Intestinal Perforation — Continued

Editorial Note: Nematodes of the genus *Eustrongylides* (Family Dioctophymidae Railliet, 1915) are parasitic as adults in the gastrointestinal tract of fish-eating birds and as larvae in the connective tissue or body cavity of freshwater fish (*1*). Amphibians, reptiles, and mammals (rarely) may become infected with larval *Eustrongylides* spp. and may play an ecological role as paratenic or transport hosts. Moreover, extensive larval migration in accidentally and experimentally infected reptilian, amphibian, and avian hosts has been observed and has sometimes been associated with high mortality (*1-3*), suggesting a possible pathologic role for *Eustrongylides* spp. However, no human infections have been reported to CDC.† Although data are incomplete, infection by larval *Eustrongylides* spp. is widespread and common in numerous species of freshwater fish. The high rates of infection for minnows (*Fundulus* spp.) reported here and earlier (*3*) may indicate a high degree of risk for persons who choose to eat these fish without cooking them first.

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†The USDA National Helminthological Collection contains a single larval specimen obtained from a human (*2*).

Silo-Filler's Disease in Rural New York

On September 18, 1981, at a farm in Mohawk, New York, a 39-year-old farmhand was overcome while climbing up the chute of a recently filled concrete stave silo and later died of presumed silo-filler's disease. The case report follows.

In the preceding 10 days, the farm owner had filled this silo with Sudex grass and chopped corn silage. On September 18, he asked a farmhand to climb up the unloading chute inside the silo and toss out fresh silage. When the farmhand climbed the chute, he became short of breath and confused and had to descend. He made a second attempt but again had to climb down and was noted to be cyanotic, pale, and diaphoretic.

At a local hospital, the examining physician noted cyanosis and respiratory distress; blood pressure of 84/60; pulse, 128; respiration, 32; and temperature, 37.5 C (99.5 F). The patient had wheezes and crackles on auscultation of his chest but no signs of consolidation; after asthma was diagnosed, he was treated with epinephrine, intravenous aminophylline, and steroids. White blood count was 31,000; hematocrit, 57.8%; and hemoglobin, 18.6 gm. Arterial blood gas examination while on 2 liters of nasal oxygen showed pH 7.35; $PaCO_2$, 32 mm Hg; PaO 45 mm Hg; and a calculated bicarbonate of 17.6 mEq/L. An electrocardiogram showed a sinus tachycardia, and a plain chest radiograph disclosed extensive fluffy bilateral infiltrates.

The patient was moved to the intensive care unit, where a tentative diagnosis of pneumonia was made. He became agitated, would not wear an oxygen mask, and remained in shock. Five hours after admission, he experienced cardiopulmonary arrest and died despite vigorous efforts to resuscitate him.

Post-mortem examination the next day showed grossly edematous lungs with pleural effusions (200 ml) on both sides; the right lung weighed 900 gm, and the left weighed 1,000 gm.

Silo-Filler's Disease – Continued

Microscopy of the lungs showed alveoli flooded with proteinaceous material; the alveolar walls were intact. No bacteria, fungi, or evidence of viral disease was found. Early bronchiolitis was present; no evidence of asthma could be seen. There were no granulomas or hyaline membranes.

An investigation at the farm 2 weeks later failed to uncover any problem with the corn silage, which was still being unloaded. The cows were eating normally and producing the usual amounts of milk. The farmer reported that, following his farmhand's illness, he had turned on the silo blower and sent another worker up to toss out corn; no ill effects had occurred.

Several factors support the diagnosis of silo-filler's disease, an illness caused by the inhalation of nitrogen oxides: i.e., rapid onset of symptoms following a recent filling of the silo and histology classic for toxic exposure.

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Editorial Note: The case outlined above is typical for massive exposure to nitrogen oxides. Silo-filler's disease represents an occupational hazard associated with ensiled crops. Laboratory studies have shown that toxic levels of NO, NO_2, and N_2O_4 are regularly produced in silos (*1,2*). Because these oxides are dense, they tend to settle in the chute and around the base of the silo, and exposure often occurs without anyone's entering the silo. Although NO_2 is brown and has an odor, N_2O_4 is colorless and odorless, and exposure can occur without warning (*3*). If undetected by smell or sight, the potent nitrogen oxides may be inhaled deep into the lungs, where contact with the mucosal moisture produces nitric acid, which burns the airways, respiratory bronchioles, and alveoli. In fatal exposures, vascular collapse and the outpouring of serum rapidly produce shock and death. In another clinical course associated with silo-filler's disease, exposure causes cough and chest tightness. Although these conditions clear spontaneously, illness may return in three weeks with severe symptoms of fever, chills, and shortness of breath. Biopsies show a bronchiolitis obliterans with granuloma formation. This second pattern appears to respond to steroids (*4*).

Fatal and serious exposures to nitrogen oxides are not unique to farming but have been reported in association with arc and acetylene welding (*4*), burning cellulose nitrate (*5*), and dynamite blasting (*6*). Diesel fumes, furnace gases, and chemical processes involving the generation of NO_2 (*3*) are also potentially dangerous.

It is possible to prevent this type of exposure in the farm industry if farmers are aware of the following dangers and use the suggested safety measures: 1) Silos begin to produce NO_2 within 4 hours after filling, and no one should enter or come in close contact with a recently filled silo. 2) Some crops (oats, corn) produce more NO_2 than others, and heavily fertilized crops, cloudy conditions, and rain raise the risk of NO_2 production. 3) Although NO_2 levels are generally low and within a safe range after 2 weeks, dangerous amounts may remain for months if the silo has not been opened (*7*). 4) If possible, enclosed areas should be ventilated for 20 minutes before anyone enters, and individuals should be equipped with a full-face mask and an air supply.

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Ciguatera Fish Poisoning — Bahamas, Miami

On March 6, 1982, the U.S. Coast Guard in Miami, Florida, received a request for medical assistance from an Italian freighter located in waters off Freeport, Bahamas. Numerous crew members were ill with nausea, vomiting, and muscle weakness and required medical evacuation for hospitalization and treatment.

A total of 14 ill crew members were airlifted to three Florida hospitals. Three were seen in emergency rooms and later released. Eleven were hospitalized; seven required admission to intensive care units. All patients were Italian males, age 24-40 years; symptoms included diarrhea—12 patients (86%), vomiting—11 (79%), paresthesias—11 (79%), hypotension—10 (71%), peripheral muscular weakness—9 (65%), nausea—8 (57%), abdominal cramping—6 (43%), pruritis—4 (29%), and peripheral numbness—2 (14%). These findings were consistent with ciguatera fish poisoning, and an epidemiologic investigation was initiated.

The ship employed 26 crew members and is permanently based near Freeport, where it ferries petroleum products ashore from large tankers. On March 4, a crew member caught a 25-pound barracuda while fishing from the ship. On March 6, 14 crew members cooked and ate the barracuda; all became ill within 6 hours. None of the 12 crew members who did not eat the barracuda became ill. Six of the ill crew members reported becoming sick 45 minutes to 6 hours after the implicated meal (median: 2.5 hours). All 14 crew members eventually recovered without sequelae and returned to work. Median length of hospital stay was 6 days.

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Editorial Note: Ciguatera is a human intoxication syndrome associated with the consumption of marine tropical reef fishes. Although recent surveys indicate that poisonings are relatively uncommon in Florida (*1,2*), one investigator recorded 280 intoxications from January 1978 to June 1980 (*2*).

The ichthyosarcotoxins are thought to be accumulated through the food chain, the toxins being produced by microalgae known as dinoflagellates (*3,4*). The toxins are lipid-soluble and appear to accumulate in the flesh, fatty tissue, and viscera of large predatory species of fish, such as barracuda, grouper, and snapper (*5,6*). The isolation, purification, and characterization of the suspected toxins have been hampered by limited availability of authentic ciguatoxic fish, lack of a specific sensitive assay, and the low concentration and heterogeneity of toxins present in specimens.

The assessment of toxicity most often used is the mouse bio-assay. Based on signs elicited following intraperitoneal (IP) injection, it includes, but is not limited to inactivity, diarrhea, labored breathing, cyanosis, piloerection, tremors, paralysis, and staggering gait. Death occurs when the injection is given in higher doses, with a lethal dose, 50% kill (LD_{50}), of

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MMWR

MORBIDITY AND MORTALITY WEEKLY REPORT

Surveillance Summary

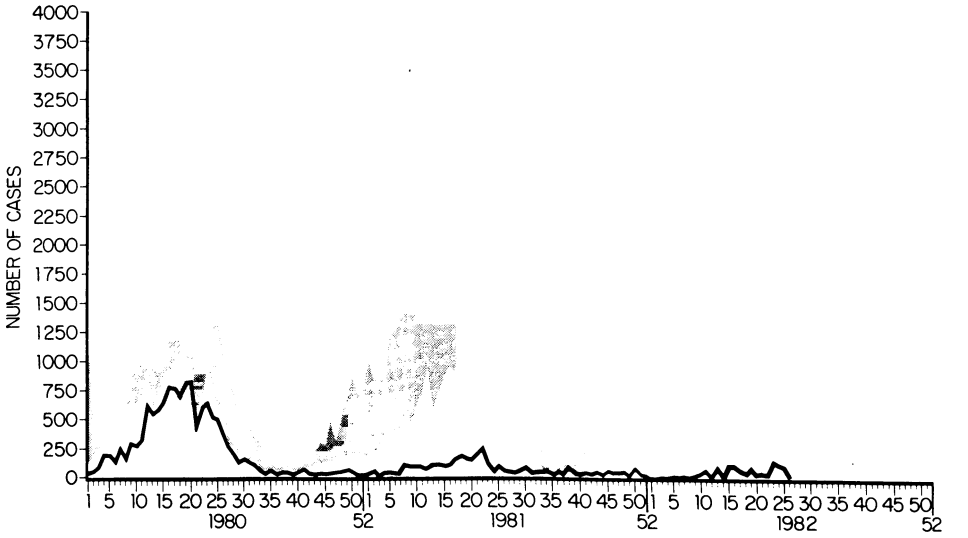
Measles — United States, First 26 Weeks, 1982

A total of 895 measles cases were reported in the United States during the first 26 weeks of 1982, a record low for the first 6 months of any year and a decrease of 60.6% from the 2,270 cases reported during the same period last year. Fewer than 100 measles cases in any 1 week were reported for the first 26 weeks of 1982, and record low numbers of cases were reported for 25 of those weeks. Fewer than 100 cases of measles per week have now been reported for 55 consecutive weeks.

As in previous years, incidence of reported measles peaked in late spring (Figure 1). However, the peak was considerably lower than in 1980 and 1981, years in which measles incidence had already declined to record lows.

The overall incidence for the United States during the first 26 weeks of 1982 was 0.4/100,000 total population. Only two states reported measles incidences of ≥1/100,000. In contrast, seven states in 1981 and 30 states in 1980 reported such rates for the first 26 weeks. The highest measles incidence in the first 26 weeks of 1982 was reported from Cali-

FIGURE 1. Reported measles cases,* United States, January 1980-June 1982



*Shaded area represents maximum and minimum weekly values during 5-year period, 1975-1979.
Source: MMWR weekly reports.
CDC, CPS, IM