Neisseria gonorrhoeae — *Continued*

furoxime. CDC treatment guidelines for PPNG infections provide the recommended schedules for these antimicrobials and emphasize the importance of the immediate use of spectinomycin as primary therapy for gonorrhea cases when treatment failures are suspected (*7*).

Since 1975, gonorrhea has generally declined in the United States (*8*). PPNG increased dramatically between 1976 and 1982 but decreased in 1983 (*8*). Unfortunately, cases of CMRNG have been reported with increasing frequency since the North Carolina outbreak. Because the extent and prevalence of CMRNG infections are not yet fully understood, screening of all β-lactamase negative (nonpenicillinase-producing) primary treatment failure gonococcal isolates for penicillin susceptibility (*1*) is encouraged at the local and state levels to improve surveillance and guide appropriate therapy. Screening at the community level should be most cost-effective, since the majority of these CMRNG strains are equally resistant to tetracycline, thereby preventing unnecessary and usually ineffective retreatment with a tetracycline. Because of high secondary treatment failure rates with tetracycline, tetracycline should not be used as the drug of choice for either PPNG or CMRNG infections that have failed primary therapy with penicillin or ampicillin. Spectinomycin, cefoxitin, or cefotaxime should be used to treat CMRNG infections at dosages recommended for PPNG (*7*).

More active surveillance for these CMRNG infections will be required to determine their accurate prevalence, and support control activities.

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Fatalities from Occupational Heat Exposure

Presented below are two of several fatalities from occupational heat stroke reported to the National Institute for Occupational Safety and Health (NIOSH) since 1977.

Indiana: In July 1980, a 24-year-old white male, who was employed at a surface coal mine, collapsed and later died after performing heavy labor in a hot environment. The worker, 5 feet 9 inches tall and weighing about 200 pounds, had been employed at the mine for 1½ weeks. On the day of the reported incident, he was assigned to load 40-pound bags of explosives into vertically drilled holes in preparation for blasting the material overlying the coal seam. He began work at 6:00 a.m., and at 3:40 p.m., informed a co-worker that he did not feel well. He walked about 50 yards to a shady area and collapsed. The outdoor dry bulb temperature was 39.4 C (103 F).

The worker was moved to a nearby hospital where his rectal temperature registered 42.2 C (108 F). By the time he was transferred to the intensive care unit (ICU), his temperature exceeded 43.3 C (110 F). He was treated with an ice pack and intravenous fluids but died at 6:30 p.m. The autopsy report listed systemic hyperthermia with extreme generalized dilation of capillaries (cardiovascular shock) and cerebral edema as the immediate causes of death.

Occupational Heat Exposure — Continued

Wisconsin: In September 1981, a 39-year-old black male, 5 feet 7 inches tall and weighing 165 pounds, was employed as a furnace attendant at an aluminum foundry. He had worked at the foundry for 2 weeks and was responsible for turning on and attending a furnace used to melt aluminum. On the afternoon of the reported incident, he had pressed the wrong button and accidentally spilled molten aluminum on the floor. He spent about 15 minutes removing the spill and wore a silver reflective suit for protection against the radiant heat emanating from the metal. The outdoor dry bulb temperature was 28.3 C (83 F), and the worksite temperature was about 29.4 C (84 F); the estimated temperature of the molten aluminum in the furnace was 982.2 C (1,800 F).

After removing the spilled material, the worker described the accident to his supervisor and, still wearing the suit, left the workplace without explanation. He was discovered 15 minutes later having seizures in the foundry parking lot. Paramedics transported him to a hospital at 5:40 p.m.; on arrival, his body temperature was 41.7 C (107 F). Medication controlled the seizures, but he remained comatose. He was treated with rubbing alcohol and an ice pack, and at 7:00 p.m., when his body temperature was 35.6 C (96 F), he was placed on a hyperthermic machine in the ICU. He began bleeding from the rectum at 9:30 p.m., and fresh, frozen plasma was administered. The bleeding apparently stopped but then recurred with hematuria. He died the next day at 9:30 a.m. in cardiac arrest. The autopsy report listed the causes of death as hyperthermia, disseminated intravascular coagulation, and coronary arteriosclerosis.

The worker had a history of treatment for alcoholism and reportedly had been drinking heavily in the days before his death; however, at the time of hospitalization, he had no alcohol in his blood. Four days before the heatstroke, he had severely lacerated his toes in a lawnmower accident and was treated with antibiotics and tetanus toxoid.

Reported by Div of Respiratory Disease Studies, Div of Biomedical and Behavioral Science, National Institute for Occupational Safety and Health, CDC.

Editorial Note: Illness and death from environmental heat are important public health problems (*1*). This is especially true in the occupational setting when workers performing physical labor outdoors are exposed to higher-than-normal ambient temperatures and when such temperatures have an additive effect on heat generated by the jobs themselves. The fatalities reported here illustrate, in both outdoor and indoor settings, the circumstances that may lead to heatstroke and, subsequently, to death.

Occupational heat-related conditions include heat cramps, heat exhaustion, dehydration, and skin disorders. In addition, the risk of unintentional injuries increases substantially with exposure to heat stress (*2*). An estimated six million workers in the United States may be exposed to occupational heat stress. Estimates of deaths and illnesses associated with occupational heat exposures are difficult to obtain, because worksite conditions and occupation are usually not listed on hospital records or death certificates; moreover, heatstroke may not be recognized as the primary cause of illness or death. However, for 1973-1976, annual reports from the California Department of Health Services alone show seven fatalities among 1,128 acute occupational heat-related illnesses (*3*). About 10%-15% of these patients required hospitalization, and an additional 40% were absent from work for varying periods after their illnesses; the remainder returned to work after medical treatment.

The health status of a worker is important in determining the response to heat exposure (*4*). Certain preexisting conditions can render a person more susceptible to heatstroke; these include obesity, drug abuse, alcoholism, acute or chronic illnesses, fatigue, poor physical condition, overeating, use of anticholinergic and certain psychotropic drugs, lack of sleep, and lack of acclimatization (*5*). The first worker described here was moderately obese and in poor physical condition; the second had a history of treatment for alcoholism and may have been affected by the wound and the medication he received 4 days before his death.

Occupational Heat Exposure — Continued

In 1969, an international panel of scientists convened by the World Health Organization recommended keeping a worker's deep body temperature at or below 38 C (100.4 F) to prevent heat illnesses (*6*). In response to this, NIOSH developed in 1972 a Criteria Document for Occupational Exposure to Hot Environments, which recommended the following preventive measures (*7*): (1) acclimatizing new workers and workers returning from vacation or absence because of illness; (2) implementing a work/rest regimen matched to the severity of the workers' heat exposure. (The Threshold Limit Value for Heat Stress adopted by the American Conference of Governmental Industrial Hygienists can be used as a guide to establish a suitable work/rest regimen [*8*]); (3) scheduling hot operations for the coolest part of the day; (4) making drinking water and salt readily available to replace the water and salt lost by sweating; (5) making protective clothing available to workers, as appropriate; (6) reducing environmental heat by engineering controls; (7) monitoring environmental heat at the job site; (8) performing pre-employment and periodic medical examinations to define those at increased risk; and (9) instructing workers and supervisors about preventive measures and early recognition of the symptoms of heat-related disorders.

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Current Trends

Tuberculosis — United States, 1983

In 1983, 23,846 cases of tuberculosis were reported to CDC, for a rate of 10.2 cases per 100,000 population. Compared with 1982, this represents a 6.6% decrease in the number of cases reported and a decline of 7.3% in the rate.

Rates for the 50 states ranged from 23.1/100,000 in Hawaii to 1.3/100,000 in North Dakota (Table 3). The rate increased in 13 states, remained unchanged in one, and decreased in 36.

The rate among persons living in 56 cities with populations of 250,000 or more was 21.2/100,000—more than twice the national rate (Table 4). Urban rates ranged from 58.4/100,000 in Miami, Florida, to 2.5/100,000 in Toledo, Ohio. Eight cities had rates at least three times the national rate: Miami, Florida; Newark, New Jersey; Atlanta, Georgia; San Francisco, California; Tampa, Florida; Honolulu, Hawaii; Washington, D.C.; and Oakland, California.

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MORBIDITY AND MORTALITY WEEKLY REPORT

Recommendation of the Immunization Practices Advisory Committee (ACIP)

Rabies Prevention — United States, 1984

*These revised recommendations of the Immunization Practices Advisory Committee (ACIP) on rabies prevention update the previous recommendations (*MMWR *1980;29: 65-72,277-80) to reflect the current status of rabies and antirabies biologics in the United States. For assistance on problems or questions about rabies prophylaxis, call local or state health departments.**

INTRODUCTION

Although rabies rarely affects humans in the United States, every year, approximately 25,000 persons receive rabies prophylaxis. Appropriate managment of those who may have been exposed to rabies infection depends on the interpretation of the risk of infection and the efficacy and risk of prophylactic treatment. All available methods of systemic prophylactic treatment are complicated by instances of adverse reactions. These are rarely severe. Decisions on management must be made immediately; the longer treatment is postponed, the less likely it is to be effective.

Data on the efficacy of active and passive immunization after rabies exposure have come from both human and animal studies. Evidence from laboratory and field experience in many areas of the world indicates that postexposure prophylaxis combining local wound treatment, vaccine, and rabies immune globulin, is uniformly effective when appropriately used. However, rabies has occasionally developed in humans who had received postexposure antirabies prophylaxis with vaccine alone.

In the United States, rabies in humans has decreased from an average of 22 cases per year in 1946-1950 to zero to five cases per year since 1960. The number of rabies cases among domestic animals has decreased similarly. In 1946, more than 8,000 rabies cases were reported among dogs; 153 cases were reported in 1982. Thus, the likelihood of human exposure to rabies in domestic animals has decreased greatly, although bites by dogs and cats continue to be the principal reasons given for antirabies treatments.

The disease in wildlife—especially skunks, foxes, raccoons, and bats—has become more prevalent in recent years, accounting for approximately 85% of all reported cases of animal rabies every year since 1976. Wild animals now constitute the most important potential source of infection for both humans and domestic animals in the United States. Rabies among animals is present throughout the United States; only Hawaii remains consistently rabies-free.

Four of the six rabies fatalities in U.S. citizens occurring between 1980 and 1983 were related to exposure to rabid dogs outside the United States. In much of the world, including

*If these are unavailable, call the Division of Viral Diseases, Center for Infectious Diseases, CDC ([404] 329-3095 during working hours, or [404] 329-2888 nights, weekends, and holidays).