Tetanus is a rare disease in the United States, with only 20 cases reported in 2003. In 1990 to 2000, the average number of cases in the United States was 50 per year. The average annual incidence from 1995 to 2000 was approximately 0.16 cases per million population. Tetanus is more common among people 60 years old or older (0.35 cases per million population), patients 60 years old or older who have diabetes (0.70 cases per million population), and Hispanic persons (0.37 cases per million population). Injecting drug users are at unique risk for tetanus and accounted for 15% of cases of tetanus from 1998 to 2000. Most (74%) injecting drug users who developed tetanus reported injecting heroin, and 100% reported “skin popping” rather than intravenous injection.

Tetanus morbidity and mortality remain high, even with appropriate treatment. Current vaccination status decreases the severity of the disease and the likelihood of death from tetanus. In 1998 to 2000, 18% of patients with tetanus died. Of fatal cases, 75% occurred in patients who were 60 years old or older. No patients with an up-to-date vaccination status died of tetanus.

Although clinical cases are rare, emergency physicians (EPs) often are the first, and sometimes only, point of contact for patients. As a result, physicians must maintain an awareness of the clinical presentation of the disease. The diagnosis can be suspected but not confirmed in the emergency department (ED).

In addition to recognizing the clinical presentation of tetanus, EPs play a vital role in the prevention of the disease. Primary pediatric vaccination and regular decennial booster vaccination are the mainstays of disease prevention and severity modulation. Herd immunity does not occur with tetanus. Therefore, only people who receive the vaccination benefit from immunization. In the United States, the prevalence of tetanus immunity decreases by age, after 40 years of age. At 40 years of age, 80% of the population is immune to tetanus. By the age of 80 years, only 30% of the population remains immune. This decrease is most striking in women and Mexican Americans. Only 36% of persons age 65 years old or older report receiving tetanus vaccination in the past 10 years. Most cases of tetanus and fatalities resulting from tetanus are in patients who either have never been vaccinated or have not had a booster in the past 10 years. EPs have the opportunity to provide booster vaccination at times of minor to severe injury and skin infection. In light of pertussis epidemics,
providers should also consider a patient’s pertussis immunization status when choosing the tetanus vaccine.

**ANATOMY**

Most cases of tetanus are associated with acute trauma (Table 178.1), but many cases are associated with abscesses, cellulitis, chronic ulcers, dental infections, frostbite, and gangrene. In one study of injecting drug users with tetanus, 69% had an abscess at the injection site. Tetanus affects postpartum women, with an increased risk after unsanitary birth or abortion practices.

Puncture wounds are the most frequent type of acute trauma associated with tetanus. Puncture wounds include nail injuries to the foot, splinters, barbed wire injuries, tattoos, drug injection, penetrating eye injuries, and spider bites. Crush injuries, burns, and eye injuries are also portals of tetanus infection. In patients with tetanus, approximately 50% of injuries are located on the lower extremity, 36% on the upper extremity, 10% on the head or trunk, and 5% on other areas. The occurrence of tetanus following minor or trivial wounds is well documented in the literature. Tetanus results from minor wounds and abrasions when proper wound care is not administered. Nearly half of the wounds that resulted in tetanus in 1998 to 2000 occurred indoors.

**PATHOPHYSIOLOGY**

Clinical tetanus is caused by two exotoxins produced by *Clostridium tetani*, a gram-positive, anaerobic rod. The bacterium produces spores that are heat resistant, surviving autoclaving at 250°F for 10 to 15 minutes, and resistant to treatment with phenols and common chemical agents. The bacterium dies with heat or oxygen exposure. The bacterium and spores are widely disseminated in soil, intestines of farm animals and pets, and feces. Spores exist on human skin and contaminated heroin. In anaerobic conditions, such as puncture wounds and crush injuries, spores germinate.

*C. tetani* enters the body through a wound and produces two exotoxins: tetanolysin and tetanospasmin. Tetanolysin causes local cell death and creates an anaerobic environment in the wound site. Tetanospasmin interferes with the transmission of inhibitory impulses in the central nervous system. It creates a presynaptic blockage of the inhibitory Renshaw cells and Ia fibers of alpha motoneurons that transmit gamma-aminobutyric acid (GABA) and glycine. Renshaw cells that transmit acetylcholine are not affected as strongly. Tetanospasmin binding prevents inhibitory signals in the central nervous system.

Tetanus becomes a systemic disease as the toxin spreads through the body. Tetanospasmin binds to nerve terminals, is internalized, and travels in retrograde fashion to the cell synapse. The toxin travels at 75 to 250 mm/day, and it affects synapses of shorter nerves before synapses of longer nerves. The toxin also travels by lymphatic and blood flow to remote nerves. The toxin exhibits local effects first and then spinal motor effects. The autonomic system is the last to be affected because of the length of the nerves. Tetanospasmin also inhibits acetylcholine release, a process that leads to flaccid paralysis between episodes of spasticity.

The result of the general loss of inhibitory signals is rigidity with periods of spasticity. The reflex inhibition of antagonizing muscles is lost, thus allowing agonist and antagonist muscle groups to contract simultaneously. Autonomic disinhibition occurs late in the disease. Toxin binding appears to be irreversible; the growth of new nerve terminals is required to overcome the effects.

**CLINICAL PRESENTATION**

The average incubation period from time of injury to the onset of symptoms is 7 to 10 days, with a range of 1 to 60 days. Shorter incubation times are associated with more severe clinical presentation and a poor prognosis. Tetanus is usually an afebrile disease until autonomic instability occurs late in the disease. Fever suggests coinfection of the wound or other infectious causes. Generalized tetanus, or tetanus affecting the whole body, is the most common form of tetanus.

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**Table 178.1  Tetanus Wound Characteristics and Risks**

<table>
<thead>
<tr>
<th>Location of injury</th>
<th>MOST THREATENING</th>
<th>MOST COMMON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Face</td>
<td>Lower extremity</td>
<td>Upper extremity</td>
</tr>
<tr>
<td></td>
<td>Head and trunk</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type of injury</th>
<th>Location of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Puncture wound</td>
<td>Face</td>
</tr>
<tr>
<td>Crush injury</td>
<td>Upper extremity</td>
</tr>
<tr>
<td>Burn</td>
<td>Chronic wound</td>
</tr>
<tr>
<td>Chronic ulcer</td>
<td>Abrasion</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Patients</th>
<th>Wound Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetic patients</td>
<td>Intravenous drug users</td>
</tr>
<tr>
<td>Age &gt; 60 yr</td>
<td>Age &gt; 60 yr</td>
</tr>
<tr>
<td>Neonates</td>
<td>Hispanic ethnicity</td>
</tr>
<tr>
<td>No prior tetanus immunization</td>
<td>Diabetic patients</td>
</tr>
<tr>
<td></td>
<td>No prior tetanus immunization</td>
</tr>
<tr>
<td></td>
<td>Last immunization &gt;10 yr ago</td>
</tr>
</tbody>
</table>

**RED FLAGS**

**Wounds at Increased Risk of Tetanus**
- Puncture wounds
- Crush injuries
- Partial-thickness or full-thickness burns
- Chronic ulcers
- Wounds contaminated with soil, feces, saliva
- Avascular or gangrenous wounds
- Frostbite
- Penetrating eye injuries
- Avulsions

**Wounds at Low Risk of Tetanus**
- Minor abrasions
- Clean, minor wounds
In the first week of illness, the patient presents with rigidity and muscle spasms. Tetanus most commonly affects the cranial nerves first. The most common first symptoms and signs are trismus, neck stiffness, and dysphagia. Muscle spasm progresses diffusely to involve the facial muscles, thus causing the classic facial grimec risus sardonius. Disinhibition of the neck muscles causes neck extension. Truncal rigidity follows head and neck involvement.

The general increased tone is interrupted by acute spastic events that can involve any muscle groups. These spastic events can be spontaneous or caused by tactile, visual, or auditory stimuli. Agonist and antagonist muscle groups can simultaneously contract. The contractions are painful and can be strong enough to break long bones and avulse tendons. Opisthotonus is a classic spastic event in tetanus. Abdominal rigidity can mimic an acute abdomen. Spasticity of the trunk and diaphragm can interfere with respiration. Laryngeal spasm interferes with the gag reflex or can occlude the airway.

Before modern mechanical ventilation, death resulted from respiratory failure or aspiration. With modern mechanical ventilation, death is more commonly caused by autonomic events. The second week of illness involves autonomic instability in addition to the muscle spasms. The sympathetic nervous system is more strongly affected. Sudden increased autonomic tone, with elevated circulating catecholamine levels, increased vascular tone, hypertension, and tachycardia alternate with profound hypotension, bradycardia, and even cardiac arrest. Cardiac dysrhythmias occur, and the patient may develop hyperpyrexia at this point.

Recovery begins in the third or fourth week. Muscle spasms decrease, but rigidity may persist. The duration of recovery, in those who survive, ranges from 2 to 4 months, as new axon terminals grow. Most patients return to their baseline with no residual deficit.

**VARIATIONS**

Local tetanus is an uncommon presentation of the disease in which only focal symptoms occur. Muscle spasticity is limited to the area adjacent to the wound. Local tetanus can progress to generalized tetanus, and the disease is generally milder. The exception is in cases of cephalic tetanus.

Cephalic tetanus is another rare form of disease. Cephalic tetanus follows a head or facial wound or, rarely, otitis media. The cranial nerves are initially affected. Spasticity or flaccid paralysis may be the presentation. Cephalic tetanus generally progresses rapidly to generalized tetanus and is associated with a severe course.

Neonatal tetanus is generalized tetanus of the neonate. It occurs in infants born to mothers who are inadequately immunized. The port of entry is usually the umbilicus, with increased risk if the umbilicus is cut with a nonsterile instrument or is packed with contaminated material, such as soil, dung, or clay. Symptoms manifest on day 3 to 9 of life.

The initial presentation is failure to feed in a child who previously fed normally. Neonatal tetanus progresses to generalized tetanus as described previously. The mortality rate of neonatal tetanus, when treated, is 25% to 90%. Thirteen cases of neonatal tetanus were diagnosed in the United States from 1992 to 2000. In these cases, 85% of the neonates had not been vaccinated because of parental religious or philosophical objections.

**DIAGNOSTIC TESTING**

The diagnosis of tetanus is clinical. The *Vaccine-Preventable Diseases Surveillance Manual* defines tetanus as “the acute onset of hypertonia or by painful muscular contraction (usually, initially of the jaw and neck) and generalized muscle spasm, without other apparent medical cause.” No laboratory tests confirm or refute the diagnosis of tetanus. The organism is rarely recovered from wounds and can be cultured from patients without clinical tetanus. Serologic testing to detect anti-tetanus antibody levels plays a small role in the diagnosis of the disease. Patients can develop tetanus with “protective” levels of antibody. The aim of testing is to rule out other causes of rigidity and spasticity. If tetanus is suspected, begin treatment immediately. Do not delay treatment, because no confirmatory test exists.

Check the patient’s electrolytes, primarily to evaluate for hypocalcemia. Order a strychnine level determination if concern exists about exposure to strychnine, with the understanding that illegally imported pesticides contain strychnine. Ask patients about pesticide exposure, and consider accidental ingestion in children.

Obtain a computed tomography scan of the head if an acute intracranial event is considered. A lumbar puncture is necessary only if meningitis or encephalitis is included in the differential diagnosis. Examination of cerebrospinal fluid is
MANAGEMENT

Management of tetanus has two aspects: prevention and treatment. Each time an EP sees a patient with a wound, the opportunity for prevention exists. Treatment of tetanus is multifaceted and includes systemic treatment of the toxin, supportive treatment of the muscle spasms, and wound care.

PREVENTION

Prevention through vaccination and proper wound care remains the mainstay of therapy for tetanus. Update the patient’s tetanus immunization, and administer tetanus immune globulin according to Centers for Disease Control and Prevention guidelines (Fig. 178.1). Maintain the patient’s current tetanus immunization status in cases of cellulitis, abscesses, eye injuries, chronic ulcers, burns, injecting drugs, minor abrasions, acute lacerations, punctures, and crush injuries (Figs. 178.2 to 178.4). Clean wounds to remove any contaminants.

Some patients have decrease in tetanus titers earlier than 10 years. For this reason, patients with wounds that are not clean or are more than minor should receive a tetanus booster at 5 years after the last booster. Clinical tetanus does not confer immunity. Therefore, survivors of tetanus require immunization when they are clinically stable. Pregnant women can receive tetanus prophylaxis, if indicated. No confirmed risk to the fetus has been determined from tetanus-diphtheria or tetanus immune globulin.

TREATMENT

Antitoxin Therapy

Administer tetanus immune globulin at a dose of 3000 to 5000 units intramuscularly to the pediatric or adult patient. Some sources recommend infiltration of some immune globulin around the wound site, if identified. Intravenous immune globulin has tetanus antitoxin and may be administered if tetanus immune globulin is not available in a reasonable amount of time.

Antibiotic Therapy

Metronidazole is the drug of choice for tetanus. Administer intravenously, 1 g every 12 hours or 500 mg every 6 hours, to adult patients. Administer intravenous metronidazole at 30 mg/kg/day, divided every 8 or 12 hours, to pediatric patients. Penicillin G is the second choice of antibiotics. Penicillin antagonizes GABA with unknown clinical significance. The dose of penicillin is 24 million units intravenously,

noncontributory in tetanus, except to rule out other disorders. Because of unpredictable muscle spasms, performance of lumbar puncture on a patient with generalized tetanus may require intubation and deep sedation or muscle relaxation.

The spatula test has been used to distinguish tetanus from other forms of spasticity. In this test, a blunt instrument such as a tongue blade is used to touch the oropharynx. A patient without tetanus gags and attempts to expel the instrument. In a patient with tetanus, the stimulus triggers masseter spasm, resulting in a reflex bite of the blade. Although this test is reported to have a sensitivity of 94% and a specificity of 100%, the results may not be applicable to the United States, where tetanus is rare.
the pediatric dose of penicillin G is 100,000 to 250,000 units/kg/day, divided every 6 hours. Erythromycin, doxycycline, tetracycline, chloramphenicol, and clindamycin are alternatives if metronidazole and penicillin are contraindicated.

Supportive Therapy

Muscle spasms are controlled with large doses of benzodiazepines to augment GABA activity. Continuous infusions improve effectiveness. Control pain with generous doses of morphine or another opiate, but avoid meperidine. If...
respiratory depression results from sedation, intubate the patient. Magnesium in a continuous intravenous infusion has been used as an adjunct to benzodiazepines in the treatment of muscle spasms. Magnesium contributes to respiratory depression. Autonomic instability is a late finding and is not likely to be treated in the ED. Sedation with morphine and maintenance of a quiet, low-stimulus environment are critical in decreasing autonomic instability. \[48,51,52\] Esmolol has been used to control hyperadrenergic states. Propranolol and labetalol are both linked to increased mortality. \[52,53\]

**Wound Therapy**
Débride necrotic wounds with wide margins, to remove the anaerobic environment and to arrest \*C. tetani*. Incise and drain abscesses. Débride necrotic tissue at abscess sites. Do not delay débridement or incision and drainage. Perform these procedures on an emergency basis.

PATIENT TEACHING TIPS
Inform all patients of the risk of tetanus for even minor, clean wounds.

Recommend routine immunization every 10 years, even without injury.

Encourage patients to record tetanus boosters in their own records and to notify their primary care doctor of vaccination boosters.

Update tetanus immunization before pregnancy and childbirth.

The tetanus-diphtheria booster is believed to be safe in pregnancy and is given if an acute indication exists.

**DISPOSITION**
Admit patients with tetanus to the intensive care unit. Consult a surgical service on an emergency basis if wound or abscess management requires surgical intervention.

**REFERENCES**
References can be found on Expert Consult at www.expertconsult.com.