

of new or unusual infections (2,3). This information is essential for public health and medical interventions.

As outlined by Louie et al., hospital autopsy rates have dropped to single digits, and concerns by pathologists about occupational risks and biosafety have likely contributed to this decline. Currently, the last stronghold of autopsy expertise is forensic pathology (4). However, the medicolegal death investigative system does not have jurisdiction over all potential infectious disease deaths nor is it adequately supported to assume the cases that are missed by our present hospital autopsy system. Additionally, many medicolegal and hospital autopsy facilities with outdated or poorly-designed air flow systems are ill suited to handle autopsies when infectious disease is suspected (5). Air-handling systems can be expensive to fix.

Reference centers such as the National Prion Disease Pathology Surveillance Center, while providing diagnostic expertise, fail to surmount the biosafety obstacles (real and perceived) that prevent pathologists from enthusiastically performing autopsies on those who died of potential infectious diseases, including prion diseases. One potential solution is the creation of regional centers of excellence for infectious disease autopsies that could operate in conjunction with a mobile containment autopsy facility (5,6). Such centers could provide diagnostic expertise as well as biosafety capacity.

Kurt B. Nolte*

*University of New Mexico, Albuquerque, New Mexico, USA

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Address for correspondence: Kurt B. Nolte, Office of the Medical Investigator, MSC11 6030, University of New Mexico, Albuquerque, NM 87131-0001, USA; fax: 505-272-0727; email: knolte@salud.unm.edu

Q Fever Wildlife Reservoir

To the Editor: To the list of zoonotic infections with wildlife sources reported by Kruse et al. (1), I would add *Coxiella burnetii* infection because of its global impact, extensive presence in the animal kingdom, and potential for use as an agent of bioterrorism (2). *C. burnetii* causes Q fever, a self-limited disease that usually appears as undifferentiated fever, pneumonia, or hepatitis, but which may progress into chronic disease, especially endocarditis, among susceptible persons. Q fever is endemic worldwide in domestic mammals, especially ungulates (cattle, sheep, and goats), but also has been found in wild mammals, birds, and arthropods. The transmission of Q fever to humans from wild rabbits was documented in the 1980s (3). More recently, a study showed seroprevalence of Q fever ranging from 7% to 53% in

brown rats (*Rattus norvegicus*) in Oxfordshire, which suggests that they are a possible reservoir for *C. burnetii* in the United Kingdom. The study also speculated why cats, as frequent predators of rats, are important in maintaining the transmission cycle of the disease (4).

A case-control study published in 2001 (5) attempted to define the risk factors for an increase in the incidence of Q fever in French Guiana in 1996. The study found no link between Q fever and domestic ungulates, the usual source of outbreaks. The role of pets, basically dogs and cats, as a reservoir was also excluded. Multivariate analysis showed that living in close proximity to the forest, exposure to wild animals (including bats), and working in public trade or public works were all associated with infection. A strong correlation between large amounts of rainfall and higher incidence of Q fever was found also. All of these findings suggested a wild reservoir as a potential source of the epidemics, although the researchers could not identify a particular species as the specific source.

Miguel G. Madariaga*

*University of Nebraska Medical Center, Omaha, Nebraska, USA

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Address for correspondence: Miguel G. Madariaga, 4239 Farnam St, Suite 710, Omaha, NE 68131, USA; fax: 402-552-6354; email: agairadam@yahoo.com

Vaccine-derived Poliovirus, Thailand, 2003

To the Editor: The Polio Eradication Campaign was started in Thailand in 1990, and the last polio case was reported in April 1997. Although no new cases have been reported, the Polio Eradication Campaign continues with 4 prevention strategies: high coverage with 3 doses of oral polio in children <1 year of age, acute flaccid paralysis surveillance, acute flaccid paralysis case investigation and response, and National Immunization Day. Also, the Ministry of Public Health is prepared for a national emergency response to polio importation and circulated vaccine-derived poliovirus (1).

In April 2003, a case of acute flaccid paralysis was reported from Phakhao district, Loei province. The patient was an 18-month-old boy with normal physical development and nutritional status. He had a history of mild asthma and had received bronchodilator drugs occasionally during upper respiratory tract infections in the past. The patient had been fully vaccinated. He had received a total of 5 doses of oral polio vaccine: a dose at 2, 4, and 6 months of age, and 2 doses on National Immunization Day in December 2002 and January 2003.

On March 27, 2003, while visiting his grandmother in Phoowiang district, Khonkaen province (80 km from his residence), the patient became ill. Pneumonia was diagnosed; injected

medications were administered into his left hip once a day for 3 days. The patient fully recovered.

On April 1, 2003, cellulitis of finger developed in the patient. The affected finger was incised and drained, and oral antimicrobial drugs were administered. The inflammation extended to his elbow but later subsided.

On April 7, fever, cough, and dyspnea developed in the patient. Two days later, the patient's left leg became weak. He was admitted to Phakhao Hospital with a diagnosis of pneumonia and with weakness in his left leg. He was later referred to Loei Provincial Hospital and acute flaccid paralysis was diagnosed on April 11. The muscle weakness progressed until he could not sit.

On April 14, the patient was referred to Khonkaen Regional Hospital with weakness in both legs and arms (grade 0–1). Chest radiograph showed perihilar pneumonia. Cloxacillin, gentamicin, and immunoglobulin (Ig) (6 g/day \times 4 days, patient weight 12 kg) were administered intravenously to the patient. He was discharged on April 30 with a diagnosis of Guillain-Barré syndrome and bacterial pneumonia. The muscle tone in his right leg and both arms was grade 3; however, he could not move his left leg.

Stool samples were collected on April 11 and 14 and tested for polio at the Department of Medical Science (reference laboratory for polio in Southeast Asia). Poliovirus type 2 was isolated in the samples; however, the results were inconclusive for strain differentiation. The isolates were sent to the Centers for Disease Control and Prevention (CDC), USA, for genetic sequencing, and the result showed poliovirus type 2 with 1.6% difference from Sabin strain poliovirus. Without evidence of recombination with other nonpolio enterovirus, the pattern of genomic change was similar to the change that occurs in immunodeficient persons. Immune

system testing of the patient on August 13 showed IgG = 205.9 mg/dL (normal 800–1,700), IgA <5.5 mg/dL (normal 100–490), IgM <16.8 mg/dL (normal 50–320), and IgE <18.0 mg/dL (normal 0–100). Antibodies to poliovirus type 1, 2, and 3 were 1:16, 1:32, and 1:8, respectively. Testing of the follow-up stool samples showed P1/Sabin on August 10. Test results were negative on October 13, and results showed nonpolio enterovirus on November 10 and December 14.

Before the large-scale outbreak response immunization was conducted, 339 serum samples were collected from children <5 years of age who lived in the same district as the patient or in the same subdistrict as his grandmother. Among 153 children who brought their vaccination records, the median dose of oral polio vaccine was 7 (range 2–15). All had antibody >1:8 to poliovirus types 1, 2, and 3. Approximately 2,000 stool samples were collected from children <5 year of age who lived in the same district as the case-patient or his grandmother. However, after the immunodeficiencies vaccine-derived poliovirus was identified, isolation of the virus was attempted only from stool samples from children who lived in the same subdistrict as the patient. From 223 stool samples, 4 Sabin strain poliovirus and 32 nonpolio enteroviruses were isolated. In addition, 2 of 18 stool samples collected in July from close contacts of the case-patient were positive for Sabin strain poliovirus and negative for vaccine-derived poliovirus.

The Loei Provincial Health Office initially did a small-scale response immunization in 3 adjacent villages (128 of 129 children) on the day that the case of acute flaccid paralysis was reported. Coverage of third dose of oral polio vaccine in these villages was 100%. No response immunization was conducted at the village in Khonkaen. On August 8, genetic