Tetanus and Trauma: A Review and Recommendations

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Background: **This review covers the pathogenesis and treatment of the disease along with the reexamination of the current recommendations for prophylaxis against tetanus in the United States. Although tetanus is still a major problem worldwide, the incidence in North America has become almost negligible because of the highly effective primary immunization program. Recently, there have been no deaths reported attributable to tetanus in the United States in trauma patients who had received the primary childhood immunization. However, tetanus immunization and prophylaxis in the acute injury setting is frequently misused and misunderstood.**

Methods: **A review of the literature regarding tetanus.**

Results: **After review, the authors recommend tetanus toxoid in adults only if it has been more than 10 years since their last immunization. There is no urgency for the administration of tetanus toxoid in the acute setting, as it provides protection against the next injury and not the current injury. Tetanus-diphtheria toxoid is not required unless there are plans for the injured patient to travel to diphtheria-prone countries in the future, as the incidence of diphtheria is negligible in the United States.**

Conclusion: **The review of reported cases of tetanus demonstrates that it is not possible to clinically determine which wounds are tetanus prone, as tetanus can occur after minor, seemingly innocuous injuries, yet is rare after severely contaminated wounds. Tetanus immunoglobulin should be reserved for patients with wounds who had never received primary immunization against tetanus.**

Key Words: **Immunizations, Guidelines, Immunoglobulins, Tetanus, Tetanus antitoxin, Tetanus toxoid, Vaccination, Wounds.**

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In countries where primary immunization programs are not effective, tetanus remains a major problem, and approximately 800,000 to 1 million deaths from tetanus occur worldwide each year. Most tetanus cases and deaths occur in sub-Saharan Africa, and 40% of them are manifested as neonatal tetanus.[1] The high incidence is associated with the use of nonsterile instruments or poultices on the umbilical cord. Maternal antibodies can cross the placenta, and immunization campaigns that cover adolescent girls and young women have resulted in a dramatic decrease of neonatal morbidity and mortality. Neonatal tetanus cases peaked in 1988 with over 325,000 cases and since have decreased to approximately 150,000 cases per year worldwide.[2] In addition to the decrease in the tetanus cases, the overall case-fatality ratio declined from 91% to 11% during the same period.[3] However, in developed countries, tetanus has nearly been eradicated as the result of the successful primary immunization programs. The overall incidence of tetanus in the U.S. civilian population declined from 0.4 per 100,000 in 1947 to 0.02 per 100,000 during the latter half of the 1990s. Tetanus is now such a rare disease that few Western clinicians have ever observed it. Despite the worldwide prevalence of this problem, according to the World Health Organization, in 2002 there were only 27 cases reported in the United States, none in Canada, and 101 in Mexico.[4]

Although some feel that the childhood primary immunization may offer lifelong immunity, there is an increase in the incidence of tetanus as one gets older. In North America, 75% of tetanus deaths were elderly adults with inadequate immunity.[5–9] According to the Centers for Disease Control and Prevention (CDC), during the years 1995–97, 60% of the reported cases were in people older than 40, and 33% were in those older than 60. The vast majority (88%) of reported cases were in people who had never been vaccinated.[10] In the last 20 years, there have been no reported deaths from tetanus in people that have had the full primary series, except in one case of an intravenous drug user.

HISTORY

Hippocrates was the first to describe tetanus.[11] After that, there was little learned regarding the science of this condition until 1884, when Carle and Rattone injected infected rabbits with pus from a fatal tetanus-causing human wound, which induced tetanus symptoms. In that same year, Nicolaier produced the same results by inoculation of soil into animals. It was 5 years later that Kitasato first cultured the agent in Koch's Berlin laboratory and also determined that tetanus resulted from the action of a systemic toxin. He and von Behring began to experiment with the antitoxin, which was produced by injecting mice with minute amounts of *Clostrid-*

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ium tetani.[12] This type of work on animals eventually led to the development of an antiserum for use in humans in 1897.[13] Despite the progress made in the understanding of tetanus, it remained a major public health issue in the early 20th century, exemplified by the 406 tetanus deaths after 3,983 injuries caused by fireworks during the Fourth of July holiday in 1903. This led the American Medical Association to call for a ban on hand fireworks.[14]

Prophylaxis against tetanus began in World War I, by injecting wounded soldiers with tetanus antiserum. The immunization program finally started in 1924, when the tetanus toxoid (Tt) (heat-inactivated toxin) was finally developed and used.[14] By 1941, the Allies had primary immunization programs in place using Tt. In contrast, tetanus was still a major problem during the treatment of civilians, as they did not have an immunization program. During the battle for Manila in 1945, Colonel Glenn of the U.S. Army reported of his experience in civilian casualties that: "in my clinical experience I have never seen such a terrifying disease as tetanus."[15] During this battle, 12,000 civilians were wounded and 40 in 1,000 developed tetanus. All U.S. Army dog tags from this period bore the date of the soldier's tetanus immunization.[16] Tetanus among U.S. troops declined from a rate of 2,000 per 1 million in the Civil War to 6 per 1 million in World War II.[17] The U.S. Army during World War II recorded only 12 cases of tetanus, 5 fatal, among 2,734,819 hospital admissions.[15,18] In the developed world, the success of tetanus immunization during World War II led to the incorporation of Tt into routine civilian immunization regimens after the war. Many persons born before World War II never received a primary vaccine series unless they served in the military, and older women are particularly likely to have missed out on receiving the series.

PATHOGENESIS

Tetanus is caused by two toxins secreted by *Clostridium tetani*, a gram-positive, spore-forming, obligate anaerobic bacillus. The primary toxin is a neurotoxin known as tetanospasmin, which causes the characteristic muscle spasms of the disease. The role of the second toxin, tetanolysin, is still not well understood.[19] Tetanospasmin prevents the release of γ-aminobutyric acid (GABA) at the junction of inhibitory nerve synapses.[20] Lack of GABA prevents inhibition of sustained excitatory nervous impulses, causing the clinical signs of tetanus. *C. tetani* forms a stable terminal spore that is resistant to moisture, to some chemical disinfectants, and to variations in temperature, including boiling. *C. tetani* is a common soil bacterium, and its spores are ubiquitous in nature and are found in the soil and in the intestines and feces of domestic animals and humans.[21]

Vegetative bacteria grow optimally at 37°C. Wounds contaminated by soil and with low oxygen tension are optimal locations for germination of *C. tetani*. The spores are noninvasive and require a skin break for germination.[22] The incubation period for tetanus varies from 1 or 2 days to a month or more; most cases have onset of symptoms within 7 to 14 days of the initial injury.[23]

Fig. 1. *Opisthonus. (From Lunds J. Tetanus: contracted body of soldier suffering from tetanus. In: Bell C.* The Anatomy and Philosophy of Expression as Connected with the Fine Arts. *5th ed. London: Henry G. Bohn; 1865 [public domain]).*

Clinical Manifestations

"Tetanus" is derived from the Greek word for stretching or rigidity. A feature of tetanus is painful muscular contractions that are sometimes violent enough to cause fractures. The muscular contractions of tetanus can be either localized or generalized. In abdominal surgical wounds causing tetanus, abdominal rigidity is common as a first sign. Contraction can also begin in the muscles surrounding other wound locations. In generalized tetanus, muscles of the jaw and neck are often affected.[17] Generalized tetanus is the most common form and is seen in 80% of cases. Its manifestation includes pain, headache, and muscle rigidity. Trismus is often the presenting symptom, caused by severe contraction of the masseter, leading to the lay term "lockjaw." Classic advanced signs of tetanospasm are opisthotonus (Fig. 1) and risus sardonicus, literally the sardonic smile.[17]

The disease may progress to laryngeal obstruction and a reduction in chest wall compliance, causing respiratory failure. Respiratory failure is the most common direct cause of death from tetanus.[21] Manifestations of autonomic instability are diverse, including hypertension or hypotension, diaphoresis, cardiac dysrhythmias, and hypermetabolism. The disease has a prolonged course, typically needing weeks to months of supportive management to resolve. The spasms and autonomic instability are usually most prominent in the first few weeks, peaking near the second week, and then resolving. However, the muscular rigidity may persist for several months.[17,24]

The diagnosis of tetanus is made on a clinical basis. Laboratory tests are used to rule out other diseases. Wound cultures are positive for *C. tetani* in only 30% of documented cases.[24] Optimal management of severe tetanus with spasms and fevers beyond supportive measures such as ventilation is hampered by a lack of clinical evidence.[25–27] Use of Tt, tetanus immunoglobulin (TIG), or intrathecal TIG have not

Table 1 Current ACIP Recommendations for Tetanus Administration for Wounds

History of adsorbed tetanus toxoid (doses)	Non-tetanus-prone wounds		Tetanus-prone wounds	
	Td[a]	TIG	Td	TIG
Unknown or less than three doses	Yes	No	Yes	Yes
Three or more doses[b]	No[c]	No	No[d]	No

Td, tetanus and diphtheria toxoids adsorbed (for adult use); TIG, tetanus immune globulin (human).

[a] For children younger than 7 years old: diphtheria tetanus pertussis (DPT, DPaT) vaccination (DT, if pertussis vaccination is contraindicated) is preferred to tetanus toxoid alone. For persons 7 years old or more, Td is preferred to tetanus toxoid alone.

[b] If only three doses of fluid toxoid have been given, a fourth dose of toxoid, preferably an adsorbed toxoid should be given.

[c] Yes, if more than 10 years since last dose.

[d] Yes, if more than 5 years since last dose.

Tetanus toxoid and TIG should be administered with different syringes at different sites.

decreased mortality, although some studies show that intrathecal TIG reduces severity and duration of disease.[28,29] Removal of the source of infection is an obvious intervention. Spasms and muscle rigidity are usually managed with benzodiazepines to augment $GABA_A$ receptor agonism. Although respiratory failure is managed by ventilation, it may be accompanied by severe autonomic instability, as manifested by severe hypertension and tachycardia, with plasma catecholamine levels 10 times normal levels, alternating with profound hypotension, bradycardia, or recurrent cardiac arrest.[30,31]

IMMUNIZATION AND PROPHYLAXIS

The current primary immunization program has been successful in drastically reducing the incidence of tetanus in developed countries. A prospective observational case series study revealed a seroprevalence of tetanus immunity of 90.2% in 1,988 patients presenting to five U.S. urban emergency departments with acute wounds.[32] Tetanus immunization is part of the five diphtheria-tetanus-pertussis (DTP) immunizations given at the ages of 2, 4, and 6 months; 12 to 18 months; and 4 to 6 years. In 2000, results of the National Immunization Survey indicated that 94% of children aged 19 to 35 months had received three doses of Tt.[33] Boosters are then recommended at 10-year intervals starting at age 11 to 12.[34,35] Although 91% of children 6 to 11 years old have immunity to tetanus, this drops to 31.0% for those over the age of 70. In contrast to the high vaccination rates among young children, the 1998 National Health Interview Survey indicated that only 40% of adults aged ≥65 years had received a booster dose of Tt during the previous 10 years.[13,36] Thus, the Advisory Committee on Immunization Practices (ACIP) Task Force on Adult Immunization supports a second option for tetanus-diphtheria toxoid (Td) use in adults: a single Td booster at age 50 for persons who have completed the full pediatric series, including the teenage/young adult booster.[37] Although the efficacy of Tt has never been studied in a vaccine trial, its worth cannot be cannot be argued, as the incidence of tetanus is extremely rare in persons whose last booster dose was within the last 10 years.

Certain groups of the U.S. population have a lower incidence of serologic immunity to tetanus.[38] There is no statistical difference between blacks and whites in prevalence of immunity to tetanus at 73%; however, Hispanic-Americans had a 65.7% prevalence of immunity and foreign-born persons had a 51% prevalence of immunity. Mexican-Americans 20 to 49 years of age who were born outside the United States were seen to be a high-risk group for tetanus. Women at all ages are less likely than men to have serologic immunity to tetanus.[39] Although more than 80% of women are immune to tetanus up to 39 years of age, this declines to 23% at age 60 or greater. Foreign-born Mexican-American women had significantly lower seropositivity to tetanus than individuals in the other racial/ethnic groups, including U.S.-born Mexican=American women, with only 12% seropositive to tetanus at age 50 to 59. Although poverty index did not prove to be a significant factor for seropositivity, educational level was significant, with 77.4% college-educated persons being seropositive, dropping to 44% for those with elementary school or less.[38] Groups identified as having significantly lower rates of immunity were persons aged 70 years or older at 59.5% seropositivity, immigrants from outside North America or Western Europe with 75.3% seropositivity, persons with a history of inadequate immunization with 86.3% seropositivity, and persons without education beyond grade school with 76.5% seropositivity.

Tetanus-Prone Wounds

The current recommendations for tetanus wound prophylaxis are given in Table 1.[39] The first issue with this recommendation is that wounds should be categorized as either "tetanus-prone" or "non–tetanus-prone." Current guidelines suggest that wounds that are oxygen deficient are tetanus prone, as *C. tenani* is an obligate anaerobe. Thus, wounds that have been crushed, devitalized, or contaminated with dirt or rust are believed to be more prone to tetanus. Wounds such as open fractures, punctures, and abscesses are also thought to be more tetanus prone. Such guidelines would suggest that severe wounds are more tetanus-prone. However, the literature demonstrates that both minor and major wounds alike can cause tetanus. In a Mayo Clinic report of their 25-year experience with tetanus, the cause included a wide variety of wounds that were both minor and major, such as scratches while gardening, stepping on a nail, stepping on a rake, head

laceration after a fall, elbow abrasion, Fournier's gangrene, and necrotizing lung mass.[40] In 1987–88, of the 99 cases of tetanus reported in the United States, 41% were caused by farming or gardening activities.[41] Other causes of tetanus reported included body piercing, tattooing, elective hernia surgery, skin ulcers, animal bites, and abscesses. One patient actually got tetanus from an ulcer after biting his own tongue. Minor trauma was responsible for 30% of reported tetanus cases.[40,42] Some cases of tetanus do not even have an identifiable source of acute injury.[23] Given the wide variety of wounds that cause tetanus, wound severity alone does not make a wound tetanus-prone. Reports of tetanus after gunshot wounds or stab wounds in the United States are extremely rare. During a 4-year period (1998–2000), the CDC reported only one case of tetanus from a gunshot wound and one case from an open compound fracture in the United States.[43] In an analysis of Vietnam War wound infections, no cases of tetanus were reported.[44] Therefore, clinical distinction between tetanus-prone wounds and non–tetanus-prone wounds is not feasible.

California reported that 40% of the tetanus cases were caused by injecting drug use (IDU) (mostly heroin) during 1987–97.[45–47] Hispanics constituted 60% of all patients with tetanus reported in California and 89% of IDU-associated cases. Mexican-Americans are the predominant Hispanic population in California. During this period, 58% of Mexican-Americans, compared with 73% of non-Hispanic whites, had protective levels of antibody to tetanus.[7] This increased susceptibility may, in part, explain the disproportionate occurrence of tetanus among Hispanic injecting drug users. Despite the lower antibody levels in Mexican-Americans, there were only 27 cases of tetanus in the United States reported during 2002, compared with 101 cases in Mexico.

Immunization

Tetanus disease itself does not impart immunity, as the amount of tetanospasmin toxin required to produces disease is minute. Tetanus toxoid does not induce immediate immunity, but produces antibodies in a manner that is typically delayed, at least 4 days for clinically detectable levels and often weeks later in the elderly.[48] The U.S. Army tetanus cases in World War II showed no difference in survival when an "emergency" Tt immunization was given after wounding.[49] Receiving toxoid acutely after the traumatic wound does not ensure protection from tetanus. Giving Tt in the emergency department after a wound does more for tetanus immunity for the next injury rather than the current injury. In the United States in 1987–88, of those tetanus cases who sought medical care for their injury, 52% received Td but still contracted tetanus. Conversely, of the 60.9% of patients that required tetanus wound prophylaxis by ACIP guidelines, less than 57.6% received it during the 1980s and 1990s.[41]

For adults, the Td booster shot is recommended by the ACIP, National Coalition for Adult Immunization, and other groups, for those who have not had a Td booster shot in the previous 10 years or have recovered from tetanus disease or for those who have never received immunization against tetanus (Table 3).[50] The recommendation for routine 10-year boosters is somewhat controversial because a previous study demonstrated those revaccinated less than 20 years after primary vaccination attained very high antitoxin concentrations (above 6 IU/mL), corresponding to a duration of immunity of at least 20 years.[51] A cost-benefit analysis recommended use of a routine tetanus booster at age 65 in place of the current decennial adult booster for the United States.[52] However, in Canada, a region of high seropositivity and very low incidence of tetanus, routine immunization of the elderly was deemed not to be cost effective.[53]

Many trauma patients cannot communicate their tetanus status and often are given a booster routinely if they have any kind of wound. However, there are considerations of cost and adverse events associated with the vaccine. Approximately 16 million doses of Td are administered annually in the United States. It is estimated that half of the doses are used for decennial boosters and the other half for the management

Table 2 Complications of Tetanus Toxoid Administration

Pain tenderness	8–13,600,000 cases (50–80%)
Erythema and swelling	4–4,800,000 cases (25–30%)
Local reaction	320,000 (2%)
Anaphylaxis	25 cases
Brachial plexus neuropathy	80–160 cases
Guillain-Barré syndrome	6.4 cases

Table 3 Recommendations for Management of Traumatic Wounds:

1. All wounds should be cleaned and debridement should be undertaken if necessary.
2. A diligent effort should be made to obtain the patients' history of tetanus immunization if possible.
3. Tetanus toxoid (Tt)* should be administered if the history of the last booster was greater than 10 years. If the history is not available, Tt may be considered when convenient.
4. If the history demonstrates that the last immunization was over 10 years ago, then Tetanus Immune Globulin (TIG)† should be administered. The severity of the wound should not be a factor in the administration of TIG.

* Tetanus toxoid (Tt) Dosing (See product insert):
Age ≥ 7: 0.5 ml (5 IU) IM.
Age < 7: Use Pediatric DTP or DTaP instead of Tt. If a contraindication to pertussis immunization exists, use Pediatric DT. Typical dose is 0.5 ml IM.

† Tetanus Immune Globulin (TIG) Dosing (See product insert):
Adult prophylaxis: 250–500 U IM in opposite extremity to tetanus toxoid.
Pediatric prophylaxis: 250 U IM in opposite extremity to tetanus toxoid.
(Note: Dosages in used clinical tetanus typically are 3,000–10,000 U IM).

of tetanus-prone wounds. The estimated number of complications associated with administering 16 million doses of Td per year are listed in Table 2.[34,50]

The cost of a dose of Td is approximately $3.50, which equates to $56 million worth of Td doses per year in the United States. There are charges for the administration of the shots as well. Assuming a nominal charge of $50.00 per administration of 16 million doses, the cost to the health care system would be $800 million annually.

A Td shortage existed during the end of 2000 when the two manufacturers that provide Td stopped production for a short period of time.[54] Only recently has increased supply of Tt and Td in the United States allowed the resumption of routine immunization.[55–56] To date, there have been no reports of an increased number of tetanus cases reported after the period that toxoid was in short supply.[57]

Although there is convincing evidence that the primary series of DPT immunizations is very useful, the evidence for routine Tt administration after traumatic wounds is lacking. There is no doubt that, over time, the antitoxin level decreases. However, the epidemiologic data strongly suggest that the protective antitoxin level is not nearly as reliable as the history of full primary tetanus immunization in predicting protection from the disease. Additional evidence shows that the protective effect of the primary immunization series may be lifelong. A report reviewing the data from the CDC found that there were no deaths from wound tetanus in those who had the complete primary series.[55,58] In 2002, a death from tetanus was recorded in a 55-year-old with a history of IDU who had received three doses of Tt more than 11 years previously.[43] There is an emergence of recommendations based on cost-benefit analysis, and this recommendation is that for adults, one midlife booster at the age of 50 to 65 is all that is required.[59]

The other component of Td is the diphtheria vaccine. Most adults do not comply with the decennial booster recommendation and approximately half do not have the protective level of antitoxin (0.1 IU/mL) against diphtheria, yet this disease remains of negligible concern (one to five cases annually), ranking behind plague, cholera, and tularemia.[10] During the Td shortage, the CDC recommended Td only if the person was planning to travel to diphtheria-prone areas such as certain parts of Asia, Africa, the former Soviet Union, or South America.[59]

Prophylactic Treatment

Tetanus immunoglobulin provides temporary immunity by antibodies directly binding toxin. This ensures that a protective level of antitoxin is achieved even if an immune response has not yet occurred. TIG is recommended in current guidelines for cases of tetanus and in tetanus-prone wounds when the history of tetanus immunization is unknown or if a primary immunization series was not completed.[34,60] As discussed previously, because a distinction cannot be definitively made between tetanus-prone and non–tetanus-prone wounds, theoretically, one would have to administer TIG to all patients presenting with a wound without a known complete immunization series. However, in a U.S., urban, five-emergency room study, TIG with toxoid was provided to none of 504 patients who gave a history of inadequate primary immunization with wounds described as tetanus-prone.[32] This indicates a significant gap between guidelines and practice in the use of TIG.

SUMMARY AND RECOMMENDATIONS

Recommendations are summarized in Table 3. The primary DPT immunization program is effective against tetanus, has been successful, and should be continued without question. Because Tt booster is a part of the immunization process, a diligent effort should be made to determine primary immunization history before administration. There is no urgency to provide Tt in the acute setting, as the administration of Tt immunizes the patient against the next wounding event but does not ensure tetanus prevention from the acute injury. Persons discovered not to have completed the three-shot primary series should do so. Persons with traumatic wounds without a complete primary immunization history or booster within 10 years should receive TIG with Tt. Because Tt and Td administration can have side effects and cost issues, unnecessary usage should be avoided. The patient should be immunized against diphtheria only if the patient is planning on traveling to a diphtheria-prone country.

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Book Review

Book Review – TOP KNIFE

Many of our colleagues trained in internal medicine or medical specialties perceive trauma surgeons to be "cowboys"; some would even say "loose cannons." This book, "Top Knife," and in particular the title, will do little to dissuade anyone from these perceptions. This might even preclude some surgeons from reading this book, and this is unfortunate, since there are a number of "pearls" within the chapters and the book clearly reflects the extensive experience of the authors.

In 1975, Alexander Walt and Robert Wilson took a similar approach in a book entitled, "Management of Trauma: Pitfalls and Practice." They avoided the jargon of the jungle, Japanese Bandits, or any implication that there is a wounded surgical soul in the right upper quadrant of the abdomen. Similar to "Top Knife," there were a number of highlighted pitfalls throughout the text lending emphasis to the preceding text. Many of these in "Top Knife" are surgical truisms.

I did find a few areas in which I have minor disagreements; two things in particular stood out as detracting from the overall good qualities of the book. The first is what I consider an overemphasis on the use of damage control and essentially no discussion on how the surgeon should prevent the unhappy triad of acidosis, coagulopathy and hypothermia, which may lead to the need for damage control. Both of the authors are experienced vascular surgeons and know the pathophysiology of reperfusion injury and are aware of preventive measures. The second area of disagreement is on page 3 of the manuscript in the introduction. The authors state, "Our focus is not so much on how you should be sewing, but rather how you should be thinking and reacting. These skills are rarely, if ever, taught in surgical training." I think there are a number of academic trauma surgeons, including myself, who take umbrage with the second sentence.

Finally, on page 71 of the manuscript, they refer to a quote from Harold Burrows: Another truism. We have all made errors, and that is how we gain experience. Teaching experience is the hallmark of a good surgical educator. The authors have shared their experience in a pithy, and sometimes flamboyant style. They have achieved a vainglorious treatise, which I will recommend to my residents.

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