Q Fever—
California, Georgia, Pennsylvania, and Tennessee, 2000-2001

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Q FEVER IS A ZOONOTIC DISEASE CAUSED by the bacterium Coxiella burnetii. The most common reservoirs are domesticated ruminants, primarily cattle, sheep, and goats. Humans acquire Q fever typically by inhaling aerosols or contaminated dusts derived from infected animals or animal products. Its highly infectious nature and aerosol route of transmission make C. burnetii a possible agent of bioterrorism. Although up to 60% of initial infections are asymptomatic, acute disease can manifest as a relatively mild, self-limited febrile illness, or more moderately severe disease characterized by hepatitis or pneumonia. It manifests less commonly as myocarditis, pericarditis, and meningoencephalitis. Chronic Q fever occurs in <1% of infected patients, months or years after initial infection. Chronic disease manifests most commonly as a culture-negative endocarditis in patients with valvular heart disease. During 2000-2001, a total of 48 patients who met the case definition of Q fever were reported to CDC. This report describes the case investigations for six of these patients, which indicate that these persons acquired Q fever probably through direct or indirect contact with livestock. To enhance surveillance efforts, health-care providers should report cases of Q fever to state health departments.

California
In May 2001, a woman aged 56 years sought treatment from her health-care provider for fever (104°F [40°C]), hepatomegaly, and elevated liver enzymes (alkaline phosphatase 532 U/L [normal: 30-100 U/L], SGOT 178 U/L [normal: 9-25 U/L], and SGPT 149 U/L [normal: 7-30 U/L]). Acute cholecystitis was diagnosed, and a cholecystectomy was performed. After the procedure, the patient’s symptoms persisted, and she developed pain and partial paralysis of the left leg. Approximately 4 weeks after the woman sought treatment initially, a computed tomography (CT) scan of the patient’s chest revealed nonspecific interstitial lung disease. Serum samples obtained near the time of the CT scan and 6 weeks later were tested by an indirect immunofluorescence antibody (IFA) assay and demonstrated IgG antibodies reactive with C. burnetii phase II antigens at reciprocal titers of ≥1,024, confirming a diagnosis of Q fever. The patient’s husband, aged 62 years, also developed a nonspecific febrile illness 3 days after the onset of his wife’s illness; serum specimens obtained from him in June and July and tested by IFA demonstrated IgG antibodies reactive with C. burnetii phase II antigens at reciprocal titers of ≥1,024. Canvassing of the neighborhood by a public health nurse revealed that a next-door neighbor aged 76 years had a nonspecific febrile illness in April 2001. His serum was obtained in August and October and was tested by IFA; both specimens demonstrated IgG antibodies to C. burnetii phase II antigens at reciprocal titers of ≥1,024. The three patients were treated with doxycycline; their symptoms resolved within 2 weeks. A serum sample drawn from 14 cattle from these herds; two animals tested by IFA reacted with phase 1 or II antigens of C. burnetii at reciprocal antibody titers (16-32).

Georgia
In March 2001, a man aged 46 years sought treatment for acute onset of fever, chills, cough, and weight loss; influenza was diagnosed. The patient’s symptoms persisted, and after 2 weeks he sought further treatment at an emergency department, where influenza again was diagnosed, and he was referred to an infectious disease specialist. A serum sample was tested by IFA and reacted with C. burnetii phase II antigens at a reciprocal titer of ≥256. The patient was administered a 5-day course of the fluorquinolone gatifloxacin, and symptoms resolved within 2 weeks. A convalescent-phase serum sample obtained in April and tested by IFA demonstrated an IgG reciprocal antibody titer reactive with C. burnetii phase II antigens of ≥16,384.

The patient owned several dairy cows, but there had been no recent animal births on the premises. Two beef cattle herds of approximately 35 animals each were pastured across the road from the patient’s farm. Serum was drawn from 14 cattle from these herds; two animals tested by IFA reacted with phase 1 or II antigens of C. burnetii at reciprocal antibody titers (16-32).

Pennsylvania
In September 2000, a man aged 90 years sought treatment for fever (101.0°F [38.3°C]) and a 4-month history of malaise and weight loss after a cholecystectomy. The patient had elevated liver enzymes (alkaline phosphatase 181 U/L [normal: 45-115 U/L] and SGOT 51 U/L [normal: 1-40 U/L]). He was admitted to the hospital for diagnostic evaluation. In 1998, the patient had undergone aortic valve replacement for culture-negative endocarditis and valvular insufficiency. A serum sample drawn in November 2000 was tested by IFA and demonstrated IgG antibodies reactive with C. burnetii phase I antigens at a reciprocal titer of ≥524,288. Presence of C. burnetii was demonstrated in two animals each were pastured across the road from the patient’s farm. Serum was drawn from 14 cattle from these herds; two animals tested by IFA reacted with phase 1 or II antigens of C. burnetii at reciprocal antibody titers (16-32).
strated in the excised aortic heart valve tissue from 1998 when tested by immunochemical (IHC) staining at CDC. The patient was started on long-term doxycycline therapy in October 2000. Since electing to discontinue this therapy 1 year later, the patient has had two recurrences. He was admitted to the hospital in September 2002 for fever and hypotension.

The patient had owned and operated a cattle farm but had retired from farming 30 years previously. The patient’s relatives raised sheep and goats nearby, but the patient denied having contact with their animals. One relative, who raised sheep, was found to have an antibody titer reactive with *C. burnetii* phase I antigens but had not experienced illness.

Tennessee

In February 2001, a man aged 49 years was admitted to a hospital with a right lower-extremity embolism. The patient reported a 6-month history of intermittent fever, night sweats, fatigue, and arthralgias. A heart murmur had been diagnosed 4 months previously. On admission, he had a temperature of 99.2°F (37.3°C) and leukocytosis (white blood cell count of 14.3x10^9/L [normal: 4.5-11.0x10^9/L]). The embolism in his leg was removed surgically. An echocardiogram after hospital admission revealed a bicuspid aortic valve with moderate stenosis and severe regurgitation, and aortic valve replacement was performed. Microscopic examination of the excised valve revealed a vegetative growth, but no bacteria or fungi were detected by histopathology or routine cultures. Serum obtained 1 week after admission was tested by IFA and demonstrated IgG antibodies reactive with *C. burnetii* phase I antigens at a reciprocal titer of ≥512, and the patient was administered doxycycline and levofloxacin. CDC detected DNA of *C. burnetii* in the excised aortic valve by polymerase chain reaction (PCR). The embolus removed from the patient’s right leg tested positive for *C. burnetii* by IHC staining. The patient was discharged but was readmitted 10 days later for pericardial effusion with tamponade, which resolved after surgical intervention.

The patient owned one goat and a herd of approximately 100 cattle. In February 2000, the patient had been present at the stillbirth of one calf and the premature delivery and death of a second calf. Serum samples from 24 cattle in his herd were collected in July and tested for antibodies to *C. burnetii* by IFA; one animal had reactivity to phase I and II antigens at a reciprocal titer of 16.

Epidemiology, Diagnosis, Treatment, and Prevention of Q Fever Epidemiology

- Classified as a zoonotic disease.
- Contracted through exposure to infected ruminants (especially parturient goats, sheep, and cattle), with incubation time of 3-30 days.
- Distributed broadly throughout the United States.
- Transmitted primarily through inhalation of airborne bacteria.
- Highly infectious.
- Designated a possible bioterrorism agent.

Clinical Findings

- Up to 60% of infections are asymptomatic. Acute disease is characterized most frequently by: (1) high fever and headache; (2) pneumonia or hepatitis in approximately 60% of acutely ill persons; (3) infrequent acute manifestations including pericarditis, myocarditis, or meningoencephalitis.
- Chronic disease occurs in <1% of infected patients: (1) occurs predominantly in patients with underlying valvular heart disease, vascular aneurysms, or vascular grafts; (2) manifests primarily as culture-negative endocarditis, less commonly as vascular or osteoarticular infection.

Laboratory Testing

Diagnosis made by (1) demonstration of fourfold or greater increases in IgG or IgM class-specific testing of paired acute- and convalescentphase serum samples by immunofluorescence antibody; (2) elevated antibody response to *C. burnetii* phase I or II antigens; (3) detection of *C. burnetii* by polymerase chain reaction or immunochemical staining.

Treatment

- Acute disease: doxycycline 200 mg/day for 2-3 weeks.
- Acute disease in patients with valvular heart disease: doxycycline 200 mg/day plus hydroxychloroquine 600 mg/day, for 1 year; dosage of hydroxychloroquine adjusted to maintain plasma level at 1±0.2 µg/ml.
- Chronic: doxycycline and hydroxychloroquine, dosage as above, for 1.5-3 years; cessation of therapy determined by appropriate serologic profile.

Prevention

- Minimize or restrict exposures to livestock birthing areas.
- Dispose of birth products properly (e.g., incinerate placenta and aborted fetuses).
- Report all human cases to state health departments (Q fever is a nationally notifiable disease).

CDC Editorial Note: These cases demonstrate acute and chronic clinical characteristics of Q fever and indicate some of the risk factors for acquiring this disease (see sidebar). The bacterium *C. burnetii* is distributed widely in the United States, and human cases of Q fever have been reported from almost every state. Human infections are associated commonly with exposure to infected animals giving birth, especially ruminants such as sheep, cattle, and goats. Cats, dogs, wildlife, and birds also are associated occasionally with human infec-

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tion. Transmission to humans usually occurs by inhalation of droplets or windborne dust containing *C. burnetii*. The persons whose cases are described in this report acquired Q fever probably through exposure to infected livestock. Most of the six patients had occupational contact with livestock (e.g., farming); however, some of these cases demonstrate that persons need not work in a high-risk environment or have direct animal contact to become infected with *C. burnetii*.

In humans, the clinical presentation of Q fever varies widely. Acute Q fever might be characterized by a non-specific febrile illness, hepatitis, or pneumonia. Acute cholecystitis is not known to be associated with *C. burnetii* infection; however, the liver manifestations observed in some patients might resemble gall bladder disease. Although one person described in this report had a peripheral neuropathy after acute infection, such symptoms are uncommon. Chronic Q fever might manifest months to years after initial infection, most commonly as a culture-negative endocarditis. Persons with underlying heart valve defects or prosthetic valves are at increased risk for chronic Q fever endocarditis, which might occur in up to 40% of persons with valvular heart disease following acute Q fever. Health-care providers should be aware of the signs and symptoms of the disease and consider laboratory testing for Q fever in patients exhibiting prolonged fever, hepatitis, atypical pneumonia, or blood culture–negative endocarditis, particularly patients whose histories suggest contact with or exposure to sheep, goats, or cattle.

Q fever usually is diagnosed by evaluating paired acute- and convalescent-phase serum samples. In humans, the antibody response is directed against phase I and phase II antigens of *C. burnetii*. Patients with acute Q fever typically produce an antibody response primarily to *C. burnetii* phase II antigen; chronic *C. burnetii* infections typically elicit a higher antibody response to phase I antigens. A diagnosis of Q fever also can be confirmed by examining biopsies of affected organs by using PCR or IHC. Serologic tests may be conducted at commercial laboratories, several state health laboratories, or CDC. In animals, serologic tests for antibodies to *C. burnetii* are more difficult to interpret. Presence of antibodies might indicate previous infection with the organism but cannot be used to predict human risk.

For treatment of acute Q fever, doxycycline is the drug of choice. Initiation of therapy is warranted in patients with disease demonstrating clinical and epidemiologic features compatible with Q fever. Because antibiotic treatment is most effective during the early phase of the illness, treatment should not be withheld pending results of confirmatory laboratory antibody tests, which provide a retrospective diagnosis. For patients with pre-existing valvular disease, progression of acute disease to endocarditis is best prevented by combination long-term therapy with doxycycline and hydroxychloroquine. This regimen also is recommended for patients with active Q fever endocarditis. If the infection does not resolve with antibiotic therapy, the patient might require excision and replacement of the damaged heart valve; however, this will not necessarily ensure elimination of *C. burnetii*, and the new valve might fail if appropriate antimicrobial treatment is not initiated or is withdrawn prematurely.


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**References**

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*Confirmed Q fever: A clinically compatible case that is laboratory confirmed with one of the following: (1) a fourfold change in antibody tier to *C. burnetii* antigen by immunofluorescence antibody assay or complement fixation antibody test, (2) a positive polymerase chain reaction assay, (3) culture of *C. burnetii* from a clinical specimen, or (4) positive immunostaining of *C. burnetii* in tissue. Probable Q fever: a clinically compatible case with single supportive IgG of IgM titer as defined by the testing laboratory.

**Nonfatal Choking-Related Episodes Among Children—United States, 2001**

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1 table omitted

Food and nonfood substances can present a choking hazard for children, particularly younger children. During 2000, the latest year for which national mortality data were available, 160 children aged ≤14 years died from obstruction of the respiratory tract associated with inhaled or ingested foreign bodies (International Classification of Diseases, Tenth Revision, codes W79–W80); food and nonfood substances were associated with 41% and 59% of these deaths, respectively (CDC, unpublished data, 2002). To characterize nonfatal choking-related episodes in children treated in U.S. hospital emergency departments (EDs) during 2001, CDC analyzed data from the National Electronic Injury Surveillance System–All Injury Program (NEISS-AIP). This report summarizes the results of this analysis, which indicate that an estimated 17,537 children aged ≤14 years were treated in EDs for choking-related episodes in 2001. Many of these episodes were associated with candy/gum (19.0%) and coins (12.7%). Parents and caregivers...
should be aware of the types of foods and objects that pose a choking risk for children, become familiar with methods to reduce this risk, and be able to treat choking in children.

NEISS-AIP is operated by the U.S. Consumer Product Safety Commission and collects data on initial visits for all types and causes of injuries treated in U.S. EDs. NEISS-AIP data are drawn from a nationally representative subsample of 66 (out of 100) NEISS-AIP hospitals, which were selected as a stratified probability sample of hospitals with a minimum of six beds and a 24-hour ED in the United States and its territories. NEISS-AIP provides data on approximately 500,000 injury- and consumer product–related ED cases each year.

Cases in this report occurred among patients aged ≤14 years treated for unintentional, nonfatal choking-related episodes in which the external cause of injury was coded as inhalation or suffocation, or a brief narrative describing the episode included “choke,” “choke,” or “choking.” Patients were excluded if the episode was related to smoke inhalation, choking on secretions or vomitus, submersion injury, strangulation, breath-holding spell, exposure to a toxic or noxious substance, or poisoning. Because deaths are not captured completely by NEISS-AIP, children who were dead on arrival or who died in EDs also were excluded. The narratives were reviewed for all cases to classify, when possible, the food and nonfood substances associated with the choking episode.

Each case was assigned a sample weight based on the inverse probability of selection; these weights were added to provide national estimates of choking-related episodes. Estimates were based on weighted data for 526 children with choking-related episodes treated at NEISS-AIP hospital EDs during 2001. Confidence intervals (CIs) were calculated by using a direct variance estimation procedure that accounted for the sample weights and complex sample design. Rates were calculated by using 2001 U.S. Census Bureau population estimates.

In 2001, an estimated 17,537 (95% CI = 12,319–22,755) children aged ≤14 years were treated in EDs for choking-related episodes for a rate of 29.9 per 100,000 population (95% CI = 21.0–38.8). Rates were highest for infants aged <1 year (140.4) and decreased with age. The rate for boys (32.1) was similar to that for girls (27.3). Although the majority of patients were treated and released, 1,844 (10.5%; 95% CI = 3.1–18.0) were hospitalized or transferred to a facility with a higher level of care.

Of the 17,537 children treated in EDs, 10,438 (59.5%; 95% CI = 39.3–79.7%) were treated for choking on a food substance, 5,513 (31.4%; 95% CI = 18.0–44.9%) on a nonfood substance, and 1,586 (9.0%; 95% CI = 4.1–14.0%) on an undetermined substance. Of overall choking-related cases, 2,229 (12.7%; 95% CI = 9.0–20.4%) were associated with coins, and 3,325 (19.0%; 95% CI = 12.1–25.8%) were associated with candy/gum. Of episodes related to candy/gum, 2,153 (64.8%; 95% CI = 35.5–94.0%) were associated with hard candy, 419 (12.6%; 95% CI = 3.8–21.4%) with other specified types of candy (e.g., chocolate and gummy candy) and gum, and 752 (22.6%; 95% CI = 8.2–37.1%) with an unspecified candy.

Food and nonfood substances associated with choking-related episodes varied by age group. Food substances accounted for 2,355 (75.7%; 95% CI = 40.3–111.2%) choking-related episodes among children aged 5–14 years, 5,302 (58.4%; 95% CI = 37.8–78.9%) episodes among children aged 1–4 years, and 2,781 (52.1%; 95% CI = 30.7–73.4%) episodes among infants aged <1 year. Candy/gum was associated with approximately one fourth of choking-related episodes among children aged 5–14 years (860 [27.6%; 95% CI = 11.4–43.9%]) and those aged 1–4 years (2,223 [24.5%; 95% CI = 14.7–34.2%]). Coins accounted for 1,658 (18.2%; 95% CI = 5.8–30.7%) choking-related episodes among children aged 1–4 years.

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**CDC Editorial Note:** This report provides national estimates of nonfatal choking-related episodes in children aged ≤14 years. On the basis of national mortality data compared with estimates described in this report, for every choking-related death in this age group, an estimated 110 children were treated for choking-related episodes in U.S. hospital EDs. Children are at risk for infection in the respiratory tract and complications associated with lack of oxygen from airway obstruction, including permanent brain damage and death.

Several public health strategies can reduce the risk for choking in children, including public education, productsafety labeling, changes in product design, and the instruction of parents and caregivers in emergency preparedness for the early treatment of choking. Public education can increase the awareness of the problem, the items that present a choking hazard, the ages at which children are at highest risk, and the importance of adult supervision when young children are eating and playing. Product-safety labeling can inform consumers of potential choking dangers through age-appropriate labeling on toys and warnings on high-risk items (e.g., balloon packages and small balls). The design of some products has changed to reduce choking risks, such as eliminating small parts of toys designed for toddlers and nonfood toys packaged with food items. In addition, parents and caregivers can receive instruction on treating choking from health-care providers or take courses that teach basic lifesaving skills and first aid. Further evaluation of all of these strategies is needed to assess their effectiveness in reducing fatal and nonfatal choking-related episodes.

Parents and caregivers can reduce choking hazards in a child’s environment. Special attention should be given to food and nonfood items (e.g., candy, nuts, and coins) commonly involved in choking. Younger children are particularly at risk because of their tendency to place objects in their mouths, poor
chewing ability, and narrow airways compared with those of older children.1,2 Recommendations are available to guide parents and caregivers about the types of food items that are inappropriate for children aged <4 years.6,7 Removal of nonfood choking hazards also is important for infants and children aged ≤4 years because approximately one third of all choking episodes involve nonfood items.

Because complete removal of all choking hazards is unlikely, parents and caregivers should learn how to treat a child who is choking. A federal campaign has been launched to encourage parents and caregivers to learn early treatment of childhood medical emergencies, including choking.8 Early and effective treatment is crucial to prevent morbidity and mortality from childhood choking. Methods taught routinely in courses on cardiopulmonary resuscitation (CPR) or first aid can be lifesaving when instituted early by trained parents and caregivers.9 Opening the airway quickly by ejecting the foreign body can avoid potentially severe injuries. The American Academy of Pediatrics recommends that all parents and caregivers participate in the American Heart Association’s Basic Life-saving Course or the American Red Cross’ Infant/Child CPR Course.10

The findings in this report are subject to at least five limitations. First, the analysis included all cases in which choking was involved. It was not possible, using information obtained in NEISS-AIP, to distinguish cases in which the child choked on a substance that entered and blocked the airway from other cases in which the child choked as the result of pharyngeal irritation or an esophageal foreign body. Second, this report considered only cases treated in EDs and did not include deaths or episodes in which medical care was obtained at a physician’s office or another health-care facility or was not received at all. For example, only 55% of choking children for whom emergency medical services were contacted were transported to EDs for care.1 Third, NEISS-AIP does not provide information on outcomes after discharge from EDs. Fourth, NEISS-AIP is designed to provide national estimates and does not provide state or local estimates. Finally, exposure to candy, food, and other items differs by age group and was not considered in this analysis.


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Expansion of Eligibility for Influenza Vaccine Through the Vaccines for Children Program

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ON JUNE 20, 2002, THE ADVISORY COMMITTEE ON IMMUNIZATION PRACTICES (ACIP) adopted a resolution expanding the group of children eligible for influenza vaccine coverage under the Vaccines for Children (VFC) program. The resolution extends VFC coverage for influenza vaccine to all VFC-eligible children aged 6-23 months and VFC-eligible children aged 2-18 years who are household contacts of children aged <2 years. The resolution becomes effective on March 1, 2003, for vaccine to be administered during the 2003-04 influenza vaccination season and subsequent seasons. ACIP is expanding VFC influenza coverage because children aged ≤23 months are at substantially increased risk for influenza-related hospitalizations.

For the upcoming 2002-03 influenza season, no changes are being made to groups of children eligible for influenza vaccine under the VFC program. Children aged 6 months–18 years who are eligible for the VFC program and who have a high-risk medical condition or are household members of a person at high risk for complications may receive influenza vaccine through the program. Groups of children with high-risk medical conditions include those who (1) have chronic disorders of the pulmonary or cardiovascular systems, including asthma; (2) have required medical follow-up or hospitalization during the preceding year because of chronic metabolic diseases (including diabetes mellitus), renal dysfunction, hemoglobinopathies, or immunosuppression (including immunosuppression caused by medications); (3) are receiving long-term aspirin therapy; (4) are residents of long-term care facilities; and (5) are adolescent females in the second or third trimester of pregnancy during the influenza season (typically November-March).

The availability of additional supplies of influenza vaccine through the VFC program for the 2003-04 season will be based on anticipated need. VFC providers should provide their state’s vaccination program with accurate and practical estimates of the number of VFC patients they plan to vaccinate. Accurate estimates are essential to ensure an adequate supply of vaccine and to avoid vaccine wastage. ACIP recommendations for the 2002-03 influenza season are available at http://www.cdc.gov/nip/flu/target-groups.htm and http://www.cdc.gov/mmwr/preview/mmwrhtml/0r5103a1.htm. Information about the VFC program is available at http://www .cdc.gov/nip/vfc/vfc.htm. The VFC Resolution for Influenza Vaccine (10/98-4), effective during the 2002-03 season, is available at http://www.cdc.gov/nip/vfc/flu.pdf.