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# Prevention of Tetanus after Medical and Surgical Procedures: A Narrative Review

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## Abstract

The World Health Organization was committed to eliminating neonatal tetanus by 1995. Three years after this date, the infection killed over 400,000 babies a year, even though a safe, effective vaccine had been available for most of this century. The frequency of tetanus in the developing world epitomizes the healthcare disparity between the developed and the developing world. Consequently, the priority of the medical profession must be prevention, with the development of simpler immunization schedules with longer protection. Consequently, the purpose of this collective review is to provide an overview to the management of tetanus as well as to review the immunization strategy that will prevent this potentially deadly illness. Tetanus is caused by *Clostridium tetani*, which is an obligate anaerobic, gram-positive rod that is motile and readily forms endospores. Although *C. tetani* is located everywhere, the disease is encountered largely in underdeveloped, overcrowded, and economically disadvantaged countries. *C. tetani* is widespread in the feces of domestic animals and humans, while spores of *C. tetani* are abundant in soil and in the environment surrounding the habitation of humans and animals. Tetanus usually follows deep penetrating wounds where anaerobic bacterial growth is facilitated. Three basic forms of tetanus may be distinguished: local, cephalic, and generalized. At least 80% of the cases are the generalized form. In the adult patient, the most characteristic sign of generalized tetanus is lockjaw, or trismus. The diagnosis of tetanus is most frequently made on clinical manifestations, rather than on bacteriologic findings. The three objectives of management of tetanus are: (1) to provide supportive care until the tetanospasmin that is fixed in tissue has been metabolized; (2) to neutralize circulating toxin; and (3) to remove the source of tetanospasmin. Because there is essentially no immunity to tetanus toxoid, the only effective way to control tetanus is by prophylactic immunization.

**Keywords:** *Tetanus, Lockjaw, Clostridium, Prevention, Immunoglobulin*

## Introduction

Tetanus with its baneful outcomes has been diagnosed for the reason that time of Hippocrates. In the United States about 200 cases according to year are recorded, the highest occurrence being in non-whites inside the southern states. Worldwide, there are probably 300,000 to 500,000 instances each year, with a mortality rate of about 45 percent [1]. Trismus is a reasonable normal for this issue, be that as it may it is additionally a side effect which the dental practitioner much of the time experiences in association with intense oral contaminations, injury, and the temporomandibular issue disorder [2].

Tetanospasmin, or tetanus toxin, is discharged from *Clostridium tetani* spores that exist wherever in the soil. It is a standout amongst the most powerful poisons in essence 240 g is evaluated to be adequate to murder the world's population [3]. *C. tetani* spores more often than not infiltrate the tissue through an infiltrating defiled injury, and posterior to time of anaerobic hatching, they progress toward becoming bacilli that discharge tetanospasmin. The poison infiltrates peripheral neurons and ventures retrograde up axons to the central nervous system. It can enter sensory, motor, and autonomic neurons, in spite of the fact that radio-labeled examines have demonstrated an inclination for inhibitory neurons. The greater part of the clinical illness comes about because of motor and autonomic hindrance. Tetanus toxin is a metalloprotease substance which obeys at the presynaptic layer to separate synaptobrevin, a protein that enables combination of neurotransmitter vesicles to nerve films. At the point when this combination procedure is forestalled, neurotransmitters are not discharged into the neurotransmitter and neurotransmission is blocked. Midway, transmission along the inhibitory gamma-amino butyric corrosive (GABA) and glycinergic neurons is interfered, and at the spinal cord, inhibitory interneurons are deflected.<sup>3</sup> It is vital to perceive that tetanus isn't an infection, and in this manner patients don't as a rule encounter indications of the sickness until the point that the poison is scattered and comes to the interneurons [4]. Early treatment with invulnerable globulin is basic,

since it ties free poison. Nonetheless, it doesn't treat the impacts of poison secured inside the neuron or cell body. Treatment of the impacts of poison as of now inside the sensory system is simply strong, as will be depicted further. Clinically, the impact of the poison on the neuron keeps going around 4–6 weeks and might be irreversible until the re-development of another nerve terminal; notwithstanding, the component of practical recuperation isn't clear [4,5].

## Clinical features:

Tetanus more often than not happens following a profound infiltrating wound where anaerobic bacterial development may happen, especially if the injury is sullied by soil, fertilizer or corroded metal [6]. It might likewise happen by different instruments, for example, tattooing, intramuscular infusions, septic premature births, poor dentition, ulcers, needle therapy, ear penetrating, burns, circumcisions, perpetual otitis media, and snakebites. In up to 30% of cases the gateway of section can't be recognized. The brooding time frame (time of immunization to the primary indication) is generally 7-10 days (run 1–60 days), and the time of beginning (time from first manifestations to the begin of fits) is 1–7 days. The severer types of the illness have shorter brooding periods and times of beginning. The clinical picture is ruled by muscle fits, unbending nature, and autonomic disturbances [6,7]. Features such as sore throat, Neck solidness, and trouble opening the mouth are the most punctual highlights. Masseter fit causes tetanus (trismus), with the fits reaching out to the facial muscles to cause a "risus sardonicus (Facial expression). Muscle fits may prompt laryngeal hindrance and also diminished chest divider consistence, bringing about respiratory trade off. Association of pivotal muscles prompts neck augmentation, and truncal unbending nature may prompt opisthotonus. Back torment, trismus, muscle firmness, and dysphagia are normal [8]. The long winded fits have a tendency to be greatly excruciating and influence agonist and enemy muscle bunches together, and might be unconstrained or jolt touchy (activated by touch, visual, sound-related, or

enthusiastic boosts). These fits may seem convulsive in essence and be sufficiently fierce to cause breaks or ligament separations. Inflexibility is most unmistakable in muscles neighboring the entryway of section. The autonomic unsettling influences can prompt pyrexia, tachycardia, labile hypertension, gastric stasis, diarrhea, bountiful sweating, and unreasonable bronchial emissions. The hypertension is prevalently caused by increments in fundamental vascular protection auxiliary to raised centralizations of flowing catecholamines like those found in pheochromocytoma [9]. Expanding muscle fits and inflexibility describe the primary seven day stretch of disease. Autonomic unsettling influences generally begin a couple of days after the fits and achieve a top amid the week, and endure for 1-2 weeks. Fits begin to die down following 2-3 weeks, yet the muscle unbending nature may proceed long after the fits and autonomic contribution have died down. Muscle inflexibility may last up to 6–8 weeks in serious cases. Different evaluating frameworks of seriousness of tetanus have been portrayed, a standout amongst the most usually utilized being the Ablett framework (table 1). Other scoring frameworks (for instance, Dakar and Phillips scores) have likewise been contrived to evaluate general prognosis [10,11].

#### **Tetanus infection following surgical and medical procedures:**

Both exogenous and endogenous sources have been ensnared in illness improvement. Equipment or dressings hardware that are deficiently cleaned and even tidy have been associated as conceivable causes with postsurgical tetanus-infection [12,13]. Mackie et al. 1928 detailed 9 cases and 8 passings in Edinburgh, in which tainted catgut was observed to be the vehicle of disease [14]. Robinson et al. 1946 depicted 2 cases in which tainted clean in the working room (OR) was considered as a wellspring of contamination [15]. The sterilizer was observed to be proficient and tests of the catgut, saline, dressings and powder utilized turned out to be sterilized. In the primary case, tests of clean from the floor, divider and fan gathered the day following the embroiled surgery uncovered tetanus bacilli. A free study of the room 2 weeks after the fact, after it had experienced watchful purification, uncovered no further tetanus bacilli. In the second case, it was tests

**Table (1): Ablett classification of tetanus infection**

Grade	Severity	Symptoms
<b>I</b>	<b>Mild</b>	Mild trismus, general spasticity, no respiratory compromise, no spasms, no dysphagia
<b>II</b>	<b>Moderate</b>	Moderate trismus, rigidity, short spasms, mild dysphagia, mild respiratory involvement, respiratory rate > 30 breaths/min
<b>III</b>	<b>Severe</b>	Severe trismus, generalized rigidity, prolonged spasms, severe dysphagia, apneic spells, pulse > 120 beats/min, respiratory rate > 40 breaths/min
<b>IIII</b>	<b>Very severe</b>	Grade 3 with autonomic dysfunction

of tidy gathered on sterile swabs that yielded tetanus bacilli [16]. Robinson's group hence recommended measures to prohibit contaminated clean, including wet-tidying of OR dividers with germicide arrangements, a sufficient sifting framework and precautionary measures against the presentation of tidy on footwear. Sevitt et al. 1948 portrayed 2 different situations where goat hair that was utilized to get ready mortar for repairs in the OR was polluted with tetanus spores. In a 1957 episode in the United Kingdom of 5 postoperative cases, tetanus spores were segregated from sterilized gloves [15].

In the vicinity of 1% and 10% of individuals express *C. tetani* in their stool. Wainwright et al 1926 assessed more than 3000 instances of tetanus and portrayed around 12% as postoperative. Most happened after stomach or gynecological surgery, and in the dominant part the intestinal tract was viewed as the wellspring of contamination. As a rule, be that as it may, firm proof of the source included was deficient. It is deserving of note that much of the time considered by

Wainwright, tetanus took after surgery that did not enter the intestinal tract: 29 stomach hysterectomies, 78 oophorectomies and 38 inguinal hernia repairs. 69 cases (19%) included operations on the gastrointestinal tract, of which 23 were for hemorrhoids. Calvert et al 1942 detailed an instance of tetanus in a 57-year-old lady in whom life forms were separated from the substance of a resected example of ileum [16].

### **Prophylaxis of tetanus infection following dental procedures:**

Three clinical types of tetanus are perceived. Nearby tetanus is unprecedented and is restricted to fit of a solitary anatomic region. It might advance to a more serious frame yet is once in a while lethal. Cephalic tetanus is characterized by the inclusion of cranial nerves as it were. It is uncommon and again not normally deadly. The most widely recognized and serious shape is summed up tetanus, which represents roughly 80% of announced cases [14]. The run of the mill arrangement of signs and side effects is trismus, trailed by dysphagia and neck firmness, advancing to stomach and respiratory muscle contribution. Laryngospasm and fit of respiratory musculature prompts respiratory pain. Cracks of the long bones and spine have been accounted for from withdrawals. Generally observed autonomic indications incorporate hypertension, arrhythmias and at times, heart failure. Introductory treatment incorporates careful debridement of the injury and organization of tetanus promoter. Treatment of summed up tetanus ordinarily incorporates organization of 3000-6000 units of tetanus invulnerable globulin, metronidazole and intravenous penicillin G, profound sedation and neuromuscular barricade with ventilatory help as required. 2 Spasms can happen for 3-4 weeks, with finish recuperation taking months [15].

Professionals ought to ask about a patient's immunization status on the off chance that they have acquired delicate tissue damage especially in the event that it has been debased with feces, soil, dirt, or salivation. Wounds caused by burns, crushing, tears, frostbite and punctures are likewise powerless to tetanus. Quick tetanus immunizer testing or immunochromatographic dipstick test can help decide

vaccination status when one can't acquire a solid history [15,16]. Following an oral injury or peri-oral delicate tissue damage, especially in a patient with poor oral cleanliness, it is fitting that the specialist decide the patient's inoculation status and conceivable requirement for a tetanus supporter (Table 2) or prophylactic TIG organization 250 units IM. Intra-oral injuries are innately in contact with spit and along these lines considered a debased injury. Facial injuries and oral injuries have been embroiled as the primary entryway of section for tetanus, and in this manner ought not be ignored [18-19]. Tetanus immunoglobulin will inactivate unbound poison, yet once the poison achieves the nerve endings the coupling is irreversible. Organization Tdap sponsor ought to happen at the earliest opportunity following vaccination, yet late showing wounds ought not be neglected as the brooding time frame ranges from one day to a while, with the normal being eight days [20].

### **Prophylaxis of tetanus infection following dental trauma:**

Tetanus has been described in affiliation with surgical techniques and oral sepsis [21]. The circumstance may additionally gift to the dental health care professional within the first example because trismus is regularly the primary symptom [22]. It is essential to do not forget that trismus may be brought on no longer only by way of local sepsis however by tetanus as properly. A dynamic vaccination program makes this a preventable disorder. Commonly the disorder should be dealt with via eliminating the organisms, neutralization of the toxin and control of the muscular spasm in conjunction with the symptomatic treatment of the breathing and cardiovascular structures. Prophylaxis is accomplished through active immunization which, in adolescence, begins with infusions of a triple antibody (diphtheria, pertussis, tetanus antigens). The first injection is given at three months of age, the second one at 18 to 20 weeks and the very last at six months. Immunity is stated to last ten years, booster injections being given at 5 to 10 years intervals [23]. Human anti-tetanus immunoglobulin is to be had and to be potent should be given early within the course of the ailment. It confers protection for at least 4 weeks but is weak after the toxin turns into bound to apprehensive tissue [24].

Impulsive re-eruption has been counseled with the aid of Ellis et al 1940 as the treatment of desire, for intrusive luxation to save you further disturbance to the apical and marginal periodontal tissues [25]. it is primarily based on the truth that lots of those enamel in particular ones with incomplete root formation do erupt on their own [26]. however recently Faria et al. have shown that even tooth with mature apices re-erupt spontaneously [27]. The negative aspects of this technique are two-fold: One, periodontal surgical procedure- e.g. gingivectomy is wanted to gain access to the basis canal while waiting for spontaneous re-eruption. Second, root resorption or ankylosis can also arise all through the commentary duration.

### Conclusion:

Indeed, even in the hands of those most experienced with tetanus, there is by all accounts little proof construct agreement in light of treatment conventions. Late upgrades in treatment in the underlying periods of tetanus introduce new difficulties for doctors, as patients are progressively making due to the further developed phases of the sickness. Tetanus status and fitting tetanus antibody as well as tetanus immunoglobulin ought to be all around looked into and oversaw by all medicinal services suppliers that experience delicate tissue damage. Auspicious organization of tetanus toxoid and additionally immunoglobulin is basic to keep the beginning of summed up tetanus as plot for this situation report. Oral injuries ought not be neglected as a potential wellspring of vaccination, particularly in those patients with prior poor oral wellbeing.

### Conflict of interests

The authors declared no conflict of interests

### References

- Harrison's Principles of Internal Medicine, ed. 7, New York, 1974, McGraw-Hill Book Company, Inc., pp. 845849.
- . Taylor AM. Tetanus continuing education in anaesthesia. *Critical Care Pain* 2006; 6: 101–4.
- Mellanby J, Green J. How does tetanus toxin act? *Neuroscience* 1981; 6: 281–300.
- Bleck TP. Pharmacology of tetanus. *Clinical Neuropharmacol* 1986;
- Farrer JJ, Yen LM, Cook T, et al. Tetanus. *J Neurol Neurosurg Psychiatry* 2000;69:292–301. c Excellent review article containing information on the structure and action of the tetanus toxin, the clinical features of tetanus, and useful details on the management of the condition.
- Thwaites CL. Tetanus. *Practical Neurology* 2002;2(3):130–7. c Recent article containing useful information on the clinical features and management of tetanus.
- Robinson OT, McLeod JW, Downie AW. Dust in surgical theatres as a possible source of post-operative tetanus. *Lancet* 1946; I:152-4. [PubMed].
- Holmes NM, Smellie GD. Tetanus: the potential for infection in the environs of a Glasgow hospital. *Scot Med* 1966;11:391.
- Mackie TJ. Post-operative tetanus: report to the Scottish Board of Health. *Br Med J* 1928; i:987-8.
- Sevitt S. Source of two hospital-infected cases of tetanus. *Lancet* 1949;11:1075-8. [PubMed]
- Mackay-Scollay EM. An outbreak of postoperative tetanus. *Proc R Soc Med* 1958; 52: 110. [PMC free article] [PubMed]
- Wainwright JM. Tetanus: its incidence and treatment. *Arch Surg* 1926;12:1062-79.
- Clay RC, Bolton JW. Tetanus arising from gangrenous unperforated small intestine. *JAMA* 1964;187:856-8. [PubMed]
- Centres for Disease Control and Prevention: Tetanus. In: *The Pink Book: Course Textbook*. Vol 13th Editi., 2015:341–352.
- Chithra A, Cariappa KM, Kamath AT, Kudva A: Role of Rapid Tetanus Antibody Test in Accident and Emergency Department. *J Maxillofac Oral Surg* 14: 784, 2015.
- Hatamabadi HR, Abdalvand A, Safari S, Kariman H, Dolatabadi AA, Shahrami A, Alimohammadi H, Hosseini M: Tetanus Quick Stick as an applicable and cost-effective test in assessment of immunity status. *Am J Emerg Med* 29: 717, 2011.
- Alhaji MA, Mustapha MG, Ashir GM, Bashir MF, Pius S: Tetanus following tongue bite from

repeated convulsions: a case report. *Trop Doct* 42: 180, 2012.

18. Paz a. De, Izquierdo M, Redondo LM, Verrier a.: Cephalic tetanus following minor facial abrasions: Report of a case. *J Oral Maxillofac Surg* 59: 800, 2001.

19. Nolla M, Diaz RM, Garces J et al: Review of tetanus points of entry. Study of 229 cases. *Rev Clin Esp* 189: 101, 1991.

20. Smith AT, Drew SJ: Tetanus: A Case Report and Review. *J Oral Maxillofac Surg* 53: 77, 1989.

21. Graves, A. M. (1930). Tetanus in New Orleans. An analysis of 813 cases. *Annals of Surgery*, 92,1075.

22. Taylor, G. S. (1970). Tetanus presenting primarily as trismus. *British Journal of Oral Surgery*, 8,77.

23. DHSS Booklet (1982). Immunisation against infective disease. HMSO.

24. *British Medical Journal* (1967). Human Antitoxin. Leading article, 4, 634.

25. Oulis C, Vadiakas G, Siskos G. Management of intrusive luxation injuries. *Endod Dent Traumatol* 1996;12:113-9.

26. Chan AW, Cheung GS, Ho MW. Different treatment outcomes of two intruded permanent incisors: A case report. *Dent Traumatol* 2001;17:275-80.

27. Faria G, Silva RA, Fiori JM, Nelson FP. Re-eruption of traumatically intruded mature permanent incisor: Case report. *Dent Traumatol* 2004;20:229-32.

