Annals of Internal Medicine | Review

# Narrative Review: Tetanus—A Health Threat After Natural Disasters in Developing Countries

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Tetanus is an expected complication when disasters strike in developing countries, where tetanus immunization coverage is often low or nonexistent. Collapsing structures and swirling debris inflict numerous crush injuries, fractures, and serious wounds. *Clostridium tetani* infects wounds contaminated with dirt, feces, or saliva and releases neurotoxins that may cause fatal disease. Clusters of infections have recently occurred after tsunamis and earthquakes in Indonesia, Kashmir, and Haiti. The emergency response to clusters of tetanus infections in developing countries after a natural disaster requires a multidisciplinary approach in the absence of an intensive care unit, readily available resources, and a functioning cold-chain system. It is essential that injured people receive immediate surgical and medical care of contaminated, open wounds with immunization and immunoglobulin therapy. Successful treatment of tetanus depends on prompt diagnosis of clinical tetanus, treatment to ensure neutralization of circulating toxin and elimination of *C. tetani* infection, control of spasms and convulsions, maintenance of the airway, and management of respiratory failure and autonomic dysfunction.

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*Clostridium tetani* is an anaerobic, gram-positive bacillus that causes an acute, toxin-mediated infection. The mature organism loses its flagella and forms a spherical terminal spore. Infection with *C. tetani* begins when spores enter the body through an area of skin breakdown from a traumatic wound. These spores are distributed worldwide in the soil and are found in the feces of such animals as horses, cows, sheep, dogs, cats, rats, pigs, and chickens. Soil contaminated by excreta from these animals or treated with manure fertilizers can contain large quantities of spores. Spores can also be found in carpeting.

Under favorable anaerobic conditions, *C. tetani* produces 2 neurotoxins: tetanolysin and tetanospasmin (commonly called *tetanus toxin*). The role of tetanolysin in human tetanus is unclear. Tetanospasmin migrates into the central nervous system from a peripheral nerve at a site of infection. Tetanospasmin inhibits the release of γ-aminobutyric acid (GABA) and glycine, which results in failure of inhibition of motor reflexes and generalized contractions of the agonist and antagonist musculature, causing tetanic spasms. Although *C. tetani* is sensitive to heat and cannot survive in the presence of oxygen, its spores resist extremes of temperatures and are stable in ambient oxygen tensions. In addition, the usual antiseptics—phenol, ethanol, and formalin—are ineffective in eliminating the spores. Rather, iodine, glutaraldehyde, or hydrogen peroxide is needed for effective antisepsis (1).

During a natural disaster, persons develop tetanus when they acquire *C. tetani* infection via puncture wounds or penetrating injuries; burns; crush injuries; gangrene; or devitalized tissue that became contaminated with dirt, feces, or saliva. Nontraumatic wounds, such as dental extractions, burns, animal bites, and abortions, may become infected (2, 3). Tetanus after intramuscular injection of quinine, which is used in some developing countries to treat malaria, has been reported and has an unusually high mortality rate (4). Spores have also been found on skin and in contaminants of heroin (3).

## Endemic Tetanus and Outbreaks of Tetanus in Developing Countries

Tetanus is a serious, potentially fatal disease. Its incidence in the developed world has substantially decreased since the introduction of vaccination with tetanus toxoid. In developing countries, however, endemic adult and neonatal tetanus remain a major public health challenge. About 1 million cases of tetanus are reported worldwide annually, suggesting a global incidence of about 18 per 100 000 persons per year and an estimated 300 000 to 500 000 deaths per year (5). The overall case-fatality rate in a Nigerian teaching hospital over 9 years was estimated to be nearly 37%, and in a study of 8697 patients with tetanus in India, the case-fatality rate was nearly 50% (6, 7). In contrast, developed countries with intensive care units have case-fatality rates less than 20% (8). Many of the endemic cases in developing countries are from neonatal tetanus (9). Developing countries also encounter a higher frequency of tetanus in adolescents and younger adults because of inadequate or irregular immunization programs (7).

Tetanus is one of the few vaccine-preventable diseases that is infectious but not contagious; it cannot be transmitted from person to person. In natural disasters, such as earthquakes and tsunamis, an epidemic of injuries in survivors from developing countries with endemic tetanus can develop into apparent clusters of infections (10, 11). "Point-source" outbreaks typically occur in younger per-

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In a natural disaster, tetanus occurs when a wound is contaminated with dirt, feces, or saliva from puncture, penetration, or another mechanism, including burns and crush injuries. Tetanus can also develop in patients with gangrene or devitalized tissue.

Clinicians must rely on clinical findings to promptly diagnose tetanus. Symptoms and signs include trismus or risus sardonicus or painful muscular contractions in adults with a history of wound infection (although tetanus is sometimes cryptogenic).

Active immunization with tetanus toxoid–containing vaccine and passive immunization with tetanus-specific immune globulin can protect against tetanus. Metronidazole is the preferred antimicrobial, and prompt irrigation and debridement of the wound should be performed.

If ventilator support is not available, benzodiazepines are the preferred agent to manage respiratory failure. Alternative agents are baclofen, magnesium, dantrolene, barbiturates, and chlorpromazine.

$\beta$-Blockers, magnesium, and morphine can improve autonomic dysfunction.

An alert system for immediate reporting and prompt investigation after a disaster in epidemic-prone regions should be instituted, and health care workers should be informed of cases of dysphagia and trismus.

sons who survive their initial injuries but have wounds that are at risk for infection. A few of the largest clusters reported to date are from Aceh, Indonesia, and Kashmir. In Aceh, by 18 days after the tsunami on 26 December 2004, 106 tetanus cases had occurred, with a case-fatality rate of 18.9% in a population with a poor immunization status (12). In Kashmir, within 30 days of the earthquake on 5 October 2005, 139 cases and 41 deaths were reported (10). A smaller series was reported after the earthquakes in Yogyakarta, Indonesia, in which 26 patients presented to 8 hospitals in the region. About 31% of the patients died; distance and type of hospital were significant predictors of death (11).

An additional challenge in both developed and developing countries is underreporting or no reporting of tetanus (13–15). In the United States, Sutter and colleagues (16) concluded that only 40% of cases are reported to the Centers for Disease Control and Prevention, 60% are reported to the National Center for Health Statistics, and almost 25% are reported to neither organization. Community-based surveys on neonatal tetanus from the 1970s and 1980s in more than 40 countries revealed that fewer than 10% of tetanus-related deaths were routinely reported (17).

Before the earthquake in Haiti on 12 January 2010, tetanus was endemic in the country. According to the World Health Organization (WHO), only 50% of the population in Haiti had adequate tetanus coverage over the past decade (18). The general epidemiology of tetanus in adults has not changed substantially. In Haiti from 2004 to 2009, 3 to 119 cases have been reported annually, according to the Centers for Disease Control and Prevention; neonatal tetanus comprises most of the cases (18). The earthquake inflicted great damage, including many collapsed structures that trapped survivors under rubble, and caused many other traumatic injuries. The incidence of tetanus after the disaster increased markedly over baseline values, although exact numbers are unknown and underreporting was likely. As of February 2010, surveillance reports documented multiple cases of clinically confirmed tetanus from the University Hospital of Haiti, Cuban Brigade, and Doctors Without Borders (19). The Ministry of Health, with support from WHO/Pan American Health Organization, United Nations Children's Fund, and nongovernmental partners, developed a postdisaster vaccination plan against tetanus and diphtheria for children and adults. The first phase targeted an estimated 250 000 children younger than 8 years and 1.2 million persons living in temporary settlements in metropolitan Port-au-Prince, surrounding communities in the Department of l'Ouest, and Jacmel. As of 28 February 2010, approximately 149 000 adults and children had been vaccinated at vaccination posts set up in more than 300 settlements. A second phase in the future will include mass vaccination of all people in the affected areas (20).

## Clinical Findings and Diagnosis

No laboratory data provide a definitive diagnosis of tetanus. The organism is rarely recovered from the site of infection, and there is no detectable antibody response, even in severe cases. Therefore, clinicians must rely on clinical findings for prompt diagnosis. The WHO definition of adult tetanus includes trismus (lockjaw) or risus sardonicus, or painful muscular contractions with a history of a wound infection (although some patients cannot recall an injury) (21).

There are 4 clinical types of tetanus: generalized, localized, cephalic, and neonatal. The variances in type reflect the site of toxin action and not toxicologic differences. Generalized tetanus is the most commonly recognized type of tetanus. Focal symptoms include trismus, rigidity of the masseter muscles, and back or shoulder stiffness. Other symptoms may include abdominal rigidity, generalized spasm with decorticate posturing, opisthotonos, flexion and adduction of the arms, clenching of the fists, and extension of the lower extremities. Localized tetanus consists of fixed rigidity of the muscles associated with a site of injury. This may be mild, persist for months, and resolve

spontaneously. Patients may experience weakness, rigidity, extreme pain, and increased deep tendon reflexes.

Cephalic tetanus involves the lower cranial nerve musculature; it occurs with injuries to the head or neck. Before the typical features of generalized tetanus appear, patients with cephalic tetanus may manifest confusing clinical findings, including dysphagia, trismus, and focal cranial neuropathy. The facial nerve is most commonly affected, but other cranial neuropathies may also be present. Patients present with facial paresis, dysphagia, otitis media, and dysfunction of extraocular movements. Cephalic tetanus causes several conditions, including ophthalmoplegic tetanus, supranuclear oculomotor palsies, and the Horner syndrome (22–24). Neonatal tetanus, which follows infection of the umbilical stump in infants born to mothers who lack immunity, may present during the second week of life with inability to suck and weakness. Survivors usually have developmental delay.

As tetanus becomes less common, cases are likely to be misdiagnosed or go unrecognized. In addition, in many areas with limited resources, the diagnosis can be clouded by the wide array of other diseases and conditions endemic to that region. Spasms of tetanus may be confused with posturing or epileptic seizures. However, tetanic spasms are very painful and, unlike generalized seizures, do not cause loss of consciousness. Strychnine intoxication due to ingestion of rat poison, which affects the central nervous system via a similar mechanism, also causes generalized spasms. The nuchal rigidity of meningitis can resemble the neck stiffness of tetanus. Cerebral malaria, meningoencephalitis, and subarachnoid hemorrhage are other considerations in the differential diagnosis of tetanus after natural disasters in developing countries. Alveolar ridge abscess can cause trismus, but the oral pain and tenderness seen with such an abscess is not characteristic of tetanus. Hypocalcemia produces the Chvostek sign, and alkalemia produces the Trousseau sign. Alveolar dystonic reaction to dopamine blockade can present with torticollis and oculogyric crisis, but reflex spasms are absent.

Patients should be queried about a history of injury or wounds, but they may not recall a specific episode. Between 7% and 21% of tetanus cases are cryptogenic (25). In a study of patients in Nigeria with tetanus of the lower limb, the most common portal of entry for *C. tetani* was the foot because of poor protective footwear (26).

The incubation period of tetanus can range from 3 to 21 days; more severe cases occur around day 8 and mild to moderate cases occur around day 11 (27). Severity of disease relates to the incubation period and the interval from the onset of symptoms to appearance of spasms (28). In general, shorter incubation periods are associated with higher mortality rates. The farther the introduction sites of the spores from the central nervous system, the longer the incubation period and the milder the disease.

*Table 1.* **Postexposure Tetanus Prophylaxis**

| Vaccination History | Clean, Minor Wounds | | All Other Wounds* | |
|---|---|---|---|---|
| | Td† | TIG | Td† | TIG |
| Unknown number or <3 doses | Yes | No | Yes | Yes |
| ≥3 doses | | | | |
| ≥10 y since most recent dose | Yes | No | Yes | No |
| 5–9 y since most recent dose | No | No | Yes | No |
| <5 y since most recent dose | No | No | No | No |

Td = tetanus and diphtheria; TIG = tetanus-specific immune globulin.
* Wounds >1 cm in depth, incurred >6 h earlier, or with stellate or avulsion configuration; crush injuries or burn injuries; devitalized tissue; and wounds contaminated with dirt, feces, or saliva.
† For children aged <7 y, vaccination with diphtheria, tetanus toxoid, and pertussis (DTaP or DTP) (or diphtheria and tetanus toxoid [DT] alone, if pertussis vaccine is contraindicated) is preferred to vaccination with tetanus toxoid alone. For children aged ≥7 y, vaccination with Td is preferred to vaccination with tetanus toxoid alone. For adolescents and adults up to age 64 y, vaccination with tetanus toxoid given as Tdap is preferred if the patient has not previously been vaccinated with Tdap.

## Management and Complications Under Adverse Conditions

### Active and Passive Immunization

Patients with newly diagnosed tetanus during a natural disaster typically have dirty, extensive wounds and should receive immunotherapy, wound management, and antibiotics immediately. The most tetanus-prone wounds have a depth greater than 1 cm; were incurred more than 6 hours previously; have a stellate or an avulsion configuration; are crush or burn injuries; have resulted in devitalized tissue or gangrene; or are contaminated with dirt, saliva, or feces (29). The disease does not confer immunity because the total amount of toxin produced is so small that it is inadequate to prompt an immune response. Initially, patients should receive immunotherapy with active and passive immunization (**Table 1**). Protection can be provided by active immunization with tetanus toxoid–containing vaccine and passive immunization with tetanus-specific immune globulin (TIG). Because of waning antitoxin titers, many people have antitoxin levels below the optimal level 10 years after the last dose of vaccine; a booster injection should be administered every 10 years to provide continued protection. Illness may be milder in patients with existing but nonprotective antitetanus antibody titers (30).

Tetanus toxoid is available as a single-antigen preparation or in combination regimens, with diphtheria toxoid as pediatric diphtheria–tetanus toxoid or adult tetanus–diphtheria or with both diphtheria toxoid and acellular pertussis vaccine. The age-appropriate tetanus toxoid–containing vaccine should be administered as 0.5 mL by intramuscular injection. In a primary series, a second dose is required 1 to 2 months after the first dose and a third dose 6 to 12 months later. A strategy used in developing countries is to identify regions with cases of neonatal tetanus and target women of childbearing age to receive at least 2 doses of tetanus toxoid (2). Nonimmunized pregnant patients

should receive at least 2 doses of tetanus toxoid; the first dose should be given as soon as possible and the second dose given no more than 4 weeks later, and preferably 2 weeks before delivery.

One challenge for a successful vaccination program is an adequate cold-chain system. All tetanus toxoid–containing vaccines should be stored at 2 to 8 °C (35 to 45 °F). Freezing reduces the potency of the tetanus component and should be avoided. Preparing for rescue efforts in a natural disaster requires provision of working refrigerators or cold rooms that have access to diesel fuel or electricity (31).

Passive immunization with TIG is of paramount importance to ensure that the tetanospasmin burden does not increase. The TIG binds the toxin that is free and not yet bound to tissues. However, it does not affect toxin that has already attached to nerve endings. Tetanus-specific immune globulin is given as an intramuscular injection at a separate site from the active vaccination; the dose is 500 U of TIG of human origin or 1500 to 5000 U of TIG of animal origin. Human TIG is the safest antitoxin, but it may not be as readily available as antitoxin of animal origin in developing countries. Because animal-origin TIG is derived from horse serum, an intradermal test dose of 0.1 mL in a 1:10 dilution of sodium chloride solution is needed before administering the full dose to evaluate for hypersensitivity (6). Clean, minor wounds in patients who have received less than the 3-injection series should receive toxoid without TIG. It is not necessary to give tetanus toxoid or TIG to patients who received the complete series of immunization and a booster within 10 years, regardless of wound type.

### Wound Care and Antimicrobial Therapy

Tetanus toxin can be reduced with antibiotics and elimination of the anaerobic environment with aeration of the wound (32). Irrigation of the wound may also be useful. Although the utility of surgical wound debridement is unclear, the wound should still be debrided in an effort to eradicate *C. tetani* and improve antimicrobial activity (33).

In nonimmunized pregnant patients, 4 factors contribute to the risk for infection during birth: cleanliness of instruments and dressings; length of the umbilical stump (a longer stump seems to be safer); the care with which the cord is ligated; and cleanliness of the environment, including the mother's clothing (1). Topical antibiotic treatment of the umbilical stump seems to be more effective than proper handwashing hygiene and immunization of the mother alone (34). The use of penicillin G (100 000 to 200 000 IU/kg daily, given intravenously in 2 to 4 divided doses) was favored in the past; however, penicillin G is a GABA-receptor antagonist and can act synergistically with tetanospasmin (32). Metronidazole is the preferred antimicrobial because it is relatively inexpensive and can better penetrate anaerobic tissues; it is given at doses of 500 mg every 6 hours intravenously or orally for 10 to 14 days (35). An alternative to metronidazole is doxycycline, 100 mg every 12 hours for 7 to 10 days. Macrolides, clindamycin, cephalosporins, and chloramphenicol are also effective.

### Supportive Care

In most developing countries, especially those of sub-Saharan Africa, critical care services may be underdeveloped. For example, critical care nursing was introduced in Nigeria in 1982, but there are currently only 2 training hospitals and 380 members of the Nigerian chapter of the World Federation of Critical Care Nurses. In Nigeria, which has a population of 140 million, that amounts to only 10 critical care nurses for each of that country's 36 states (36). Invasive monitoring, parenteral nutrition, and the daily cost of an intensive care unit (ICU) bed are expensive even for developed countries. In regions with erratic water and power supplies, the "ICU" may simply be a ward of 30 patients with a nurse and a kerosene lantern (6, 36).

In the absence of a fully configured ICU and ventilator support, acute respiratory failure in tetanus remains a leading cause of death. In an analysis of 335 consecutive patients with tetanus at the University Hospital of Caracas, Caracas, Venezuela, who were treated before an ICU was implemented, the mortality rate was 44% compared with 15% among the 306 consecutive patients who were managed after the ICU was developed (37). Most of this improvement was due to prevention of deaths from acute respiratory failure.

If an ICU cannot be established, a separate ward or location should be designated where patients can avoid tactile and auditory stimulation. Tetanic spasms may be triggered by loud noises or other sensory stimuli, such as physical contact or light. The use of nondepolarizing paralytic agents, including vecuronium and pancuronium, is not safe in the absence of ventilator support; therefore, GABA agonists are preferred in such settings. Benzodiazepines control both the rigidity and spasms. Diazepam is most frequently used, in increments of 5 mg. Lorazepam in 2-mg increments has similar efficacy to and a longer duration of action than diazepam (38). Dosages are titrated to achieve spasm control without significant hypoventilation. It is not unusual to reach daily dosages of diazepam greater than 500 mg; high doses are preferentially given through a feeding tube to prevent metabolic acidosis from the propylene and polyethylene glycol vehicle of the intravenous solution (39). Midazolam, a drug in the same class as diazepam, is safer because it is water soluble and will not cause acidemia.

Baclofen, a GABA agonist, can also be used (40). Given intrathecally, baclofen aids in the management of spinal convulsions and limits the need for tracheal intubation. In a study from Burkina Faso, where resources are lacking, intrathecal baclofen was effective in 12 of 14 patients with severe tetanus (41). The dose of baclofen can be titrated slowly to avoid respiratory depression. However,

local infection at the site of the intrathecal catheter is a potential complication (41).

Magnesium sulfate may be used alone or in combination with benzodiazepines to control spasm and autonomic dysfunction. An intravenous loading dose of 5 g (or 75 mg per kg of body weight) is given, followed by 2 to 3 g/h until spasm control is achieved. To avoid overdose, the patellar reflex can be monitored; areflexia (absence of the patellar reflex) occurs at the upper end of the therapeutic range (4 mmol/L) (42). If areflexia develops, the dose of magnesium sulfate should be decreased. Other agents used for spasm control include dantrolene (1 to 2 mg/kg intravenously or by mouth every 4 hours); barbiturates, preferably short-acting (100 to 150 mg every 1 to 4 hours in adults; 6 to 10 mg/kg in children; by any route); and chlorpromazine (50 to 150 mg intramuscularly every 4 to 8 hours in adults; 4 to 12 mg intramuscularly every 4 to 8 hours in children) (21).

### Surgical Care

When conservative measures are unsuccessful in maintaining an airway, the patient can develop upper airway obstruction from laryngospasm. Upper airway obstruction occurs from spasms, including those of the vocal cords. Contributing factors to respiratory failure include phrenic neuropathies and spasms of the respiratory muscles with the diaphragm and abdominal musculature. In such instances, tracheostomy may be necessary. The Ablett classification has been used in ICUs outside the United States to identify moderate, severe, and very severely affected patients (43, 44). **Table 2** shows the Ablett classification and can be used as a guide to identify patients with upper airway obstruction of grade 2 or higher severity, who may benefit from early tracheostomy for upper airway obstruction or have difficulty managing secretions (44).

### Complications

Patients who survive the initial muscle spasms and respiratory failure, which typically occur in the first 2 weeks, may encounter further complications. Prolonged contractures and convulsions may result in fractures of the spinal or long bones and rhabdomyolysis with renal failure (45). A common challenge of tetanus management in developed countries is autonomic dysfunction, including hypertension and tachycardia alternating with bradycardia and hypotension (32). The cause of autonomic dysfunction is most likely excessive catecholamine release similar to pheochromocytoma. Treatment includes parenteral labetalol, which produces both $\alpha$- and $\beta$-adrenergic receptor blockade. The optimal therapy for autonomic dysfunction with sympathetic overactivity has not been well defined. $\beta$-Blockers, such as propranolol, were used in the past but can cause hypotension or sudden death (21). Esmolol and clonidine are alternative agents. Morphine has also been shown to be effective for autonomic dysfunction (46). Magnesium is useful both for its protective effects against arrhythmias and its antispastic properties.

*Table 2.* **Ablett Classification of Tetanus Severity**

| Grade | Severity | Symptoms |
|---|---|---|
| 1 | Mild | Mild trismus, general spasticity, no respiratory compromise, no spasms, no dysphagia |
| 2 | Moderate | Moderate trismus, rigidity, short spasms, mild dysphagia, moderate respiratory involvement, respiratory rate >30 breaths/min |
| 3 | Severe | Severe trismus, generalized rigidity, prolonged spasms, severe dysphagia, apneic spells, pulse >120 beats/min, respiratory rate >40 breaths/min |
| 4 | Very severe | Grade 3 with autonomic dysfunction |

Patients who are more seriously affected remain ill longer and thus are at increased risk for nosocomial infections with prolonged hospitalization. The metabolic demands in patients with tetanus can exceed those of other severe diseases, including sepsis. Patients enter a catabolic state because of the constant muscle spasms and excessive autonomic activity. Therefore, adequate fluid resuscitation and early nutritional support are imperative (42). Enteral feeding is preferred and sometimes necessitates placement of a nasogastric tube. In patients who develop severe abdominal spasms or ileus, central venous nutrition is necessary but is difficult to provide in resource-constrained countries.

## Response and Challenges After a Natural Disaster

In a natural disaster, the problems characteristic of developing countries are further amplified. Unreliable supplies of electrical power and diesel fuel, insufficient clean water, inadequate supplies, and lack of functioning equipment are barriers to optimal care in patients with tetanus. Poor personal hygiene, insufficient disease-specific knowledge, poor wound management, and lack of immunization have been implicated in the high prevalence of tetanus in developing countries (28). The average delay in seeking health care of 3.3 days reflects the lack of perception of the seriousness of the disease on the part of the patient or family, lack of transportation from remote areas, and lack of money to pay for health care (6). However, in the 1920s and 1930s—before a tetanus vaccine and mechanical ventilation were available—careful monitoring and nursing care alone improved survival (21). If patients with tetanus can be supported through 1 to 2 weeks of spasm and other complications, the chances of complete recovery greatly increase, particularly in nonelderly and previously healthy patients (21). An alert system for immediate reporting and prompt investigation after a disaster in epidemic-prone regions should be instituted, with health care workers being informed of cases of dysphagia and trismus.

Preventive strategies are necessary, including the WHO initiative to reduce the burden of tetanus infections through its Maternal and Neonatal Elimination Program. This program aims to reduce the number of maternal and neonatal tetanus cases to such low numbers that tetanus is no longer a major public health problem. Although tetanus cannot be eradicated because tetanus spores are present in the environment worldwide and are always a potential pathogen in traumatic injuries, strategies are in place to prevent infection. In 1989, the 42nd World Health Assembly called for elimination of neonatal tetanus by 1995. The WHO estimates that in 2008, 59 000 newborns died of neonatal tetanus, a 92% reduction from the late 1980s. Progress was achieved with strengthening of the health services; increase in diphtheria, pertussis, and tetanus vaccination; coverage in childhood vaccination programs at schools; and a strong effort to eliminate neonatal tetanus since the 1980s. However, as of December 2010, 39 countries have not eliminated maternal and neonatal tetanus (47). Neonatal tetanus remains a public health problem, but advocating childbirth under hygienic conditions and efforts to vaccinate all women of childbearing age can help control disease burden and outbreaks in natural disasters. Nevertheless, within clusters of infections, surviving tetanus requires prompt diagnosis, neutralization of circulating toxin, elimination of *C. tetani* infection, control of spasms and convulsions, and maintenance of the airway. Adequate care of patients necessitates a multidisciplinary approach, with cooperation among all members of both national and international health care teams, to provide careful monitoring and aggressive therapy.

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**2009 *ANNALS* POETRY PRIZE**

Congratulations to Sarah Leeper, winner of the 2010 *Annals* Poetry Prize. Her poem "What I Remember Most" was published in the 4 January 2010 issue (vol. 152, no. 1, page 68). Ms. Leeper is a medical student at Brown University, Providence, Rhode Island.

*Annals* extends thanks to the contest judges: Daniel Bosch, whose most recent manuscript of poems is *Death's Doorman*, and Abigail Zuger, MD, a regular contributor to *The New York Times* and author of *Strong Shadows: Scenes from an Inner City AIDS Clinic* (WH Freeman, 1995).

For information on the Poetry Prize contest, visit www.annals.org/site/shared/poetry_prize.xhtml.

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