

*Purpuric Fever — Continued*

## Reference

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*Perspectives in Disease Prevention and Health Promotion***Leading Work-Related Diseases and Injuries — United States**

The National Institute for Occupational Safety and Health (NIOSH) has developed a suggested list of the leading work-related diseases and injuries (Table 1). The first four categories have been described previously (1-4); a discussion of the fifth category, "Cardiovascular Diseases," appears below.

**CARDIOVASCULAR DISEASES**

Cardiovascular diseases, including hypertensive disease (International Classification of Diseases 9th Revision [ICD] codes 401-405), ischemic heart disease (ICD codes 410-414), other forms of heart disease (ICD codes 420-429), and cerebrovascular disease (ICD codes 430-438), are responsible for more deaths in the United States each year than any other category of disease (5). In 1980, cardiovascular diseases claimed over 960,000 lives, with ischemic heart disease responsible for over 565,000 of these deaths (6). Although the rates of death from cardiovascular diseases have declined gradually over the last decade, coronary atherosclerosis and acute myocardial infarction remain the leading causes of death in the United States.

The role of occupation as a factor in cardiovascular disease is far from clear (7). Most investigators believe that personal risk factors, such as cigarette smoking, blood pressure, diet, personality, and heredity, are more important than environmental factors (8). Specific data are sparse on the role of occupational factors. Nevertheless, some occupational factors are clearly associated with heart diseases, and evidence on other factors is accumulating (9). Be-

**Table 1. The 10 leading work-related diseases and injuries — United States, 1982\***

1. Occupational lung diseases: asbestosis, byssinosis, silicosis, coal workers' pneumoconiosis, lung cancer, occupational asthma	6. Disorders of reproduction: infertility, spontaneous abortion, teratogenesis
2. Musculoskeletal injuries: disorders of the back, trunk, upper extremity, neck, lower extremity; traumatically induced Raynaud's phenomenon	7. Neurotoxic disorders: peripheral neuropathy, toxic encephalitis, psychoses, extreme personality changes (exposure-related)
3. Occupational cancers (other than lung): leukemia; mesothelioma; cancers of the bladder, nose, and liver	8. Noise-induced loss of hearing
4. Severe occupational traumatic injuries: amputations, fractures, eye loss, lacerations, and traumatic deaths	9. Dermatologic conditions: dermatoses, burns (scaldings), chemical burns, contusions (abrasions)
5. Cardiovascular diseases: hypertension, coronary artery disease, acute myocardial infarction	10. Psychologic disorders: neuroses, personality disorders, alcoholism, drug dependency

\*The conditions listed under each category are to be viewed as *selected examples*, not comprehensive definitions of the category.

*Leading Work-Related Diseases and Injuries — Continued*

cause heart diseases are still so prevalent in the United States, identifying and preventing occupational factors that result in even a small increase in the relative risk of cardiovascular disease would involve large numbers of persons. Thus, preventing any occupational contribution to this problem would be an important public health measure.

In 1978, an ad hoc task force was formed by the American Heart Association to review the data regarding the environmental impact on cardiovascular disease (8). Its report, "The Impact of the Environment on Cardiovascular Disease," was published in 1981. The task force identified and reviewed six environmental factors that have potential impact on cardiovascular health: water hardness; trace elements; inhalant occupational exposures; carbon monoxide; noise and radiofrequency; and physical and psychosocial stress. The workplace is a specific source of potential exposure for all but the first.

**Metals, Dusts, Trace Elements.** The development of congestive heart failure that results from restrictive lung disease (cor pulmonale) has been observed in studies of occupational respiratory diseases, such as chronic beryllium disease and silicosis. Other metals, such as antimony, cobalt, and lead, have been implicated as possible causes of cardiovascular diseases.

**Occupational Inhalants and Other Chemical Exposures.** These include:

1. **Carbon monoxide:** Carbon monoxide decreases the oxygen-carrying capacity of hemoglobin and thus reduces the oxygen supply available to heart muscle and other tissues. In persons with preexisting coronary artery disease, occupational exposures to carbon monoxide may precipitate acute cardiovascular events, such as untoward changes in cardiac rhythm. In animal studies, life-threatening arrhythmias, such as ventricular tachycardia and ventricular fibrillation, have been observed in response to exposures to carbon monoxide that produced a carboxyhemoglobin concentration of 9% and above.

In one study of workers, short-term exposure to carbon monoxide at levels within the current Occupational Safety and Health Administration permissible exposure limit (50 ppm) was associated with decreased exercise tolerance and electrocardiographic evidence of myocardial ischemia. In another study among Finnish foundry workers exposed to carbon monoxide, the overall prevalence of angina pectoris was increased; this was most pronounced among workers who also smoked. Among British steelworkers, investigators found end-of-shift carboxyhemoglobin saturations substantially higher among blast furnace workers than among steelworkers in other jobs. This was observed for both smoking and nonsmoking employees.

2. **Carbon disulfide.** Carbon disulfide, a widely used solvent, has been shown to increase the risk of cardiovascular disorders, including coronary artery disease and hypertension, in both epidemiologic and experimental studies. It has also been shown to pose a significant risk for coronary death (10). The atherogenic potential of carbon disulfide involves both cerebrovascular and cardiovascular systems.
3. **Halogenated hydrocarbons.** Acute exposures to many common industrial solvents (e.g., chloroform, trichloroethylene) and fluorocarbon aerosol propellants have precipitated sudden death probably due to cardiac arrhythmias in workers exposed at high levels. Other common aerosols or solvents may be arrhythmogenic at concentrations permitted by current occupational exposure standards. A recent study of pathologists exposed to monochlorodifluoromethane (a fluorocarbon aerosol propellant) in hospitals showed an increased incidence of "palpitations" at levels of exposure far below the recommended standard.
4. **Nitroglycerin and nitrates.** Workers exposed to nitroglycerin and nitrates during the manufacture of explosives experienced increased risk of cardiac chest pain, myocardial infarction, and sudden death, particularly after a period of time away from exposure. The mechanism is thought to be "rebound vasospasm" as a consequence of withdrawal from exposure.

*Leading Work-Related Diseases and Injuries – Continued*

**Noise.** Tens of millions of workers are exposed to high levels of sustained and/or intermittent noise in the workplace. A number of studies have demonstrated that single exposures to noise cause transient increases in blood pressure. Chronic exposure to occupational noise has also been associated with sustained increases in blood pressure, particularly in workers with noise-induced hearing loss (11,12). Increases in serum cholesterol and changes in circulating hormones have been observed in humans in association with noise. In studies of animals, abnormalities in platelet aggregation have been documented following exposure to noise.

**Psychosocial Stress.** Stress has long been thought to adversely affect the cardiovascular system (13). A relationship between psychologic factors and cardiovascular disease is supported by the correlation between "Type A personality" and such disorders. A 1976 assessment suggested that "work-overload," role conflicts, and thwarted career goals were related to evidence of cardiovascular disease. A prospective evaluation of health changes among air traffic controllers, published in 1978, showed an increased prevalence of hypertension among controllers, attributed by the authors to difficulties in coping with working conditions.

An updated analysis of the Framingham heart study in 1980 (14,15), indicated that rates of coronary heart disease were nearly twofold greater among women employed in clerical jobs than among housewives. Significant predictors of the risk of coronary heart disease included a "nonsupportive supervisor" and decreased job mobility. Occupation may also affect the risk of cardiovascular disease in a spouse. Men whose wives worked in white collar jobs were observed to experience heart disease at a rate three times greater than men whose wives worked in clerical or blue collar jobs or were housewives. Similarly, men appeared to have a higher risk of cardiovascular disease if they had well-educated, working wives who reported nonsupportive supervisors or few opportunities for job promotion. These and similar results suggest that adjustments to the conflicting demands of job and family may be important factors in the risk of cardiovascular disease.

Recent evaluations of data from a large random sample of the Swedish working male population (16), and from other surveys, also suggest that certain working conditions, such as limited autonomy and heavy workloads, are associated with clinical indicators of coronary heart disease.

Epidemiologic studies are clearly needed to define the significance of these and other occupational stress factors in the etiology of cardiovascular diseases. Such physical stresses as noise, vibration, and heat also merit investigation for possible interaction with the psychologic risk factors of cardiovascular disease.

*Reported by Div of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, CDC.*

**Editorial Note:** Because cardiovascular diseases are so prevalent, they clearly affect large numbers of workers in the United States. A proportion of these diseases are caused or aggravated by risk factors in the work environment. NIOSH is currently collecting epidemiologic data to properly evaluate the role of the workplace in these diseases.

Since some occupational exposures clearly contribute to the morbidity and mortality attributable to cardiovascular diseases, further epidemiologic research is essential to define the extent of their occupational role and to define etiologic mechanisms. In addition, since cardiovascular diseases cause so much mortality in the United States, preventing even a small increase in relative risk due to occupational exposures would have major consequences to the public health. That mortality from cardiovascular disease has declined markedly in recent years indicates that these diseases are preventable.

The workplace is an important focus for efforts to prevent cardiovascular disease because: (1) it is the source of some preventable environmental exposures and psychosocial stresses that adversely affect cardiovascular health; (2) it offers unique opportunities for health promotion activities that encourage workers to improve their personal health behaviors (e.g., smoking

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cessation, appropriate exercise, and sound nutrition) and; (3) it provides an advantageous setting for delivering preventive services, such as screening for hypertension.

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\*Additional references are available on request from the National Institute for Occupational Safety and Health, CDC.

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**TABLE I. Summary—cases of specified notifiable diseases, United States**

Disease	16th Week Ending			Cumulative, 16th Week Ending		
	Apr. 20, 1985	Apr. 21, 1984	Median 1980-1984	Apr. 20, 1985	Apr. 21, 1984	Median 1980-1984
Acquired Immunodeficiency Syndrome (AIDS)	158	88	N	2,041	1,095	N
Aseptic meningitis	41	63	63	1,066	1,228	1,228
Encephalitis: Primary (arthropod-borne & unspec)	10	21	14	271	250	250
Post-infectious	2	4	4	40	31	29
Gonorrhea: Civilian	15,279	15,630	17,485	239,292	248,229	283,524
Military	252	358	484	5,485	6,224	8,203
Hepatitis: Type A	445	423	423	6,458	6,463	7,169
Type B	463	493	412	7,535	7,539	6,248
Non A, Non B	88	73	N	1,600	1,086	N
Unspecified	114	115	166	1,594	1,436	2,607
Legionellosis	10	11	N	160	152	N
Leprosy	15	4	4	107	62	62
Malaria	15	26	23	199	206	231
Measles: Total*	94	128	88	803	878	878
Indigenous	79	116	N	600	773	N
Imported	15	12	N	203	105	N
Meningococcal infections: Total	52	72	81	935	1,104	1,104
Civilian	51	71	81	934	1,102	1,102
Military	1	1	-	1	2	5
Mumps	82	93	93	1,275	1,156	1,708
Pertussis	25	118	21	387	651	328
Rubella (German measles)	5	16	68	122	172	799
Syphilis (Primary & Secondary): Civilian	490	552	564	7,551	8,705	9,335
Military	3	6	6	56	102	115
Toxic Shock syndrome	11	16	N	110	144	N
Tuberculosis	476	432	569	5,857	6,159	7,396
Tularemia	-	-	2	24	19	31
Typhoid fever	5	10	6	77	99	114
Typhus fever, tick-borne (RMSF)	1	8	8	14	30	26
Rabies, animal	71	126	160	1,366	1,467	1,752

**TABLE II. Notifiable diseases of low frequency, United States**

	Cum 1985		Cum 1985
Anthrax	-	Plague	-
Botulism: Foodborne	1	Poliomyelitis: Total	1
Infant (Calif. 3)	13	Paralytic	1
Other	-	Psittacosis (Calif. 1)	42
Brucellosis (Md. 1, Okla. 2)	26	Rabies, human	-
Cholera	-	Tetanus (Upstate N.Y. 1, Mich. 1)	15
Congenital rubella syndrome	-	Trichinosis (Upstate N.Y. 1, Calif. 2, Alaska 2)	28
Diphtheria (Colo. 1)	1	Typhus fever, flea-borne (endemic, murine)	3
Leptospirosis	8		

\*Twelve of the 94 reported cases for this week were imported from a foreign country or can be directly traceable to a known internationally imported case within two generations.

# MMWR

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### Epidemiologic Notes and Reports

#### Preliminary Report: Epidemic Fatal Purpuric Fever Among Children — Brazil

From October 14, to December 15, 1984, an outbreak of an unusual illness occurred in a small town (population 20,000) in the state of São Paulo, Brazil. Ten children, aged 3 months to 8 years, developed acute onset of high fever associated with vomiting and abdominal pain. Within 12-48 hours after onset of fever, these children developed purpura, followed by vascular collapse and necrosis of peripheral tissues. All 10 children died.

In all cases, laboratory examinations demonstrated only nonspecific findings, and cerebrospinal fluid (CSF) examinations did not suggest meningitis. White blood cell counts, elevated initially, were depressed immediately before death; hematocrits were normal; and thrombocytopenia was present. Bacterial cultures were negative, although antibiotics had been administered before obtaining cultures in most, if not all, instances. Autopsy examination characteristically demonstrated cerebral edema without meningitis, adrenal hemorrhage, lymphocyte depletion in lymphoid tissue, early fatty changes in the liver, fibrin deposition in small blood vessels, and changes consistent with acute shock in other organs. No organisms were visualized on microscopic examination by Gram or Dieterle staining.

At the same time the outbreak was occurring, another group of children in the same town developed what may have been a milder form of the same illness consisting of high fever, vomiting, and abdominal pain without progression to purpura or shock. No other illnesses were occurring in the town at above background level; no cases of meningitis occurred during this period; and no increase in any illnesses among older children or adults was noted.

The parents of children who died were extensively interviewed. No evidence was uncovered to suggest a common toxic exposure or preceding vaccination and no secondary transmission of the illness occurred. One consistent finding was noted—in most instances, a self-limited purulent conjunctivitis had preceded the onset of other symptoms by 3-15 days. A case-control study confirmed that children who died or children with suspected disease were significantly more likely than control children to have had conjunctivitis during the time of the outbreak (18/20 cases and suspected cases, compared with 9/20 controls [ $p = 0.006$ ]). A systematic survey of households with children 10 years of age or under documented that a small epidemic of purulent conjunctivitis, distinguishable from previous epidemics of viral hemorrhagic conjunctivitis, had occurred in the low income peripheral sections of the town during October and November. *Haemophilus aegyptius* was the most common organism isolated from conjunctival cultures taken from children with purulent conjunctivitis during this time. No cultures of conjunctivitis were obtained from children who developed severe illness; however, *H. aegyptius* was isolated from a nonseptically obtained skin scraping of a petechia from one case-patient.