptoms and time spent in the assembly room. Unfortunately, the cause of the elevated carboxyhemoglobin levels of February 3 was not identified. Contact with strong alkalies may cause trichloroethylene to decompose into CO, phosgene, and dichloroethylene.[6] However, no strong alkalies were found in the assembly room and symptoms suggestive of phosgene poisoning were not reported.

The February 19 outbreak shares a number of common features with other reports of collective stress reaction.[7–12] No exposures were identified in the work environment at the time of the illness. The ill workers developed a wide variety of relatively severe symptoms, which contrasted sharply with a lack of physical findings. The spread of illness was very rapid, resembling an acute chemical poisoning rather than an outbreak of infectious disease. Perhaps most important, a very high level of stress had been building in this workforce since the boiler explosion on January 29.

In the workplace, collective stress reaction has been reported most often among groups of unskilled workers with less than a high school education performing highly repetitive unskilled tasks.[11] In those reports, women were more likely to have been affected than men. It should be emphasized that the association of gender and educational level with collective anxiety phenomenon does not imply that these are causative factors. The nature of the job, rather than gender or education, may place these workers at high risk.

Whenever workers are exposed to toxic substances, both they and their unexposed co-workers are subjected to psychological stress and the situation may produce collective stress reactions above and beyond the primary symptoms. This, in turn, can precipitate additional illness episodes, such as the one illustrated in this paper. This emphasizes the importance of a thorough and sensitive debriefing of the workers following any environmental or medical assessment. If worker fears and suspicions are not sufficiently allayed and episodes of illness do recur, both the investigators and the management can lose credibility, and the situation may escalate.

The conclusion that an outbreak is the result of collective stress reaction should be made with caution. There is always the chance that an undetected toxic exposure has been overlooked.[13–15] Illness resulting from psychologic factors is "real" in every respect. The term refers to illness in which the primary cause is psychological stress, arising from the occupational and/or general social environment, rather than environmental chemical, physical, or infectious agents. The occurrence of a collective stress reaction does not imply a psychiatric disorder. It represents a normal psychophysiological response to a stressful environment.

TABLE 1—Percentage of Ill Workers by Exposure in the Assembly Room, Risk Ratios, and p-values

	Headache + 1 Symptom	Headache + 2 Symptoms	Headache + 3 Symptoms	Headache + 4 Symptoms	Headache + 5 Symptoms
February 3					
% Assembly Room employees ill (N = 87)	17.2	16.2	13.1	13.1	12.1
% Other employees ill (N = 153)	13.0	9.7	5.8	5.2	3.9
Risk Ratio	1.33	1.68	2.17	2.53	3.07
95% CI	0.75,2.36	0.90,3.12	1.03,4.55	1.18,5.42	1.33,7.04
February 19					
% Assembly Room employees ill (N = 79)	15.4	12.1	10.3	8.8	8.8
Other employees ill (N = 166)	13.1	10.9	9.7	8.0	4.6
Risk Ratio	1.16	1.15	1.03	1.09	1.97
95% CI	0.63,2.16	0.58,2.27	0.49,2.20	0.48,2.47	0.80,4.88

*X^2 test

TABLE 2—Illness by Duration of Exposure in the Assembly Room

Case Definition	% ill *(RR)	% ill (RR)	% ill (RR)	Summary RR	95% CI
Minutes in Assembly Room	0	1–60	61–390		
		February 3, 1986			
Headache + 1 symptom	13.07	13.9	19.6		
	(1.00)	(1.06)	(1.41)	1.27	0.79,2.05
Headache + 2 symptoms	9.8	11.1	19.6		
	(1.00)	(1.12)	(1.75)	1.50	0.93,2.42
Headache + 3 symptoms	5.9	5.6	17.6		
	(1.00)	(0.95)	(2.21)	1.77	1.07,2.95
Headache + 4 symptoms	5.2	5.6	17.6		
	(1.00)	(1.05)	(2.36)	1.91	1.16,3.15
Headache + 5 symptoms	3.9	2.8	17.6		
	(1.00)	(0.74)	(2.70)	2.12	1.28,3.52
Number of employees	153	36	51		
		February 19, 1986			
Headache + 1 symptom	13.9	17.2	12.0		
	(1.00)	(1.24)	(0.88)	1.01	0.57,1.79
Headache + 2 symptoms	11.4	17.2	8.0		
	(1.00)	(1.48)	(0.73)	1.01	0.54,1.90
Headache + 3 symptoms	10.2	13.8	6.0		
	(1.00)	(1.33)	(0.63)	0.88	0.44,1.82
Headache + 4 symptoms	8.4	10.3	6.0		
	(1.00)	(1.21)	(0.75)	0.92	0.43,1.96
Headache + 5 symptoms	4.8	10.3	6.0		
	(1.00)	(1.93)	(1.19)	1.47	0.72,3.00
Numbers of employees	166	29	50		

*RR = the risk among the exposed/the risk among the unexposed.
@Mantel-Haenszel X^2 rest for dose-response.[15]

ACKNOWLEDGMENTS

A version of this work was presented at the 1987 meeting of the American Public Health Association in New Orleans. Copies of the questionnaire used in this study and reprints may be obtained by contacting the first author (TS). The authors thank Peter Boxer and Michael Colligan for reviewing the original manuscript and Pat McGraw for its preparation.

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The 1990 Census: Size and Scope

The 1990 Census of the United States will be the 21st in the nation's history and the largest and most complex ever undertaken. The Census Bureau has been planning the mammoth operation since 1984. The size and scope of the 1990 Census will include:

- *Expected count*—250 million people, 106 million housing units.
- *Employment*—Will hire about 565,000 people, with 300,000 working at peak.
- *Location:* 484 field offices, 13 Regional Census Center, 7 processing offices.
- *Coverage*—Over 106 million questionnaire packages to print, label, and process.
- *Geography*—Computerizing entire map base, including over 250,000 different base maps. Nearly 7 million maps will be generated to help census takers collect questionnaires.
- *Participation*—Over 70 million mail returns within two weeks of Census Day, April 1, 1990.
- *Technology*—Using 570 minicomputers in field offices, to be set up, used, and dismantled in one year.
- *Mandated Completion*—Deliver apportionment counts to the President by December 31, 1990; provide data necessary for redistricting to states no later than April 1, 1991.

Most households will receive a mailed questionnaire about a week before Census Day. Others, depending on location, will have forms delivered by census takers. Roughly five out of six households will receive a short form; one in six will be asked to fill out a longer form.

The questions are basic. The *short form* asks about race, Hispanic origin, age, marital status, rent or own residence, number of rooms, etc. The *longer form* asked additional questions on ancestry, employment, education, income, type of housing, utilities, etc.

The population count for various areas of the nation is put to numerous uses, e.g., to determine how many seats a state will have in the US House of Representatives; to allocate seats in state legislatures, and to benefit specific populations and programs. Billions of dollars of federal funds are returned to states, local governments, and to American Indian reservations and Alaska Native villages. State funds are returned to cities and counties, with amounts based in part on population or housing data. Planners use census facts to determine where to locate hospitals, schools, day care and senior citizen centers. Various special programs receive funds based on census information. Businesses use the data for decision-making involving expansion and jobs.

For a number of reasons, about 2.2 million people were not counted in the last US Census (about 1 percent). Among minority populations, the undercount was higher—about 6 percent. To help assure a complete count in the 1990 Census, the Bureau of the Census began distributing information in June to US local and tribal government officials. Known as the Local Review Program, the effort encourages officials in the nation's 39,000 local and tribal governments to voluntarily check Census Bureau maps and block-by-block housing unit counts compiled from a master list created and updated throughout the census period. Coupled with that mailout, approximately 470 half-day local review training sessions will be held throughout the US before the end of 1989.

For further information about the Local Review Program and other Census matters, contact: Chief, Decennial Planning Division, Bureau of the Census, Washington, DC 20233. Tel: Census Promotion Office, Information Service, Census '90, (301) 763-1990. Or, contact Census Bureau Regional Census Centers in: Atlanta, GA, Boston, MA, Charlotte, NC, Chicago, IL, Dallas, TX, Denver, CO, Detroit, MI, Kansas City, MO, Los Angeles, CA, New York, NY, Philadelphia, PA, San Francisco, CA, and Seattle, WA.