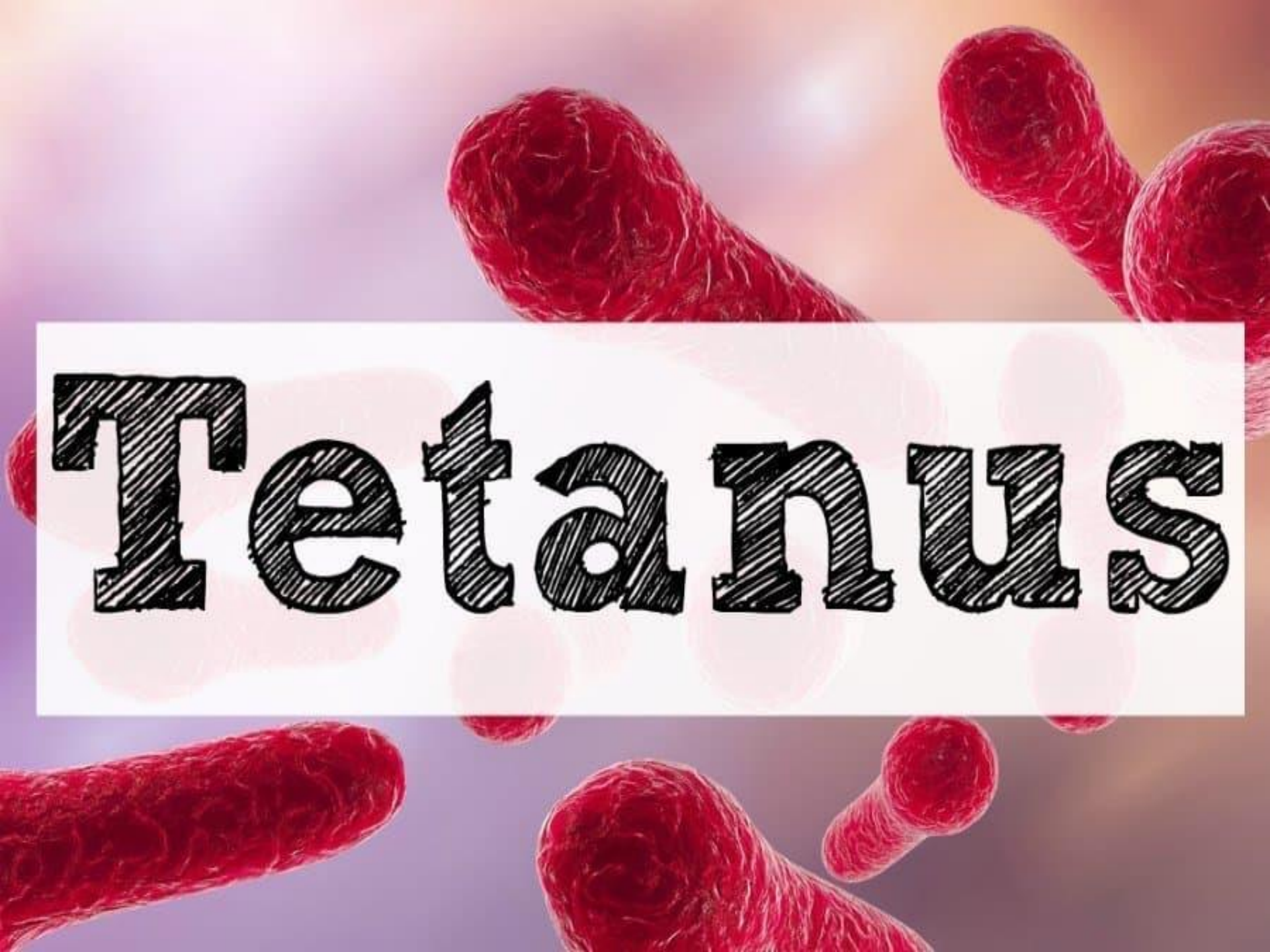

Tetanus

Definition.

Tetanus is an infectious disease caused by *Cl.tetani* exotoxin (tetanospasmin) and characterized by increased muscle tone and spasms. .

Etymology

The word tetanus comes from the Ancient Greek:

***τέτανος tetanos* "taut"**

Tetanus, also known as lockjaw, is an infection characterized by muscle spasms.

In the most common type the spasms begin in the jaw and then progress to the rest of the body.

These spasms usually last a few minutes each time and occur frequently for three to four weeks.

Spasms may be so severe that bone fractures may occur.

Other symptoms may include
fever,
sweating,
headache,
trouble swallowing,
high blood pressure,
and a fast heart rate.
Onset of symptoms is typically three to twenty one days following infection.
It may take months to recover.
About 10% of those infected die.

Tetanus occurs in all parts of the world but is most frequent in hot and wet climates where the soil contains a lot of organic matter.

Description of the disease

by Hippocrates exists from at least as far back as the 5th century BCE.

The cause of the disease was determined in 1884 by Antonio Carle and Giorgio Rattone at theUniversity of Turin with a vaccine being developed in 1924.

Tetanus

In 1884, Arthur Nicolaier isolated the strychnine-like toxin of tetanus from free-living, anaerobic soil bacteria

The etiology of the disease was further elucidated in 1884 by Antonio Carle and Giorgio Rattone, two pathologists of the University of Turin, who demonstrated the transmissibility of tetanus for the first time. They produced tetanus in rabbits by injecting pus from a patient with fatal tetanus into their sciatic nerves.

In 1891, *C. tetani* was isolated from a human victim by Kitasato Shibasaburō, who later showed that the organism could produce disease when injected into animals, and that the toxin could be neutralized by specific antibodies

In 1897, Edmond Nocard showed that tetanus antitoxin induced passive immunity in humans, and could be used for prophylaxis and treatment.

Tetanus toxoid vaccine was developed by P. Descombey in 1924, and was widely used to prevent tetanus induced by battle wounds during World War II.

Etiology

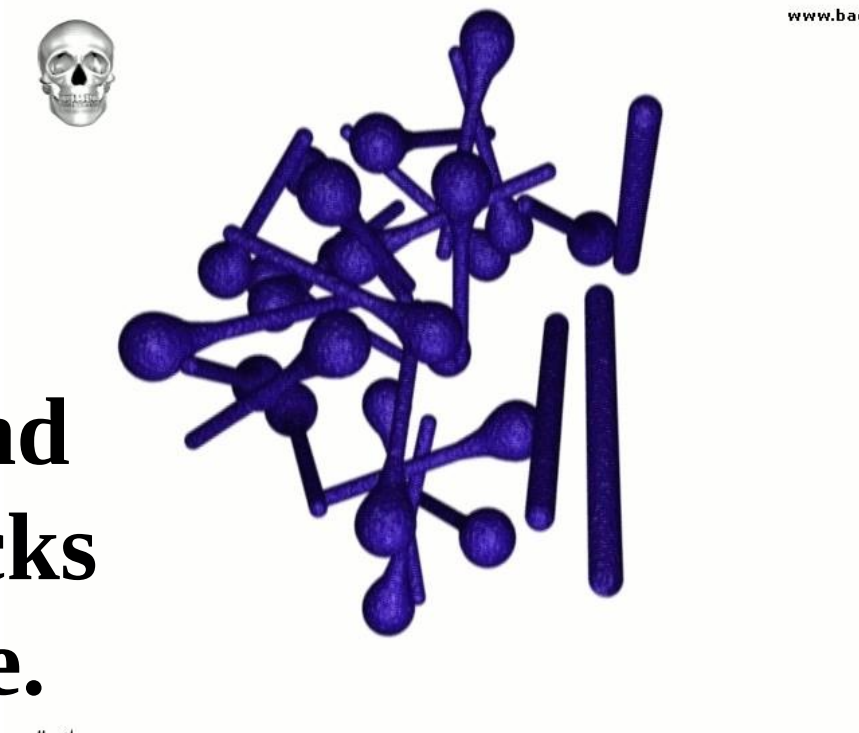
C. tetani **is an anaerobic, motile, gram-positive rod that forms an oval, colorless, terminal spore and thus assumes a shape resembling a tennis racket or drumstick.**

The organism is found worldwide in soil, in the inanimate environment, in animal feces, and occasionally in human feces. Spores may survive for years in some environments and are resistant to various disinfectants and to boiling for 20 min. Vegetative cells, however, are easily inactivated and are susceptible to several antibiotics,
Including metronidazole and penicillin.

Vegetative cells of *Cl.tetani* can produce exotoxin that causes different forms of tetanus in people.

Tetanospasmin is formed in vegetative cells and released at the moment of their autolysis.

It finally consists of a heavy chain which mediates binding to and entry into nerve cells and a light chain which blocks neurotransmitter release.



Tetanospazmin
acts on the nervous system, causes convulsions

The pathogen forms tetanus exotoxin - one of the strongest bacterial poisons, second only to botulinum toxin.

Tetanolysin
a hemolysin, that causes destruction of tissues

- The toxin is destroyed by heating,
- exposure to sunlight, alkaline environment.
- Not absorbed through the intestinal mucosa, and therefore safe if swallowed.

EPIDEMIOLOGY

Tetanus occurs sporadically and almost always affects unimmunized persons; partially immunized persons and fully immunized individuals who fail to maintain adequate immunity with booster doses of vaccine may be affected as well.

Although tetanus is entirely preventable by immunization, the burden of disease worldwide is great.

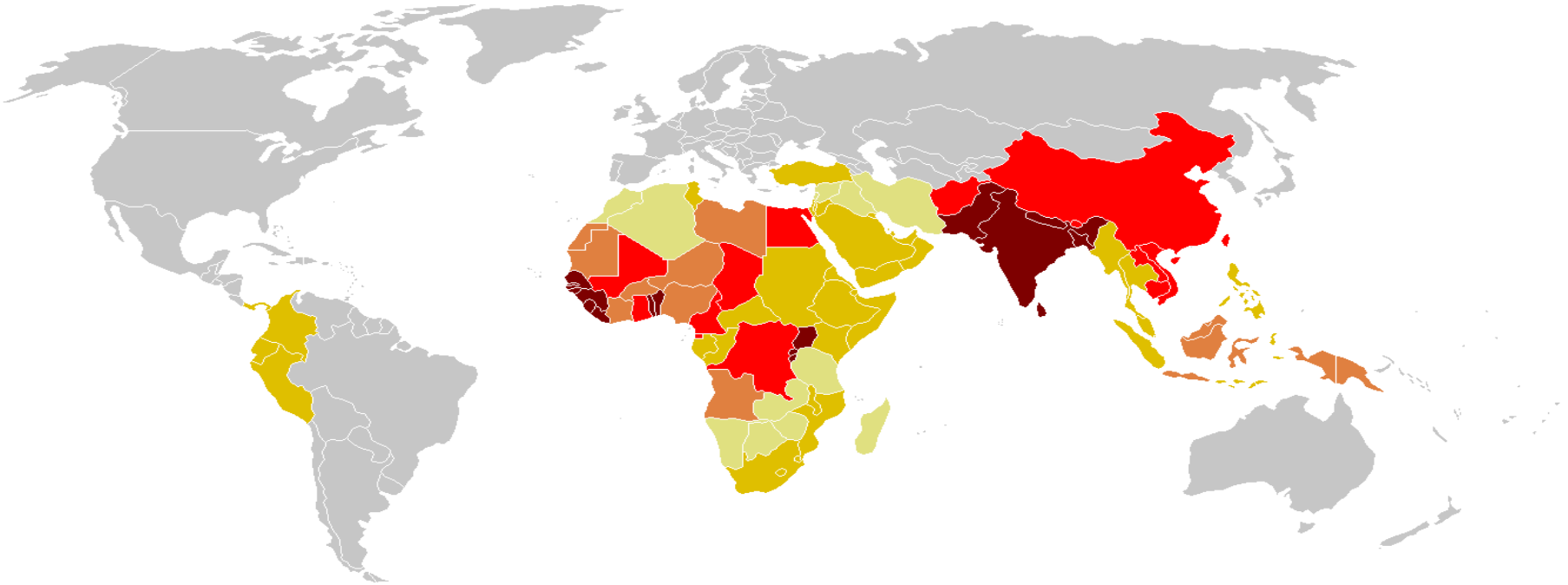
Tetanus is a notifiable disease in many countries, but reporting is known to be inaccurate and incomplete, particularly in developing countries.

As a result, the World Health Organization considers the number of reported cases to be an underestimate and periodically undertakes case/death estimates to assess the burden of disease.

The disease remains an important public health problem in many parts of the world, but especially in low-income countries or districts, where immunization coverage is low, and unclean birth practices are common.

Neonatal tetanus occurs when nonsterile instruments are used to cut the umbilical cord or when contaminated material is used to cover the umbilical stump.

Deliveries carried out by people with unclean hands or on a contaminated surface are also risk factors.



Response

World Health Organization

The global neonatal tetanus elimination goal was launched at the World Health Assembly in 1989 to reduce neonatal tetanus as a public health problem (defined as less than one case of neonatal tetanus per 1000 live births in every district) in all countries.

The Maternal and Neonatal Tetanus Elimination (MNTE) Initiative was launched by UNICEF, WHO and the United Nations Population Fund (UNFPA) in 1999, revitalizing the goal of MNTE as a public health problem.

As of April 2018, there are 14 countries that have not achieved MNTE.

Tetanus is common in areas where soil is cultivated, in rural areas, in warm climates, during summer months, and among males.

In countries without a comprehensive immunization program, tetanus occurs predominantly in neonates and other young children.

It is noteworthy that international programs to eliminate neonatal Tetanus have been in place for some time.

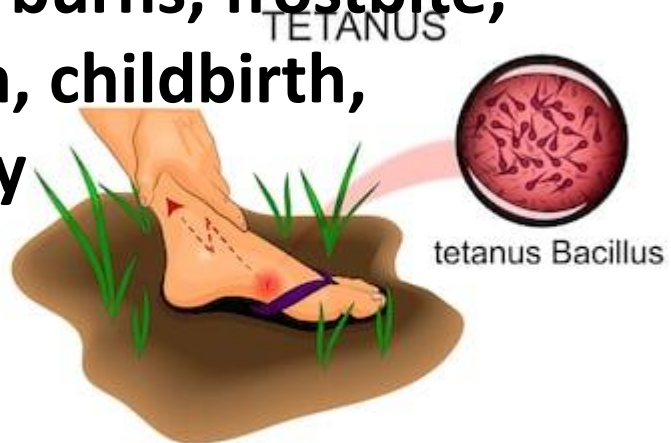
In the United States, most cases of tetanus follow an acute injury (puncture wound, laceration, abrasion, or other trauma).

Tetanus may be acquired indoors or during outdoor activities (e.g., farming, gardening). The implicated injury may be major, but can be so trivial that medical attention is not sought. In some cases, no injury or portal of entry can be identified.

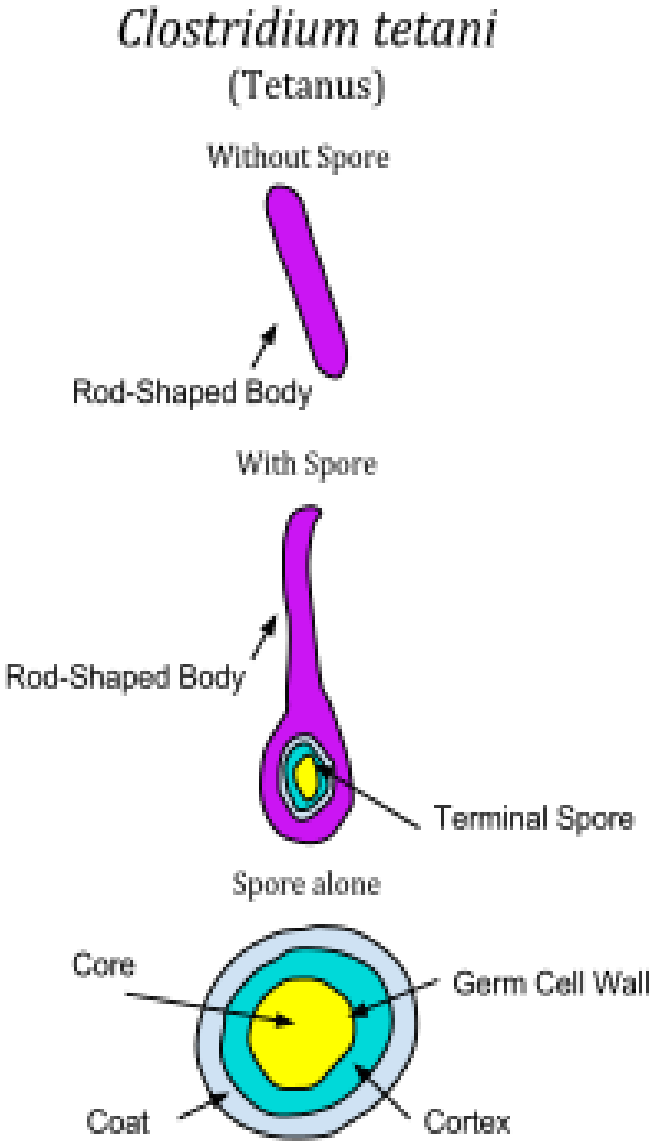
The disease may complicate chronic conditions such as skin ulcers, abscesses, and gangrene.

Tetanus has also been associated with burns, frostbite, middle-ear infection, surgery, abortion, childbirth, body piercing, and drug abuse (notably "skin popping").

Recurrent tetanus has been reported.



Clostridium tetani **is strongly durable due to its endospores. Pictured is the bacterium alone, with a spore being produced, and the spore alone.**

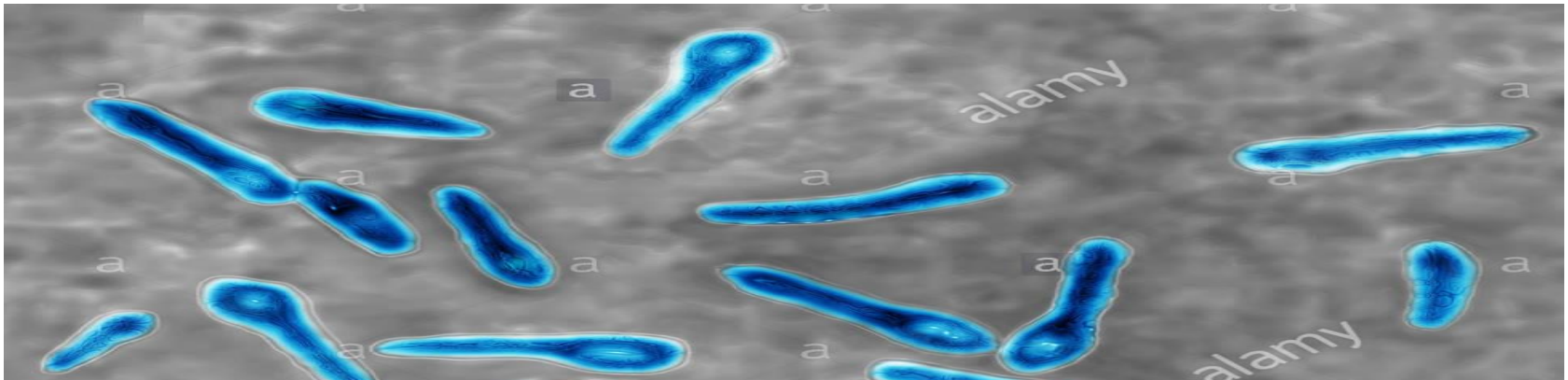


In the presence of oxygen and a temperature not lower than 4 ° C forms spores.

Spores are resistant to external effects: they withstand heating to 90 ° C for 2 hours, die only after 1-3 hours at boiling, transfer to 150 ° C in a dry state, live up to 6 months in salty seawater.

In feces, soil, on various objects are stored for more than 100 years [source not specified 1586 days].

In the absence of oxygen, a temperature of 37 ° C, and sufficient moisture, the spores germinate in the unstable vegetative form.



Tetanus is often associated with rust, especially rusty nails. Objects that accumulate rust are often found outdoors, or in places that harbour anaerobic bacteria, but the rust itself does not cause tetanus nor does it contain more *C. tetani* bacteria. The rough surface of rusty metal merely provides a prime habitat for *C. tetani* endospores to reside in (due to its high surface area), and the nail affords a means to puncture skin and deliver endospores deep within the body at the site of the wound.

Tetanus is an international health problem, as *C. tetani* spores are ubiquitous. The disease occurs almost exclusively in persons unvaccinated or inadequately immunized.

It is more common in hot, damp climates with soil rich in organic matter.

This is particularly true with manure-treated soils, as the spores are widely distributed in the intestines and feces of many animals such as horses, sheep, cattle, dogs, cats, rats, guinea pigs, and chickens.

.

Spores can be introduced into the body through puncture wounds.

In agricultural areas, a significant number of human adults may harbor the organism.

The spores can also be found on skin surfaces and in contaminated heroin.

Heroin users, particularly those that inject the drug subcutaneously, appear to be at high risk of contracting tetanus

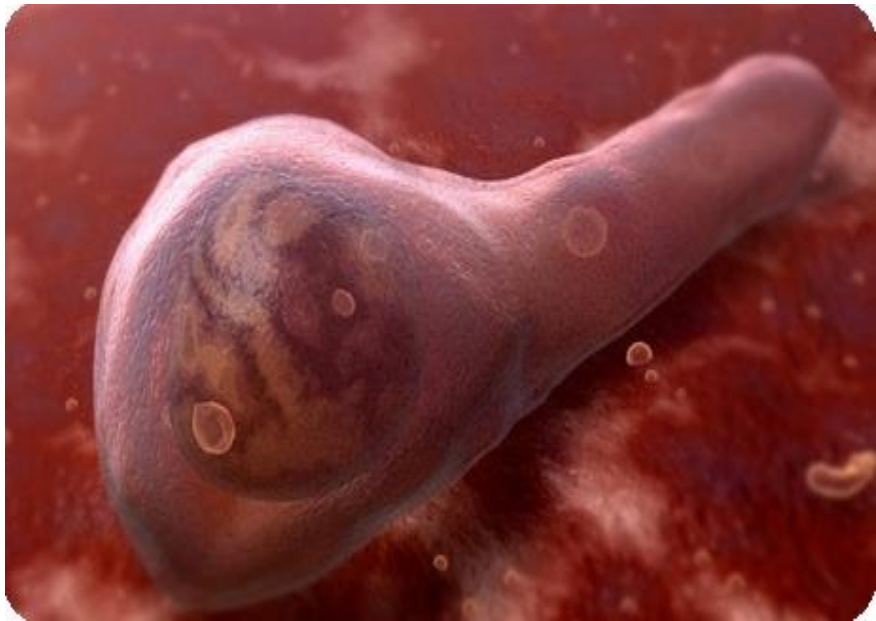
An endospore is a non-metabolizing survival structure that begins to metabolize and cause infection once in an adequate environment.

Because *C. tetani* is an anaerobic bacterium, it and its endospores thrive in environments that lack oxygen. Hence, stepping on a nail (rusty or not) may result in a tetanus infection, as the low-oxygen (anaerobic) environment is caused by the oxidization of the same object that causes a puncture wound, delivering endospores to a suitable environment for growth.

PATHOGENESIS

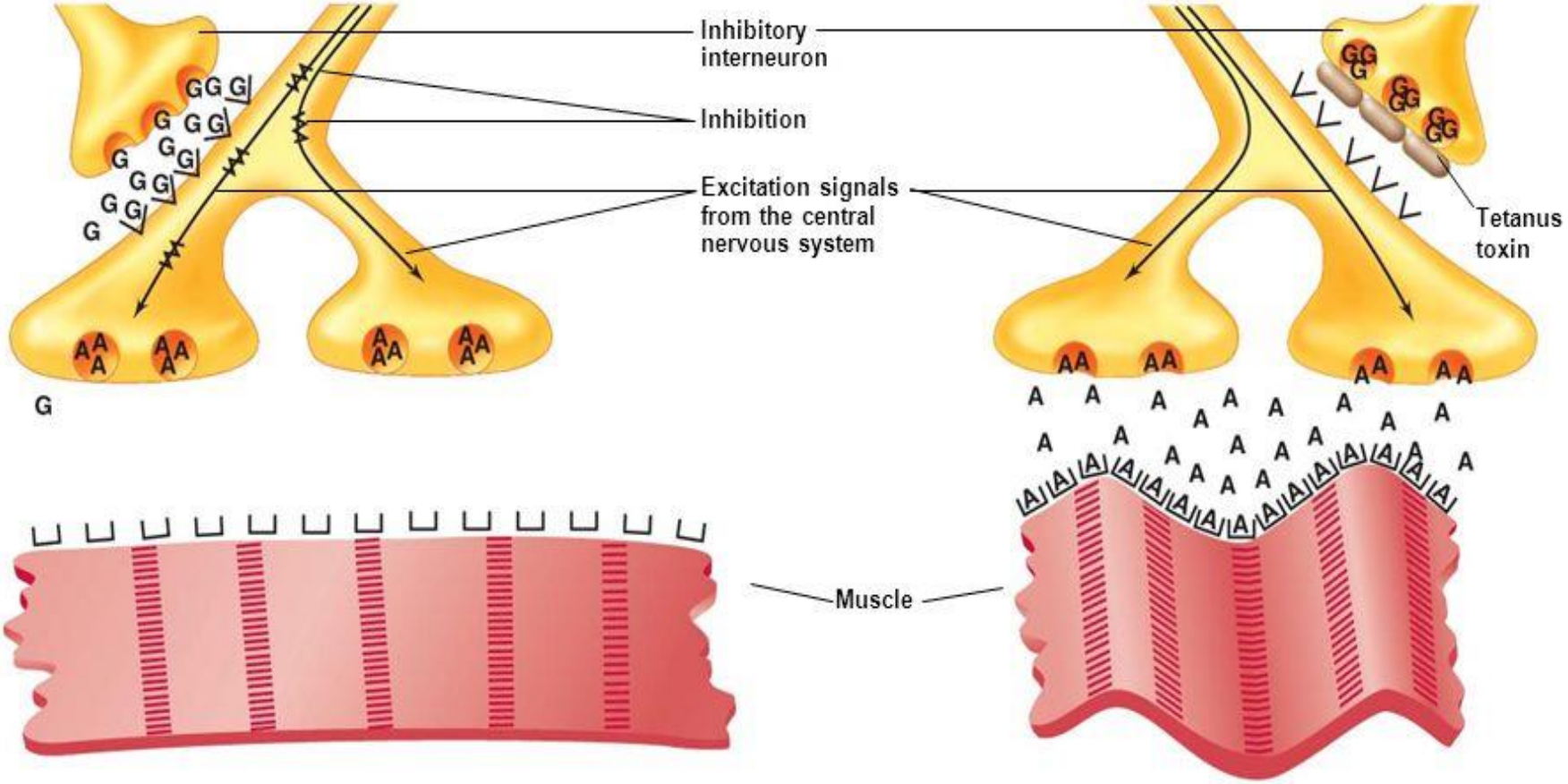
Contamination of wounds with spores of *C. tetani* is probably a frequent occurrence. Germination and toxin production, however, take place only in wounds with low oxidation-reduction potential, such as those with devitalized tissue, foreign bodies, or active infection.

C. tetani does not itself evoke inflammation, and the portal of entry retains a benign appearance unless coinfection with other organisms is present.



Tetanus toxin: blocks glycine release – constant acetylcholine release.

Exotoxins



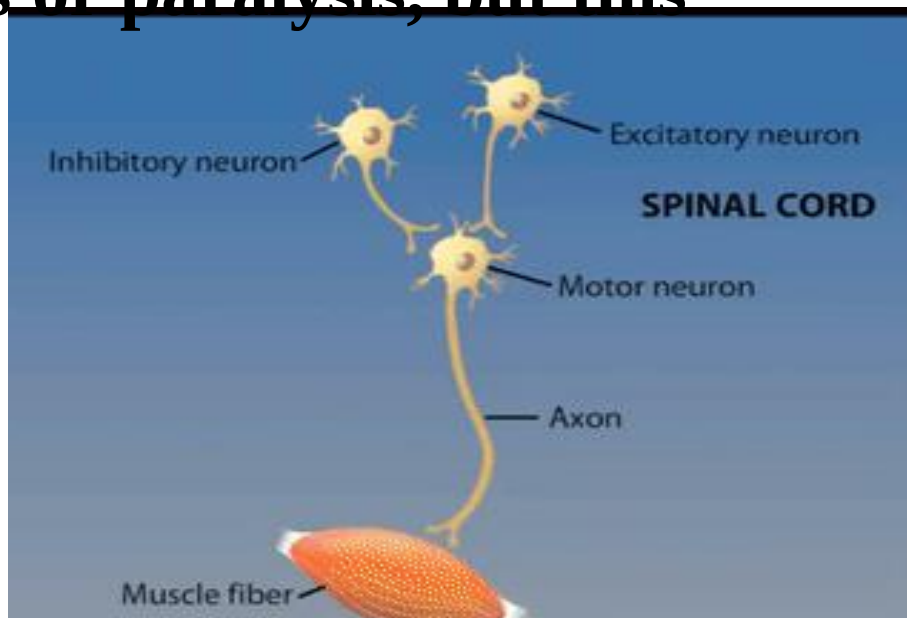
Toxin released in the wound binds to peripheral motor neuron terminals, enters the axon, and is transported to the nerve-cell body in the brainstem and spinal cord by retrograde intraneuronal transport. The toxin then migrates across the synapse to presynaptic terminals, where it blocks release of the inhibitory neurotransmitters
glycine and γ-aminobutyric acid (GABA) from vesicles .
The blocking of neurotransmitter release by tetanospasmin, a zinc metalloprotease, involves cleavage of synaptobrevin, a protein essential to proper function of the synaptic vesicle release apparatus.
With diminished inhibition, the resting firing rate of the α motor neuron
increases, producing rigidity.
With lessened activity of reflexes that limit polysynaptic spread of impulses (a glycinergic activity), agonists and antagonists may be recruited rather than inhibited, with the consequent production of spasms.

Toxin may also affect preganglionic sympathetic neurons in the lateral gray matter of the spinal cord and parasympathetic centers.

Loss of inhibition of preganglionic sympathetic neurons may produce sympathetic hyperactivity and high circulating catecholamine levels.

Tetanospasmin, like botulinum toxin, may block neurotransmitter release at the neuromuscular junction and produce weakness or paralysis, but this effect is clinically evident only in cephalic tetanus.

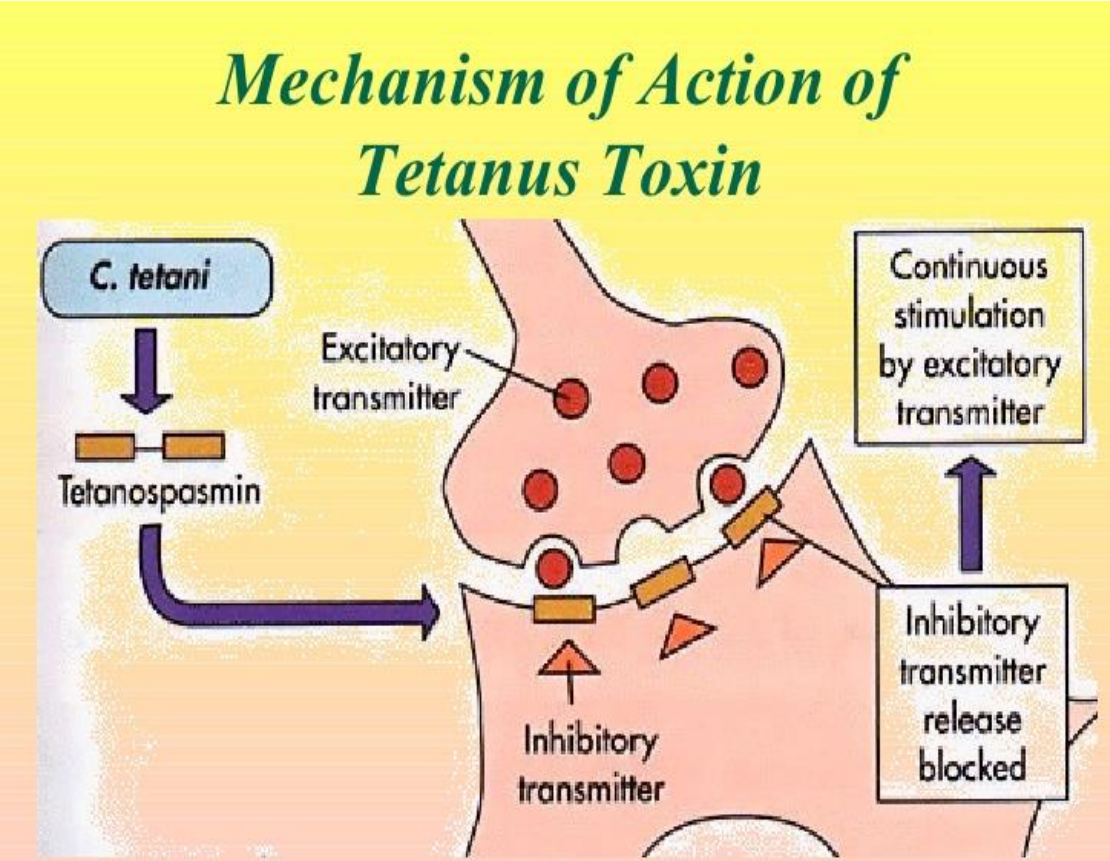
Recovery requires sprouting of new nerve terminals.

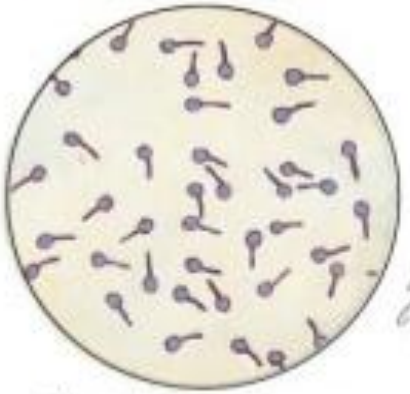


In local tetanus, only the nerves supplying the affected muscles are involved.

Generalized tetanus occurs when toxin released in the wound enters the lymphatics and bloodstream and is spread widely to distant nerve terminals;
the blood-brain barrier blocks direct entry into the central nervous system.

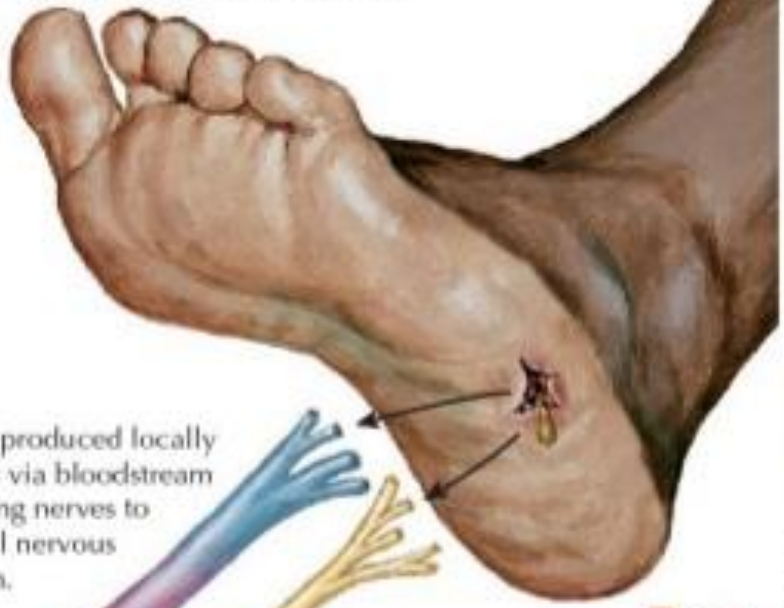
If it is assumed that intraneuronal transport times are equal for all nerves, short nerves are affected before long nerves: this fact explains the sequential involvement of nerves of the head, trunk, and extremities in generalized tetanus.

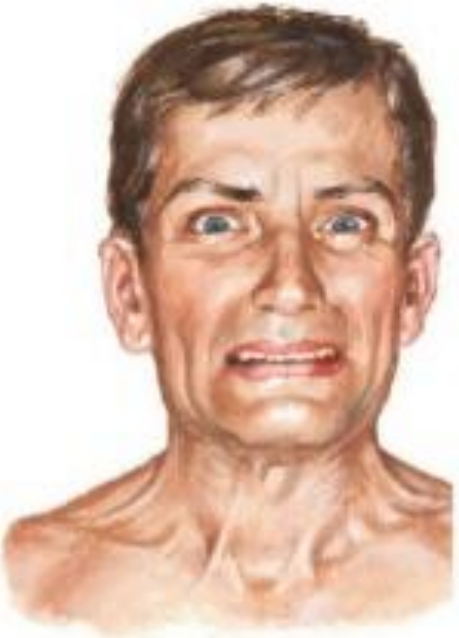




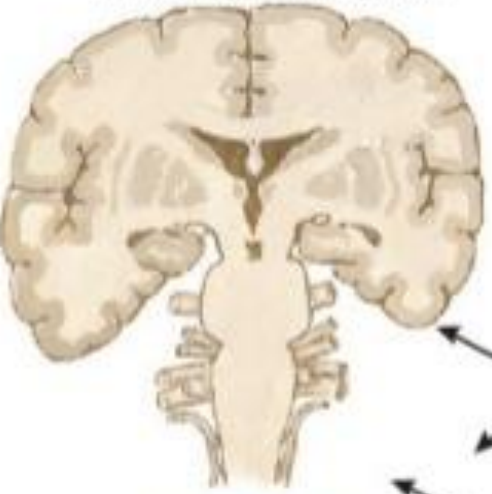
Clostridium tetani: gram-positive, spore-bearing rods

Organisms enter through large, small, or even unrecognized wound. Deep, infected punctures are most susceptible, since organisms thrive best anaerobically.

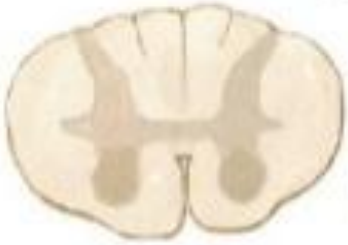




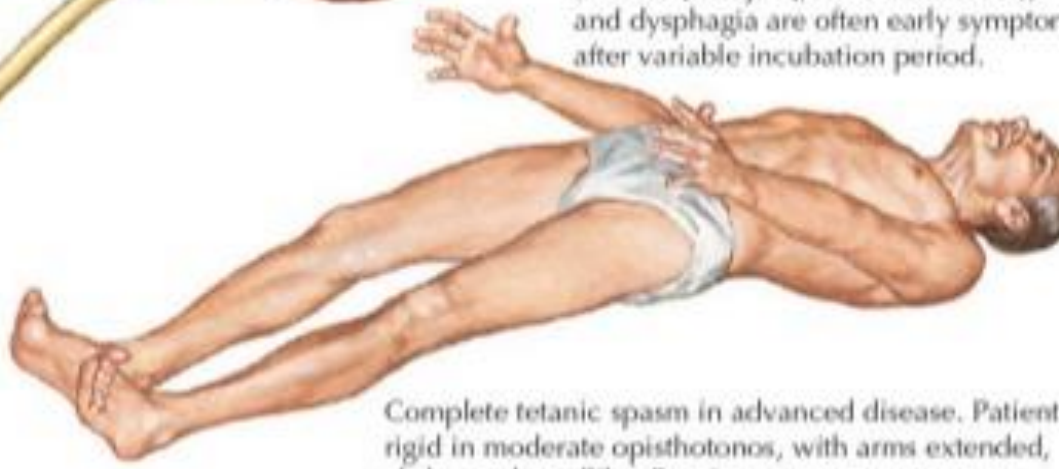
Spasm of jaw, facial, and neck muscle (trismus [lockjaw], risus sardonicus), and dysphagia are often early symptor after variable incubation period.



Toxin produced locally passes via bloodstream or along nerves to central nervous system.



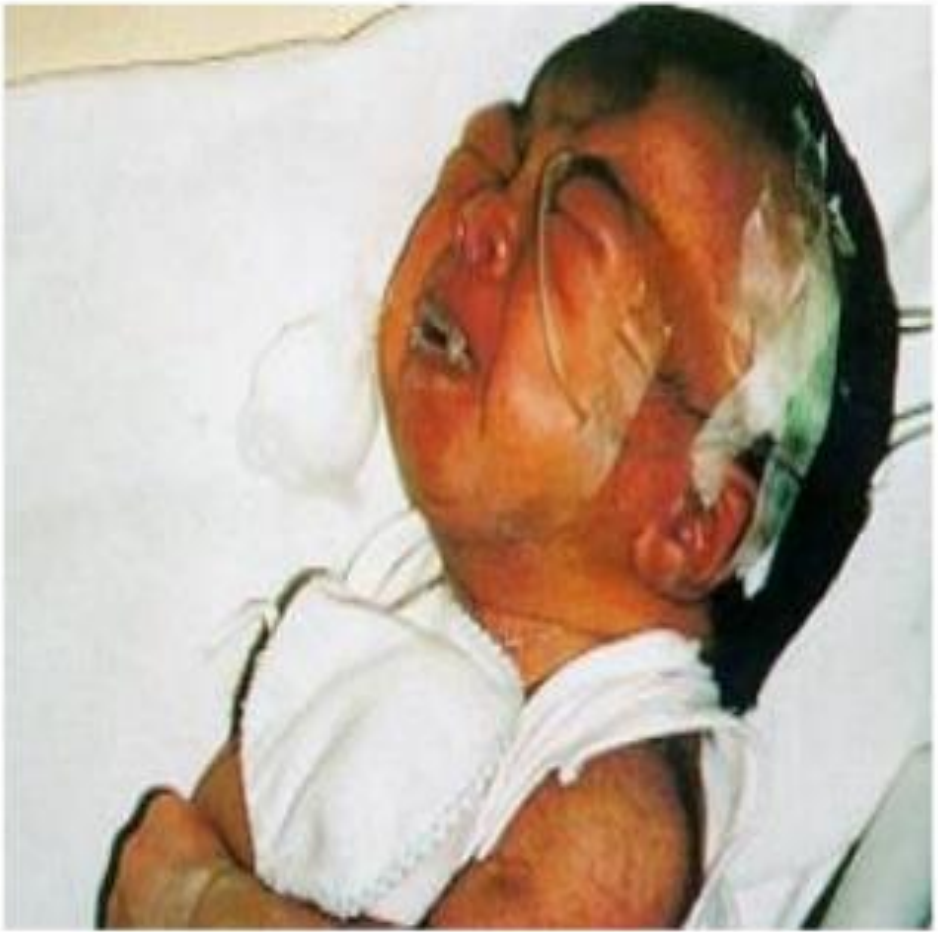
Motor neurons of spinal cord (anterior horn) and brainstem become hyperactive because toxin specifically attacks inhibitory (Renshaw) cells.



Complete tetanic spasm in advanced disease. Patient rigid in moderate opisthotonos, with arms extended, abdomen boardlike. Respiratory arrest may occur.

Type of Tetanus

- Traumatic tetanus
- Puerperal tetanus
- Otogenic tetanus
- Idiopathic tetanus
- Tetanus Neonatroum



Tetanus occurs in several clinical forms, including

generalized

neonatal

localized disease

The severity of illness may be

mild (muscle rigidity and few or no spasms),

moderate (trismus, dysphagia, rigidity, and spasms),

or severe (frequent explosive paroxysms).

The patient may be febrile, although many patients have no fever; mentation is unimpaired.

Deep tendon reflexes may be increased.

Dysphagia or ileus may preclude oral feeding.

Incubation period

The incubation period of tetanus may be up to several months, but is usually about eight days.

In general, the farther the injury site is from the central nervous system, the longer the incubation period.

The shorter the incubation period, the more severe the symptoms.

In neonatal tetanus, symptoms usually appear from 4 to 14 days after birth, averaging about 7 days.

On the basis of clinical findings, four different forms of tetanus have been described.

CLINICAL MANIFESTATIONS

Generalized tetanus,

the most common form of the disease, is characterized by increased muscle tone and generalized spasms.

The median time of onset after injury is 7 days; 15% of cases occur within 3 days and 10% after 14 days.

Typically, the patient first notices increased tone in the masseter muscles (trismus, or lockjaw).

Dysphagia or stiffness or pain in the neck, shoulder, and back muscles

appears concurrently or soon thereafter.

The subsequent involvement of other muscles produces a rigid abdomen and stiff proximal limb muscles; the hands and feet are relatively spared.

Sustained contraction of the facial muscles results in a grimace or sneer (risus sardonicus), and contraction of the back muscles produces an arched back (opisthotonos).

TETANUS

(Lockjaw)

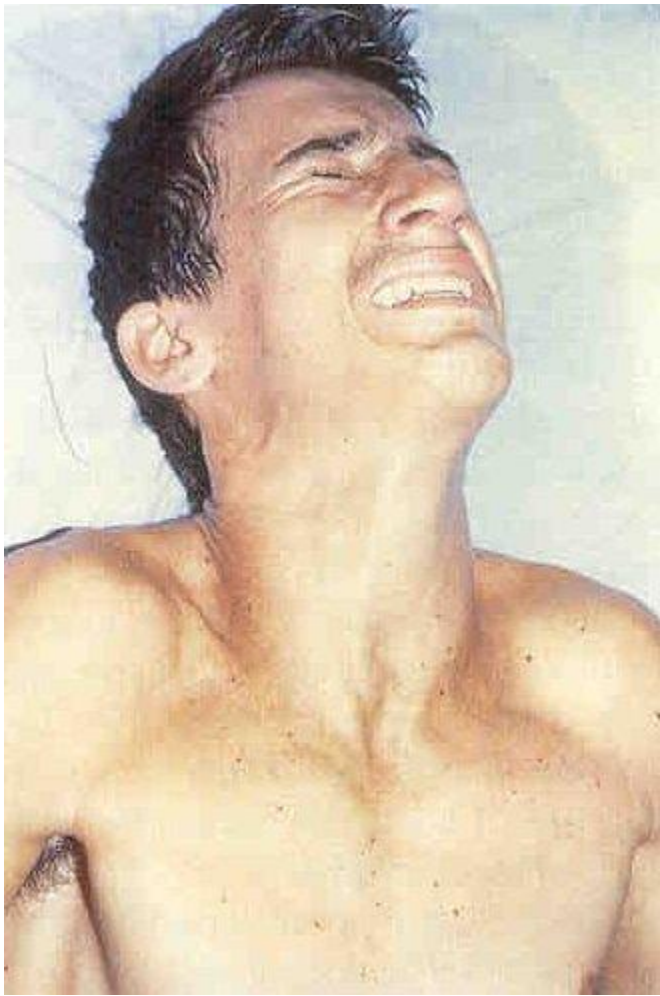
* Intact Sensorium
* Headache
* Difficult Swallowing
* Sore Throat
* Irritability
* Tonic Spasms
* Prevention - Childhood Immunizations

* Spasms of Facial Muscles
  - Fixed Smile
  - Elevated Eyebrows
* Jaw Stiffness
* Fever
* Restlessness
* Chills
* Exaggerated Reflexes
* Profuse Sweating

Some patients develop paroxysmal, violent, painful, generalized muscle spasms that may cause cyanosis and threaten ventilation. These spasms occur repetitively and may be spontaneous or provoked by even the slightest stimulation.

A constant threat during generalized spasms is reduced ventilation or apnea or laryngospasm.

Autonomic dysfunction commonly complicates severe cases and is characterized by labile or sustained hypertension, tachycardia, dysrhythmia, hyperpyrexia, profuse sweating, peripheral vasoconstriction, and increased plasma and urinary catecholamine levels. Periods of bradycardia and hypotension may also be documented.
Sudden cardiac arrest sometimes occurs, but its basis is unknown.
Other complications include aspiration pneumonia, fractures,
muscle rupture, deep-vein thrombophlebitis, pulmonary emboli, decubitus ulcer, and rhabdomyolysis.

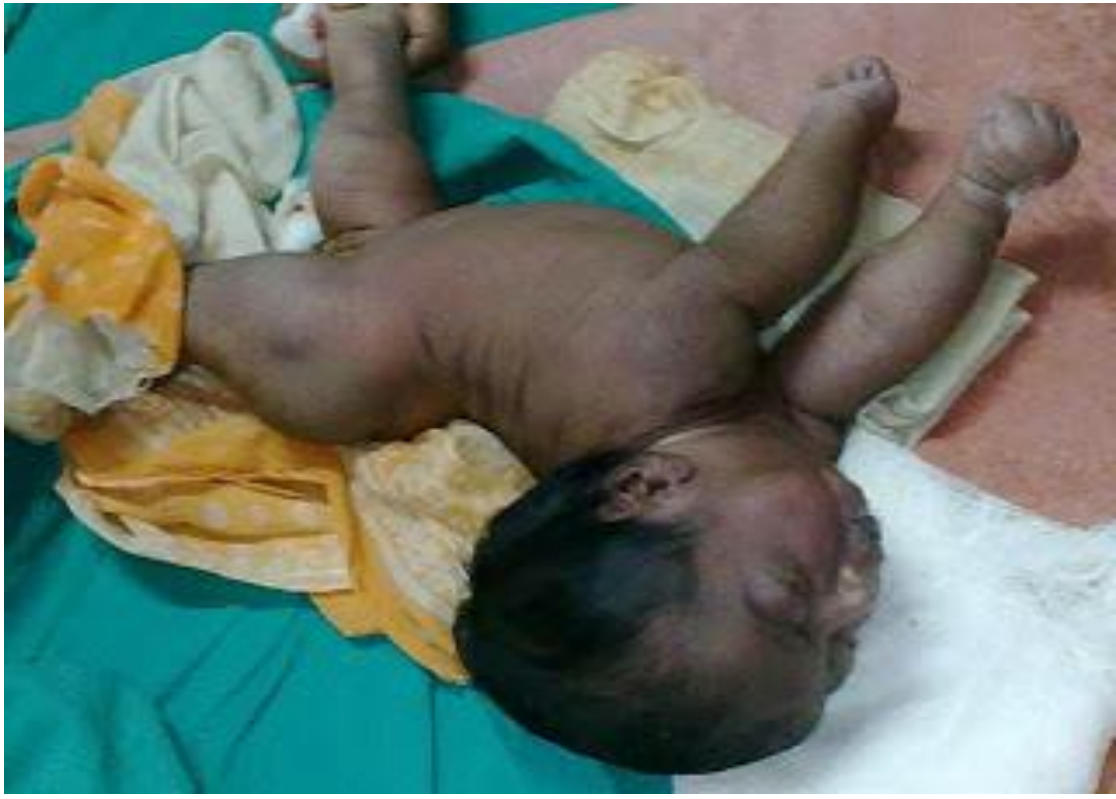


Neonatal tetanus

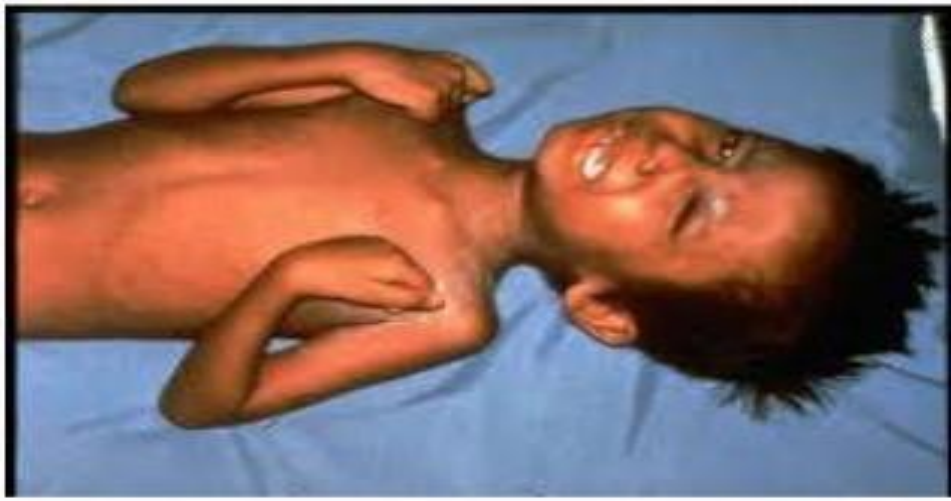
usually occurs as the generalized form and is usually fatal if left untreated.

It develops in children born to inadequately immunized mothers, frequently after unsterile treatment of the umbilical cord stump.

Its onset generally comes during the first 2 weeks of life.



a baby having a tetanus spasm.

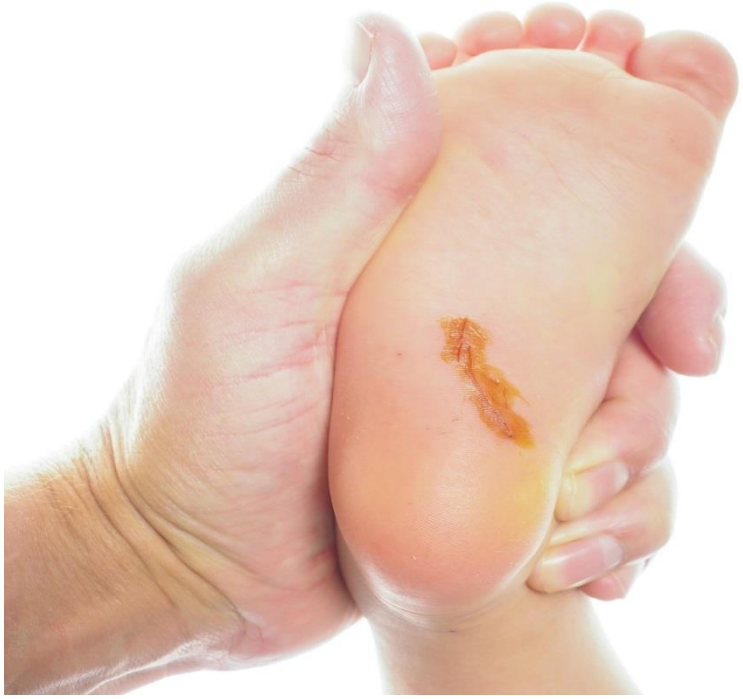


Tetanus neonatorum

Child Tetanus

Local tetanus **is an uncommon form in which manifestations are restricted to muscles near the wound.**

The prognosis is excellent.



Cephalic Tetanus

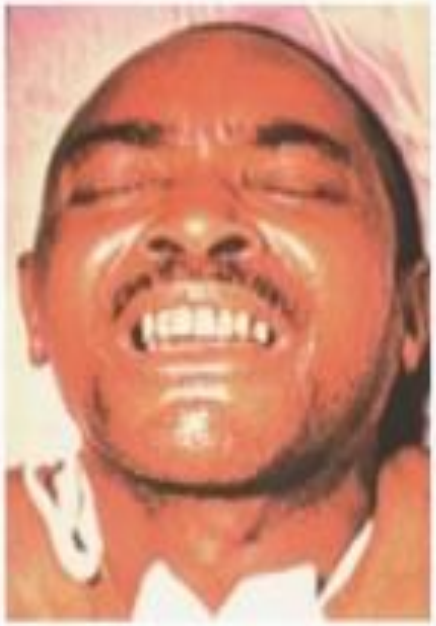
Cephalic tetanus is ***a rare form*** of the disease, occasionally ***occurring with otitis media(ear infections)*** in which ***C. tetani*** is present in the flora of the middle ear , ***or following injuries to the head*** .

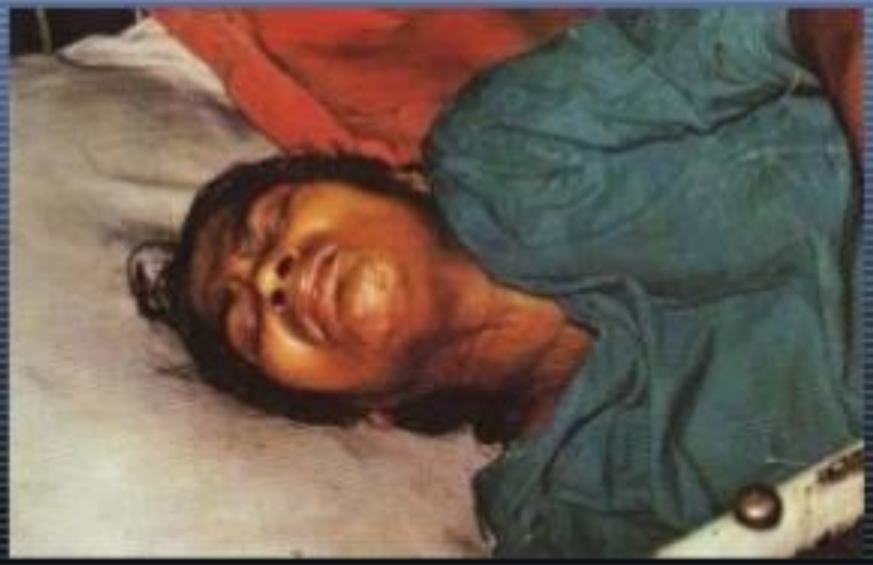
There is ***involvement of the cranial nerves***, especially in the facial area.

incubation period
is a few days and mortality is high.

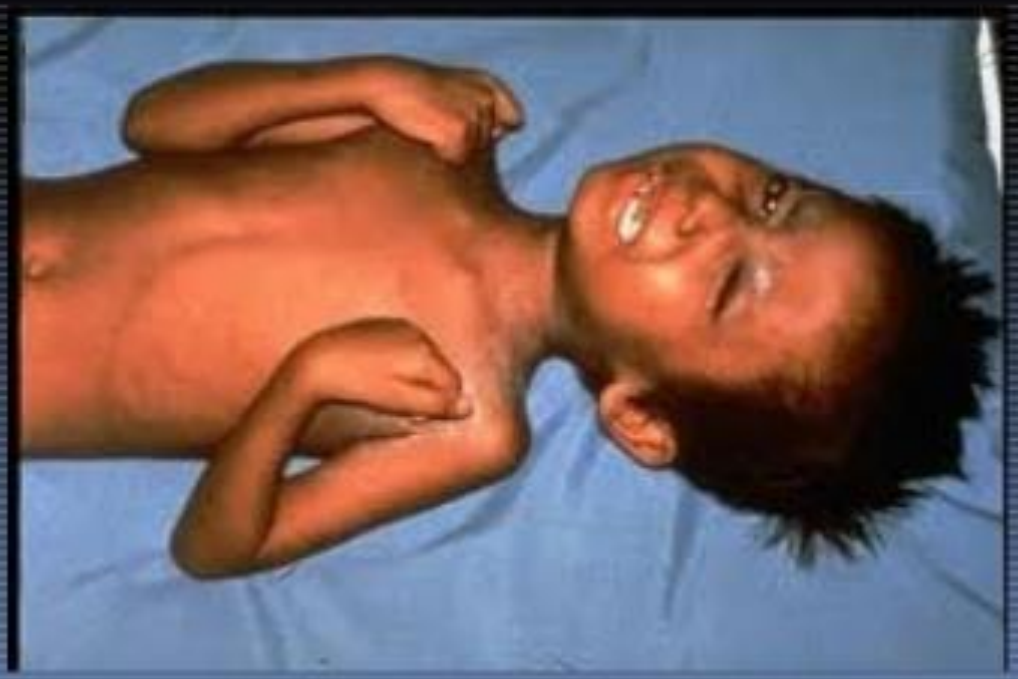
Signs

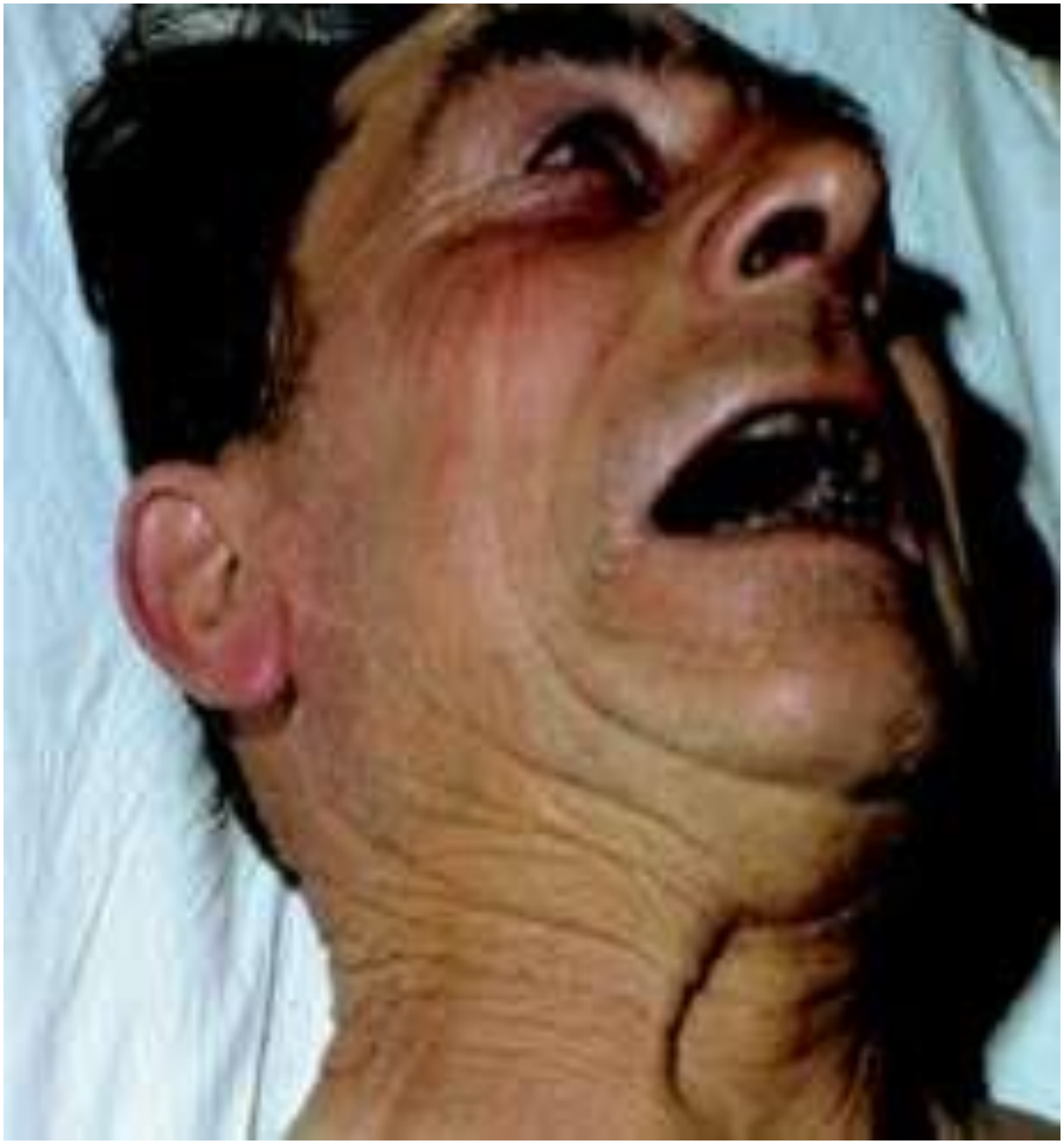
- **Risus Sardonicus :** Spasm of facial muscles (frontalis & angle of mouth muscles) producing grinning facies



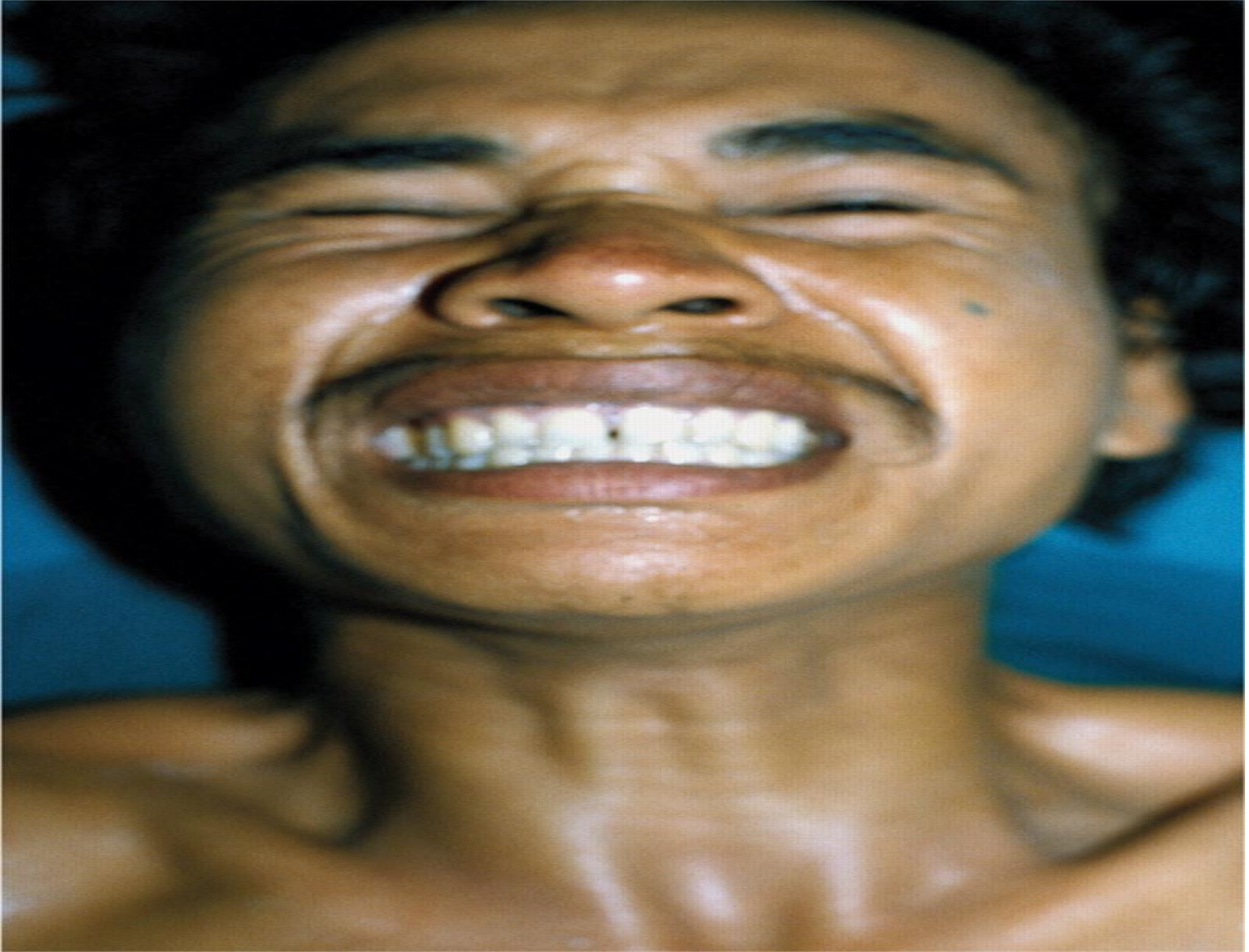


Trismus of masseter muscles and risus sardonicus, contraction of muscles





trismus



The diagnosis of tetanus is based entirely on clinical findings.

Tetanus is unlikely if a reliable history indicates the completion of a primary vaccination series and the receipt of appropriate booster doses.

Wounds should be cultured in suspected cases.

However, *C. tetani* can be isolated from wounds of patients without tetanus and frequently cannot be recovered from wounds of those with tetanus.

The leukocyte count may be elevated.

Cerebrospinal fluid examination yields normal results.

Electromyograms may show continuous discharge of motor units and shortening or absence of the silent interval normally seen after an action potential.

Nonspecific changes may be evident on the electrocardiogram.

Muscle enzyme levels may be raised.

Serum antitoxin levels of ≥0.1 IU/mL (as measured by enzyme-linked immunosorbent assay) are considered protective and make tetanus unlikely, although cases in patients with protective antitoxin levels have been reported.

Laboratory Diagnosis

- There are no laboratory findings characteristic of tetanus.
- The diagnosis is entirely clinical and does not depend upon bacteriologic confirmation.
- C. tetani is recovered from the wound in only 30% of cases and can be isolated from patients who do not have tetanus.
- Laboratory identification of the organism depends most importantly on the demonstration of toxin production in mice.

Tetanus. Laboratory diagnosis

A biological test is employed for detecting the exotoxin in the test material extract. Two white mice are given intramuscular injections of 0.5-1.0 ml of filtrate of the extract. An equal amount of the filtrate is mixed with antitoxic serum, left to stand for 40 minutes at room temperature, and then injected into another two mice in a dose of 0.75 or 1.5 ml per mouse. If the toxin is present in the filtrate, the first two mice will die in 2-4 days while the other two (control mice) will survive.

differential diagnosis

trismus, *alveolar abscess,
*strychnine poisoning,
dystonic drug reactions (e.g., phenothiazines and metoclopramide),
*hypocalcemic tetany.

meningitis/encephalitis, rabies,
acute abdomen (because of the rigid abdomen).

Markedly increased tone in central
muscles (face, neck, chest, back, and abdomen), with superimposed generalized spasms and relative sparing of the hands and feet, strongly suggests tetanus

Differential Diagnosis:

1- Tetany

2- Encephalitis

3- Strychnine Poisoning

4- Rabies

5- Bacterial meningitis

6- Birth Trauma

7- Epilepsy

DIFFERENTIAL DIAGNOSIS (OF LOCKJAW)

- PARAPHARYNGEAL, RETROPHARYNGEAL, OR DENTAL ***ABSCESSES*** RESULTING IN TRISMUS
- ***RABIES*** MAY PRESENT AS TRISMUS WITH SEIZURES
- ***STRYCHNINE POISONING*** MAY RESULT IN TONIC MUSCLE SPASMS
- ***HYPOCALCEMIA*** MAY PRODUCE TETANY, CHARACT. BY LARYNGEAL AND CARPOPEDAL SPASMS
- OCCASIONALLY, EPILEPTIC SEIZURES, NARCOTIC WITHDRAWAL, OR OTHER DRUG REACTIONS MAY SUGGEST TETANUS

Treatment

As soon as clinical tetanus is suspected, steps to neutralize existing toxin and prevent the formation of new toxin must begin.

- Antitoxin (*tetanus immune globulin*) should be administered immediately. This will inactivate toxins in the blood.
- Wounds should be debrided to remove dead tissue or foreign bodies.
- Antibiotics should be given to inhibit growth of *C. tetani*.
- A tetanus toxoid booster immunization should be given to patients who have not received one within the last 5 years.
- If spasms occur, antispasmodic drugs should be used and respiration maintained by a breathing apparatus if necessary.

GENERAL MEASURES

The goals of therapy are to eliminate the source of toxin, neutralize unbound toxin, and prevent muscle spasms while monitoring the patient's condition and providing support—especially respiratory support—until recovery.

Patients should be admitted to a quiet room in an intensive care unit, where observation and cardiopulmonary monitoring can be maintained continuously, but stimulation can be minimized.

Protection of the airway is vital. Wounds should be explored, carefully cleansed, and thoroughly debrided.

Treatment:

- **Antitoxin** (Drug of choice) should be given at once in order to neutralize toxin, human or horse antitoxin may be given (human antitoxin 3,000-10,000 unit I.V, or horse antitoxin 100,000 unit 1/2 I.M and 1/2 I.V.).
- **Penicillin** or metronidazole.
- Removal of necrotic debris from wound.


10-1500 units
10-1500 units
TETANUS ANTITOXIN
Equine Origin
THE PEAK OF QUALITY
Colorado Serum Company
4950 York Street
Denver, Colorado 80216
TETANUS ANTITOXIN
Equine Origin
FOR VETERINARY USE ONLY
U.S. Vet. License No. 188
COLORADO SERUM COMPANY

- ***SPECIFIC TREATMENT***
- ***HUMAN TETANUS IMMUNOGLOBULIN(TIG)*** IS GIVEN AS SOON AS POSSIBLE TO NEUTRALIZE TOXIN BEFORE IT CAN BIND AT DISTANT MUSCLE GROUPS

- A SINGLE INTRAMUSCULAR INJECTION OF 500 U OF TIG IS SUFFICIENT , BUT TOTAL DOSES AS HIGH AS 3,000–6,000 U ARE ALSO RECOMMENDED

- ***ANTIBIOTIC–*** PENICILLIN G (100,000 U/KG/24 HR DIVIDED Q 4–6 HR IV FOR 10–14 DAYS) REMAINS THE ANTIBIOTIC OF CHOICE

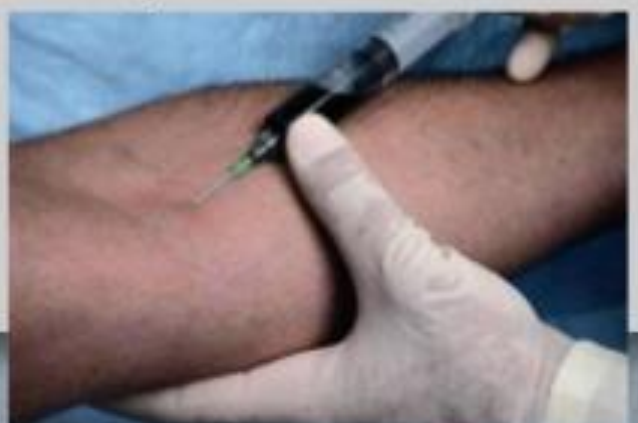
- METRONIDAZOLE ,ERYTHROMYCIN AND TETRACYCLINE ARE

Treatment

- Antibiotics (for example, *metronidazole[Flagyl, Flagyl 375, Flagyl ER], penicillin G or doxycycline [Adoxa, Alodox, Avidoxy, Doryx, Monodox, Oracea, Oraxyl, Periostat, Vibramycin, Vibramycin Calcium, Vibramycin Monohydrate, Vibra-Tabs*]) to kill the bacteria, tetanus booster shot, if necessary, and occasionally, antitoxin (termed tetanus immune globulin or TIG) to neutralize the toxin

Treatment

- If tetanus develops antitoxin injections can be administered which contains antibodies to kill the original invading toxin.
- Muscle relaxants such as *Diazepam* to reduce spasms
- In severe cases a tracheotomy can occur: artifically opening the trachea to ensure the patient can breath (as mouth doesn't function correctly).



CONTROL OF MUSCLE SPASMS

Many agents, alone and in combination, have been used to treat the muscle spasms of tetanus, which are painful and can threaten ventilation by causing laryngospasm
or sustained contraction of ventilatory muscles.
In some developing countries, cost, availability, and the ability to provide ventilatory support are important factors in the choice of therapy. The ideal therapeutic regimen would abolish spasmodic activity without causing oversedation and hypoventilation.
Diazepam, a benzodiazepine and GABA agonist, is in wide use. The dose is titrated, and large doses (≥250 mg/d) may be required. Lorazepam, with a longer duration of action, and midazolam, with a short half-life, are other options.
Barbiturates and chlorpromazine are considered secondline agents.

RESPIRATORY CARE

Intubation or tracheostomy, with or without mechanical ventilation,may be required for hypoventilation due to oversedation or laryngospasm or for the avoidance of aspiration by patients with trismus, disordered swallowing, or dysphagia. The need for these procedures should be anticipated, and they should be undertaken electively and early.

ADDITIONAL MEASURES

Like all patients receiving ventilatory support, patients with tetanus require attention to:

- **hydration;**
- **nutrition;**
- **physiotherapy;**
- **prophylactic anticoagulation;**
- **bowel, bladder, and renal function;**
- **decubitus ulcer prevention;**

and

- **treatment of intercurrent infection.**

Prevention

- Tetanus is completely preventable by active tetanus immunization (vaccine).
- Tetanus immunizations are begun in infancy as a series of DPT shots (D = diptheria, P = pertussis or whooping cough, and T = tetanus).
- Boosters are given to teenagers and older adults as Td shots (adult tetanus and diphtheria) or singly as just tetanus.
- Immunization is considered to provide protection for ten years.
- Studies in the army suggest that good protection persists up to 12 years after the last immunization.

All partially immunized and unimmunized adults should receive vaccine, as should those recovering from tetanus.

The primary series for adults consists of three doses: the first and second doses are given 4–8 weeks apart, and the third dose is given 6–12 months after the second.

A booster dose is required every 10 years and may be given at mid-decade ages—35, 45, and so on. Combined tetanus and diphtheria toxoid, adsorbed (Td, for adult use)—rather than single-antigen tetanus toxoid—is preferred for persons >7 years of age. Adsorbed vaccine

is preferred because it produces more persistent antibody titers than fluid vaccine. Two combined tetanus/ diphtheria/attenuated pertussis vaccines have recently been approved: one (ADACEL) for adults 19–64 years of age and the other (BOOSTRIX) for adolescents 11–18 years of age.

The Advisory Committee on Immunization

Practices has recommended a single dose of Tdap (ADACEL) for adults 19–64 years old who have not received Tdap.

People of all ages need
TETANUS VACCINES

DTaP
for young children

- ✓ 2, 4, and 6 months
- ✓ 15 through 18 months
- ✓ 4 through 6 years

Tdap
for preteens

- ✓ 11 through 12 years

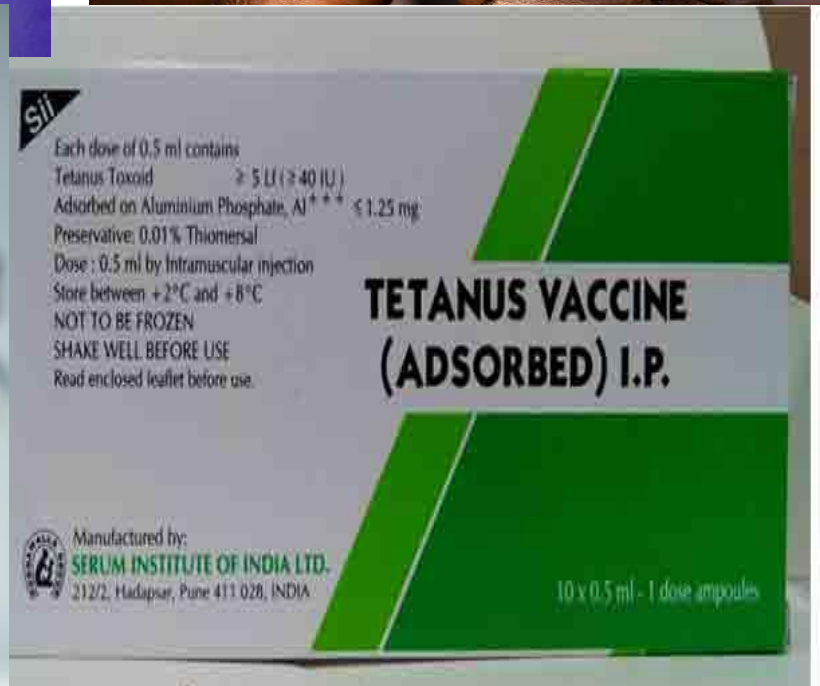
Td
for adults

- ✓ Every 10 years


10 vials x 10,0 ml
TETATOX
TT VACCINE
Tetanus vaccine (adsorbed)
Susp. Inj., vials 10,0 ml - 20 doses IM
Manufactured for InterVax Ltd. - Toronto, Canada
by BB - NCIPD Ltd., Sofia, Bulgaria


ADSORBED
TETANUS
VACCINE BP
10 ml


Tetanus
Vaccine
10 ml


Sii
Each dose of 0.5 ml contains
Tetanus Toxoid ≥ 5 Lf (≥ 40 IU)
Adsorbed on Aluminium Phosphate, Al+++ ≤ 1.25 mg
Preservative: 0.01% Thiomersal
Dose : 0.5 ml by Intramuscular injection
Store between +2°C and +8°C
NOT TO BE FROZEN
SHAKE WELL BEFORE USE
Read enclosed leaflet before use.
TETANUS VACCINE
(ADSORBED) I.P.
Manufactured by:
SERUM INSTITUTE OF INDIA LTD.
212/2, Hadapsar, Pune 411 028, INDIA
10 x 0.5 ml - 1 dose ampoules

PROGNOSIS

The application of methods to monitor and support oxygenation
has markedly improved the prognosis in tetanus.
Mortality rates as low as 10% have been reported from units accustomed to handling such cases.
The outcome is poor in neonates and the elderly and in patients with a short incubation period, a short interval from the onset of symptoms to admission, or a short period from the onset of symptoms to the first spasm (period of onset).
Outcome is also related to the extent of prior vaccination.
The course of tetanus extends over 4–6 weeks, and patients may require prolonged ventilator support.
Increased tone and minor spasms can last for months, but recovery is usually complete.