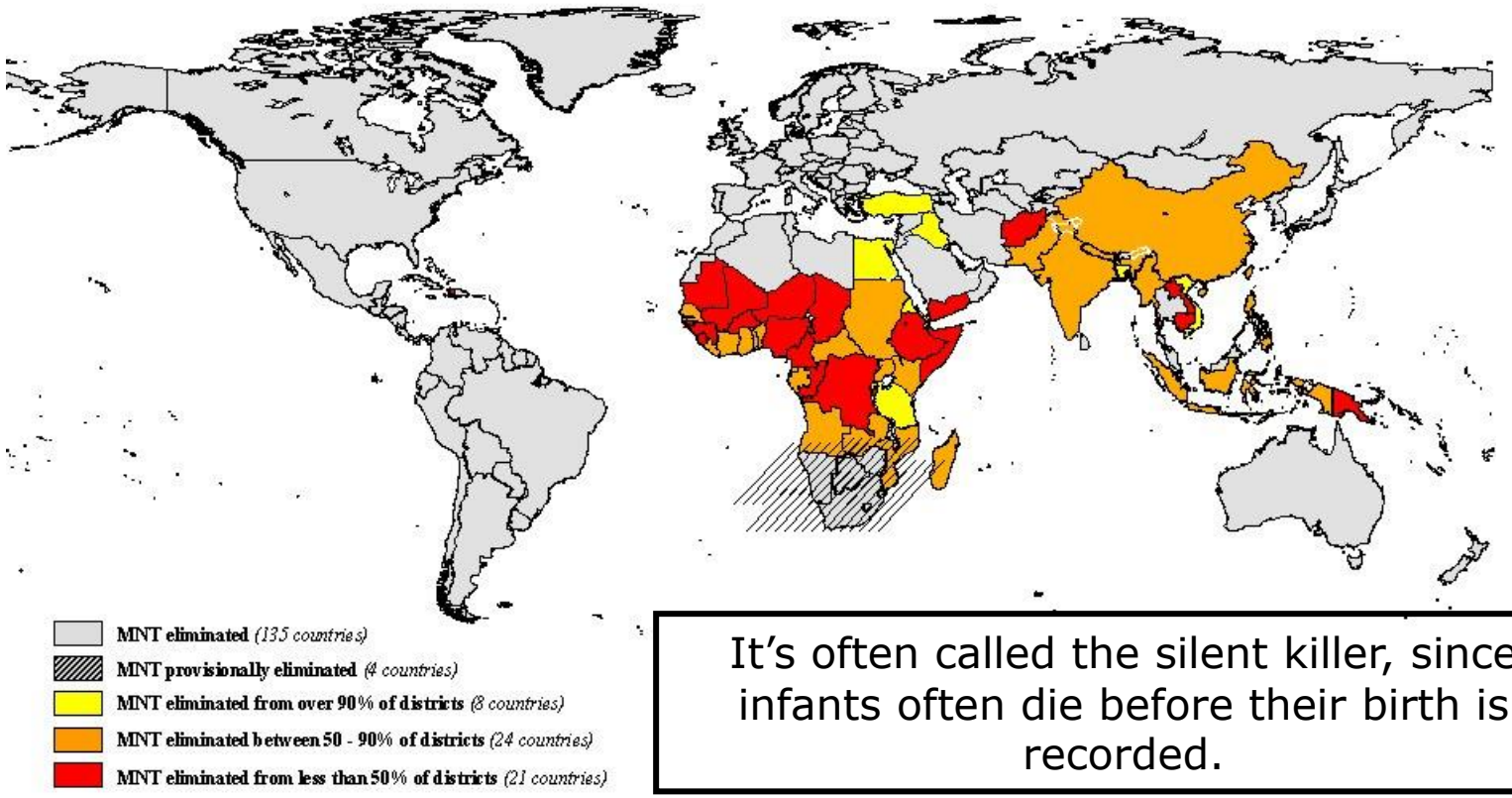
Tetanus

Brief history of disease

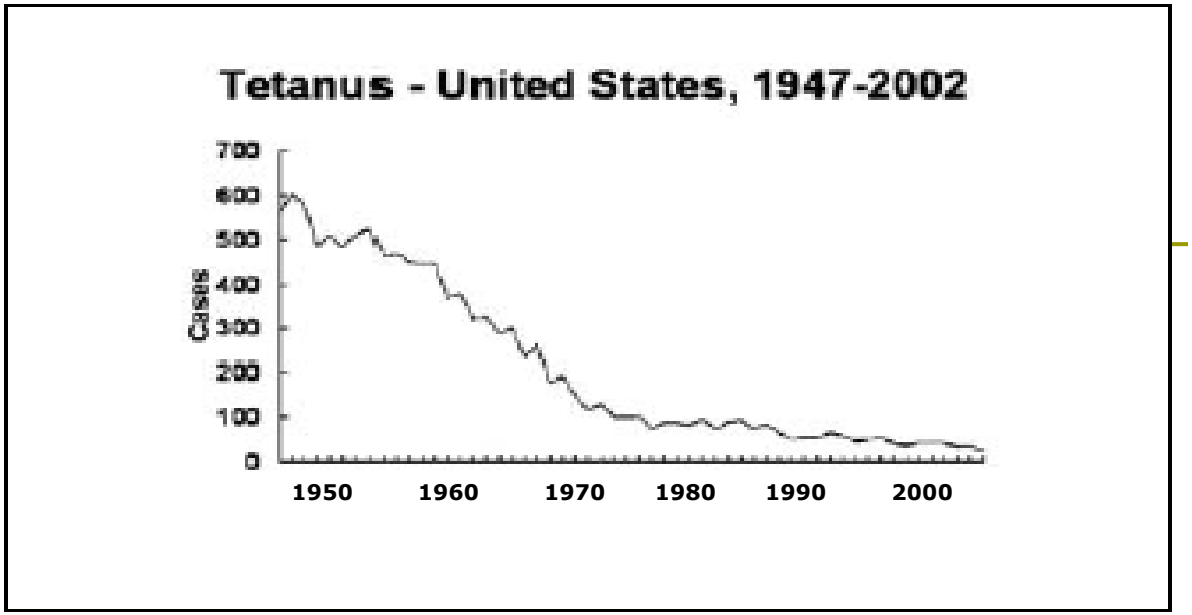
- 5th century BC: Hippocrates first **described** the disease
- 1884: Carle and Rattone discovered the **etiology** *(cause/origin of disease)*
  - Produced tetanus by injecting pus from a fatal human case
  - Nicolaier was able to do the same by injecting soil samples into animals
- 1889: Kitasato **isolated** the organism from human victim, showed that it could produce disease when injected into animals. Reported that **toxin** could be **neutralized** by specific antibodies.
- 1897: Nocard demonstrated the protective effect of **passively transferred antitoxin** → used in WWI
- 1924: Descombey developed tetanus toxoid for **active immunization** → used in WWII

Distribution

In developing countries, neonatal tetanus is a leading cause of neonatal mortality, accounting for over 250,000 deaths annually.



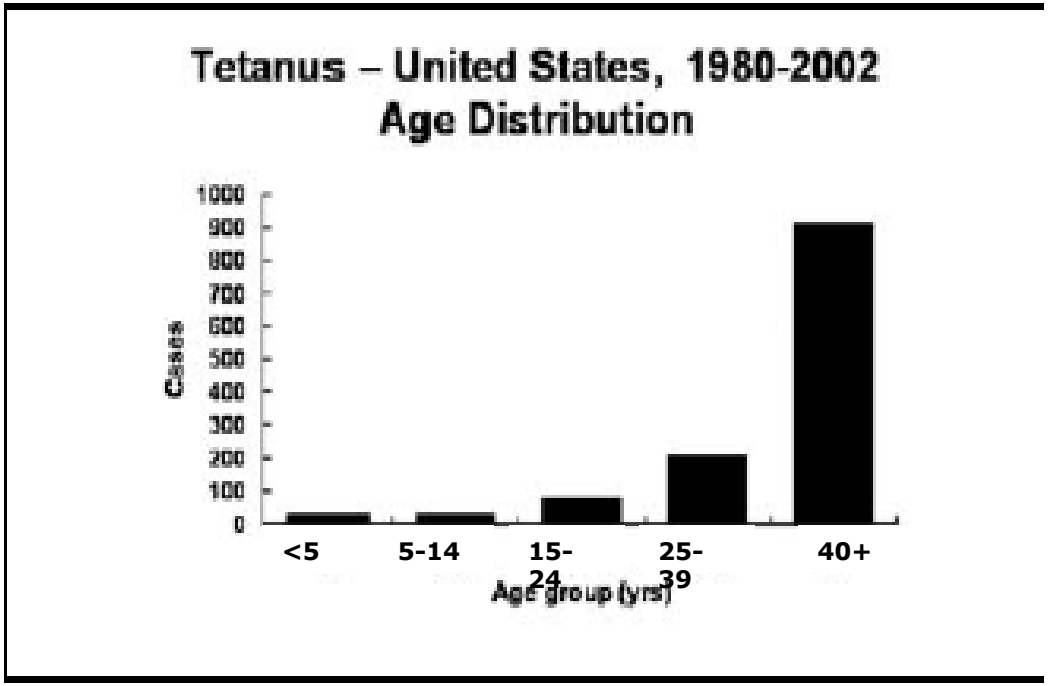
It's often called the silent killer, since infants often die before their birth is recorded.



A sharp decrease after tetanus toxoid was introduced into routine childhood immunization in the late 1940s.

All time low in 2002 – 25 cases (0.4 cases in 100,000 population)

* Affects those over the age of 40 the most → is taken to mean that waning immunity is a significant risk factor.



India: Burden of disease

- Endemic (high risk, low risk)
- Immunization coverage
- Domestic deliveries by untrained personnel
- Birth customs

- EAG states, medically under-served, livestock raising regions…

- High risk areas: preventive measures
- Low risk areas: surveillance

- Districts classified:
  - NT incidence
  - Immunization coverage level in preg.
  - Clean delivery by trained person

NT high risk= inc > 1/1000 LB
or TT_2<70%
or del by TBA <50%

NT control = inc < 1/1000 LB
& TT_2>70%
& del by TBA >50%

NT elimination= inc<0.1/1000LB
& TT_2>90%
& del by TBA >75%

Hospital records: male preponderance

so, while classifying districts, twice the no. of reported male cases considered…

Seasonal trend: >50% of annual cases in months of July – September

Reporting by all hospitals and treatment facilities: Mandatory

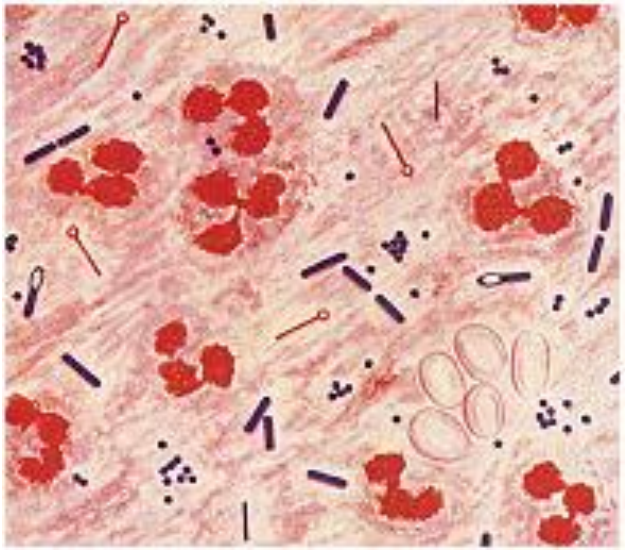
Even nil reporting

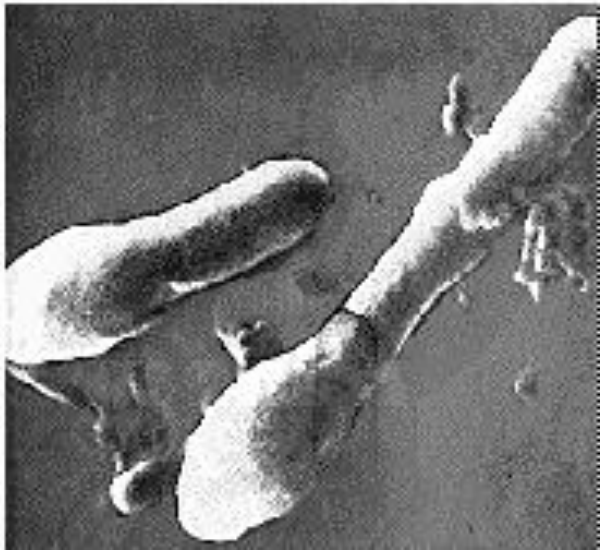
To CDHO

Line listing

Causative agent

- *Clostridium tetani*





Left. Stained pus from a mixed anaerobic infection. At least three different clostridia are apparent.

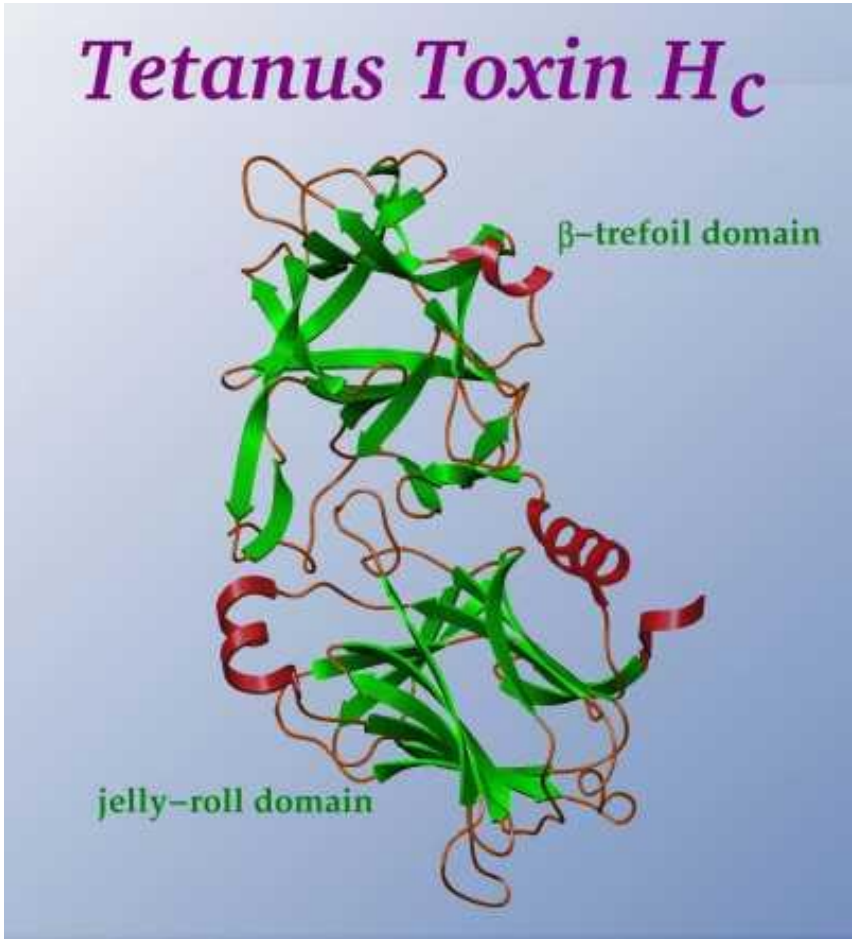
Right. Electron micrograph of vegetative *Clostridium tetani* cells.

Morphology & Physiology

- Relatively large, Gram-positive, rod-shaped bacteria
- Spore-forming, anaerobic.
- Found in soil, especially heavily-manured soils, and in the intestinal tracts and feces of various animals.
- Strictly fermentative mode of metabolism.

Virulence & Pathogenicity

- Not pathogenic to humans and animals by invasive infection but by the production of a potent protein toxin
  - **tetanus toxin** or **tetanospasmin**
  - The second exotoxin produced is tetanolysin—function not known.

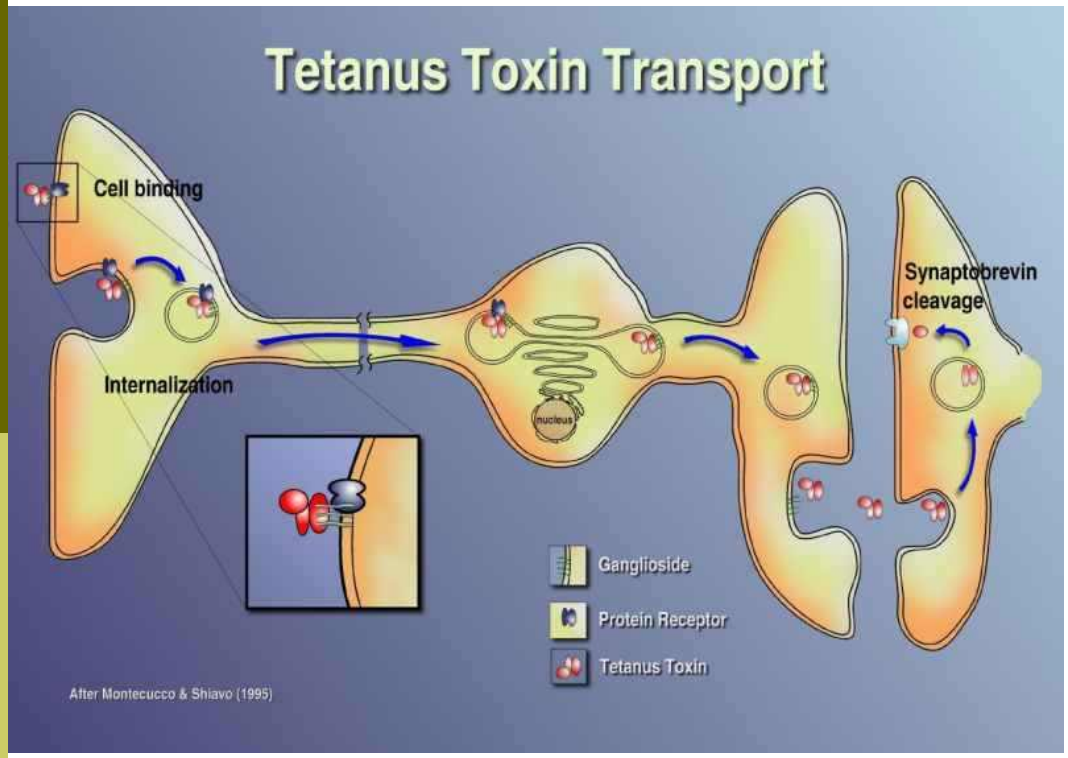


Tetanus toxin

- Produced when spores germinate and vegetative cells grow after gaining access to wounds. The organism multiplies locally and symptoms appear remote from the infection site.

- One of the three most poisonous substances known on a weight basis, the other two being the toxins of botulism and diphtheria.
  - Tetanus toxin is produced in vitro in amounts up to 5 to 10% of the bacterial weight.
  - Estimated lethal human dose of Tetanospamin = 2.5 nanograms/kg body

- Because the toxin has a specific affinity for nervous tissue, it is referred to as a **neurotoxin**. The toxin has no known useful function to *C. tetani*.



- Initially binds to peripheral nerve terminals
- Transported within the axon and across synaptic junctions until it reaches the central nervous system.
- Becomes rapidly fixed to gangliosides at the presynaptic inhibitory motor nerve endings, then taken up into the axon by endocytosis.

- Blocks the release of inhibitory neurotransmitters (glycine and gamma-amino butyric acid) across the synaptic cleft, which is required to check the nervous impulse.
  - If nervous impulses cannot be checked by normal inhibitory mechanisms, it leads to unopposed muscular contraction and spasms that are characteristic of tetanus.

Methods of transmission

- *C. tetani* can live for years as spores in animal feces and soil. As soon as it enters the human body through a major or minor wound and the conditions are anaerobic, the spores germinate and release the toxins.
- Tetanus may follow burns, deep puncture wounds, ear or dental infections, animal bites, abortion.
- Only the growing bacteria can produce the toxin.
- It is the only vaccine-preventable disease that is *infectious but not contagious* from person to person.

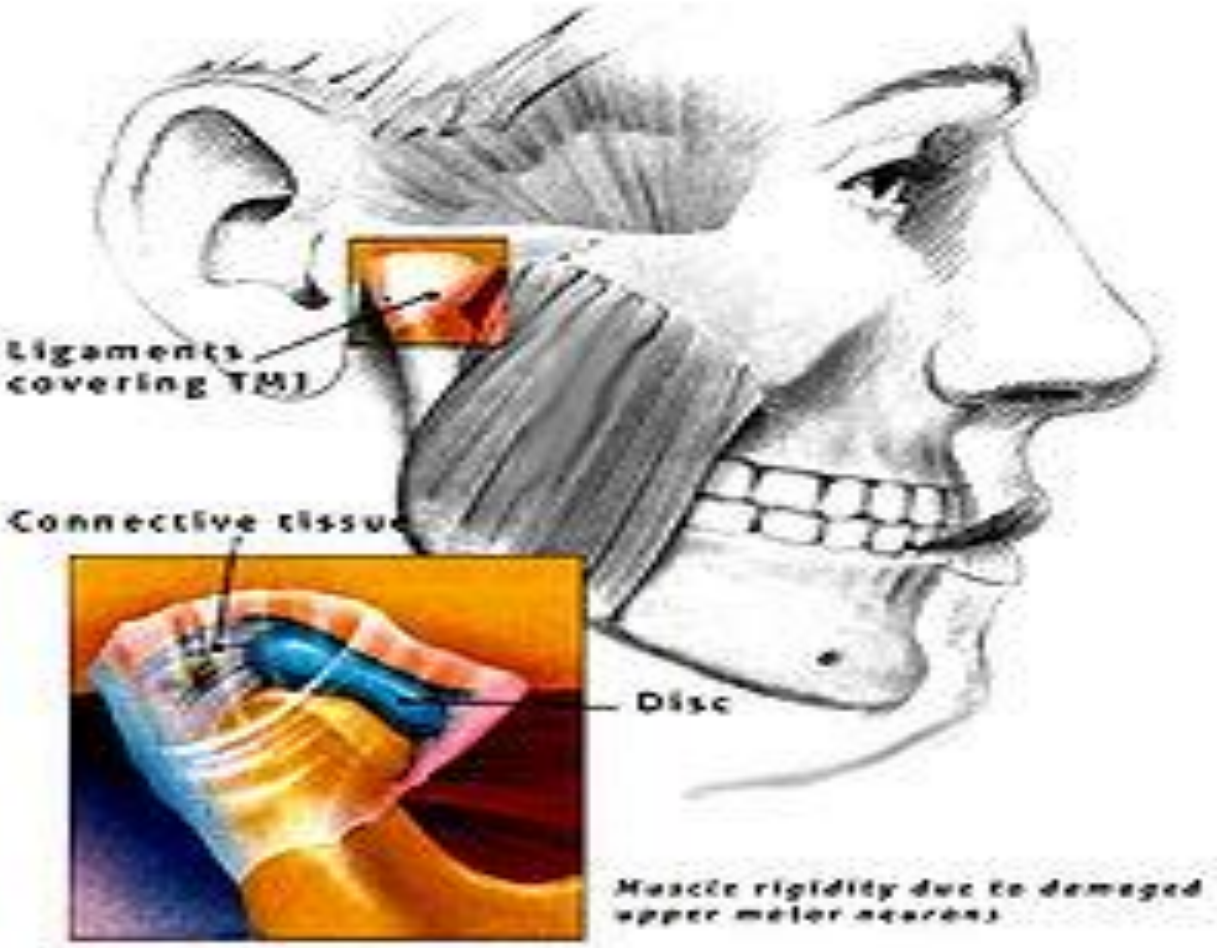
Symptoms

- Tetanic seizures (painful, powerful bursts of muscle contraction)
- if the muscle spasms affect the larynx or chest wall, they may cause asphyxiation
- stiffness of jaw (also called lockjaw)
- stiffness of abdominal and back muscles
- contraction of facial muscles
- fast pulse
- fever
- sweating

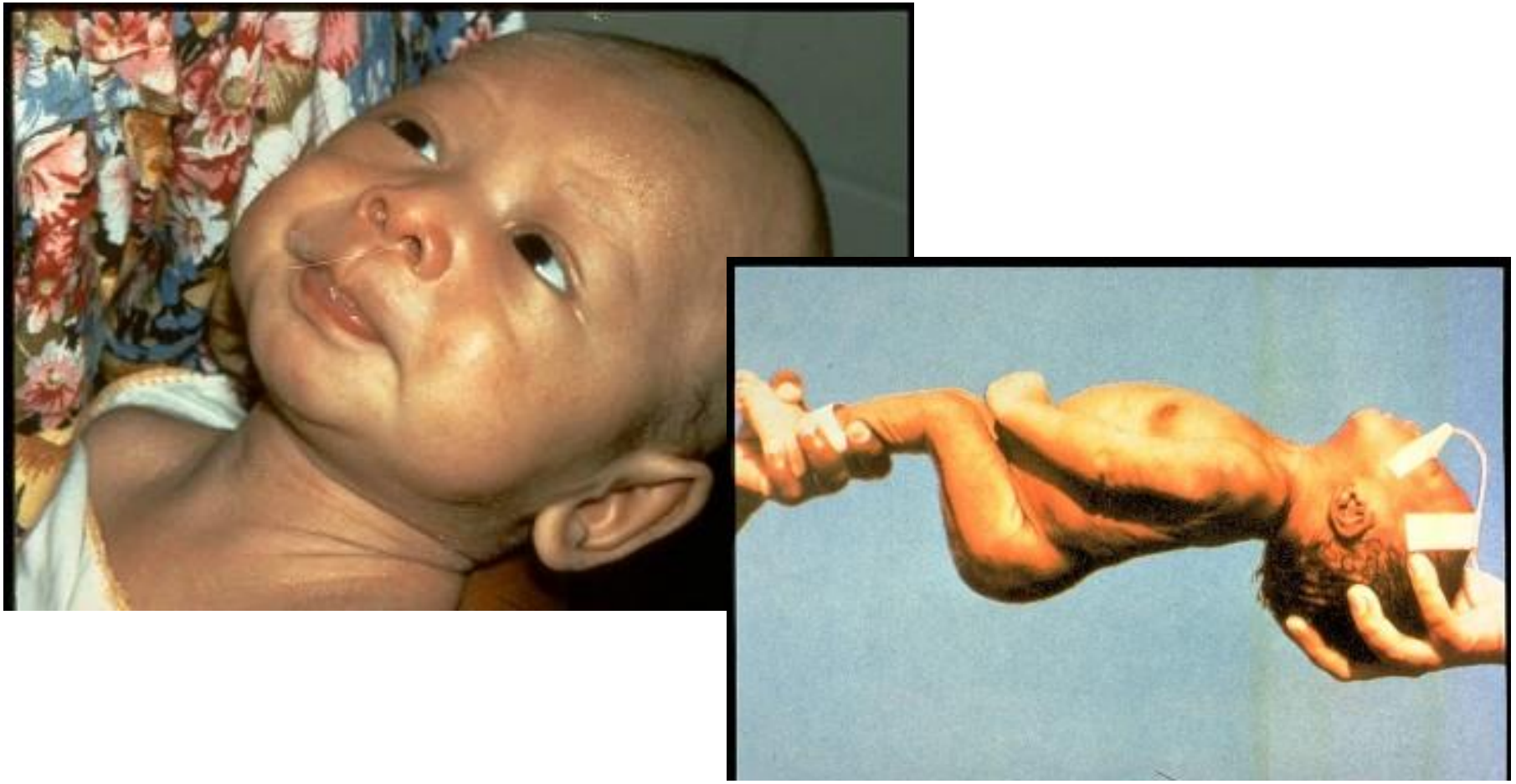
The back muscles are more powerful, thus creating the arc backward

"Opisthotonus" by Sir Charles Bell, 1809.

Baby has neonatal tetanus with complete rigidity


Ligaments covering TMJ
Connective tissue
Disc
Muscle rigidity due to damaged upper motor neurons

Clinical Neonatal Tetanus



Clinical Aspects of Neonatal Tetanus

- ***C. tetani*** **enters through unclean umbilical cord**
- **Signs & Symptoms**
  - **Trismus (risus sardonicus)**
  - **Neck stiffness**
  - **Body rigidity**
  - **Opisthotonus**
  - **Convulsions**
  - **Respiratory muscle weakness resulting in death**

Neonatal Tetanus

Any newborn baby that

- **sucks and cries normally during the first 2 (two) days of life;**

- **becomes ill between 3 and 28 days of life with BOTH**

1. **Inability to suck**

- and -

2. Generalized muscle rigidity (stiffness)

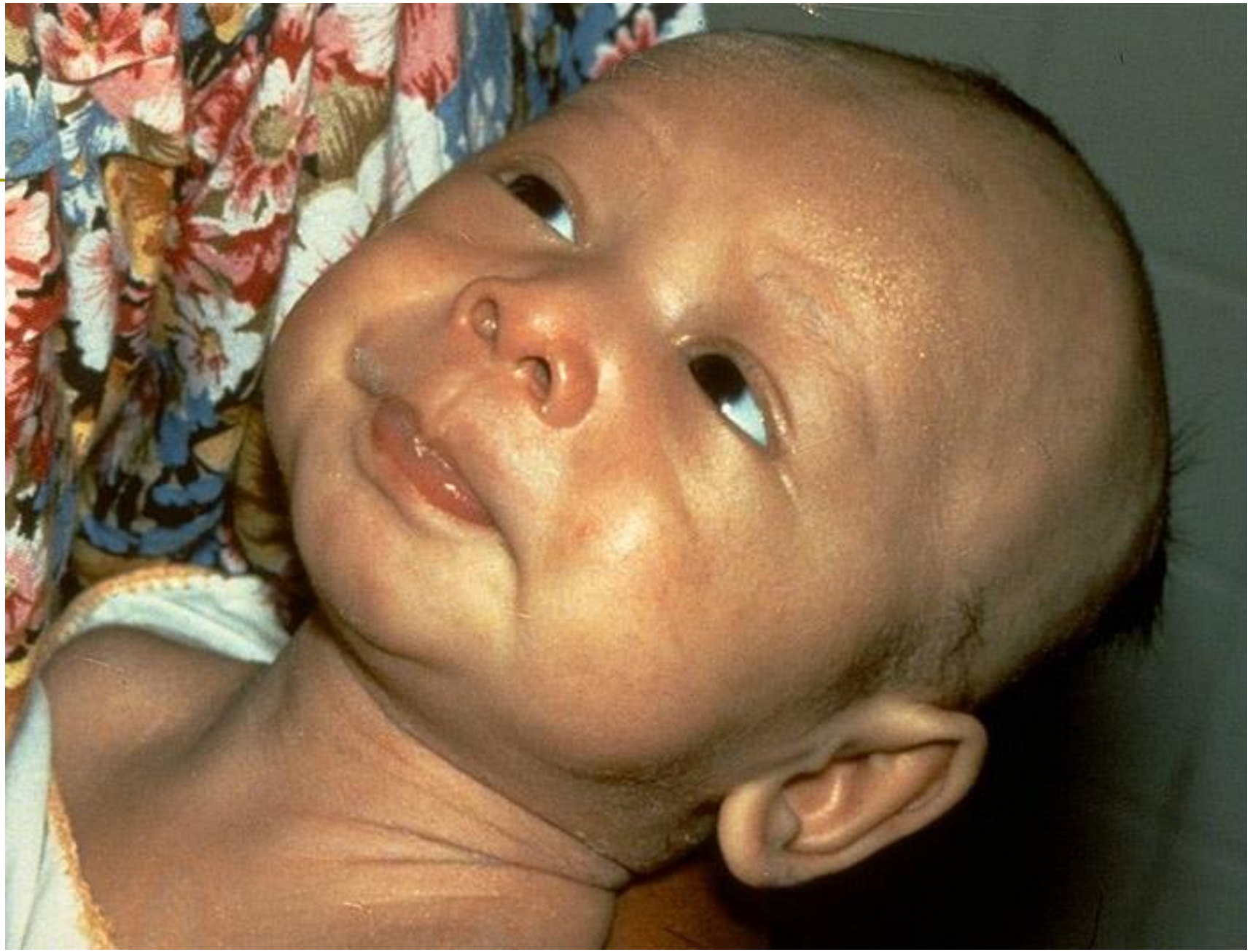
How to Prevent Neonatal Tetanus

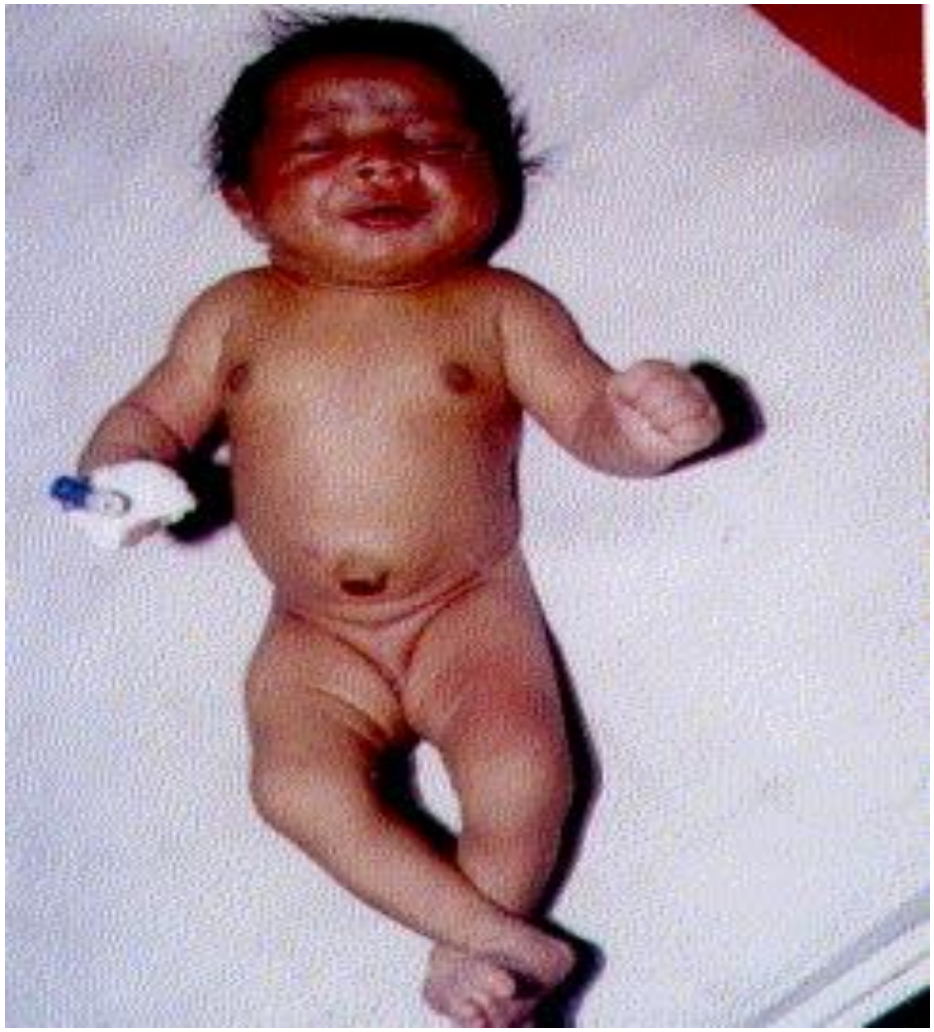
Two complimentary strategies

1. Clean delivery - “ 5 cleans”

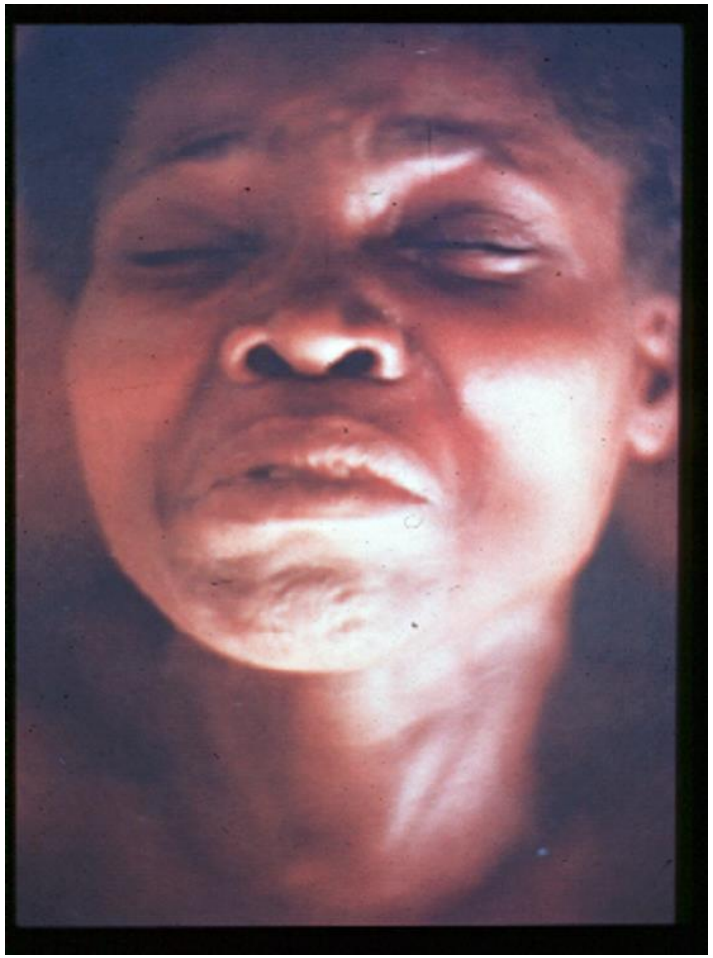
- **Clean delivery surface**
- **Clean hands**
- **Clean Thread**
- **Clean and New Blade**
- **Clean umbilical cord and stump care**

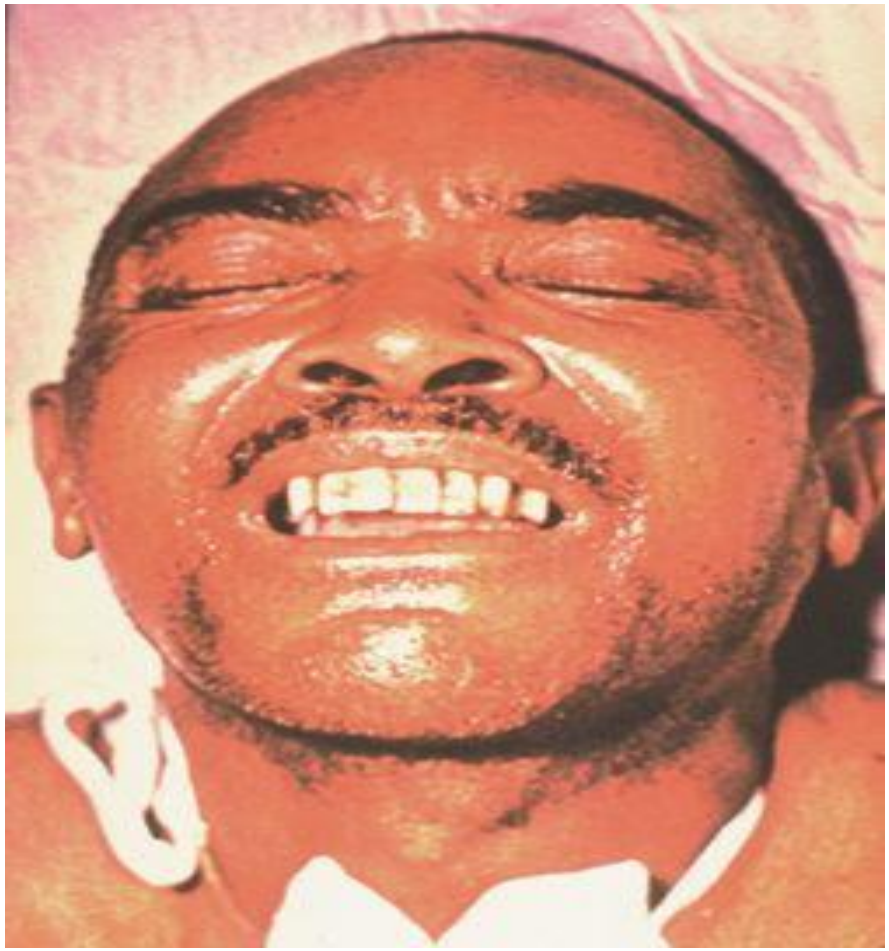
2. Immunization of mother with TT

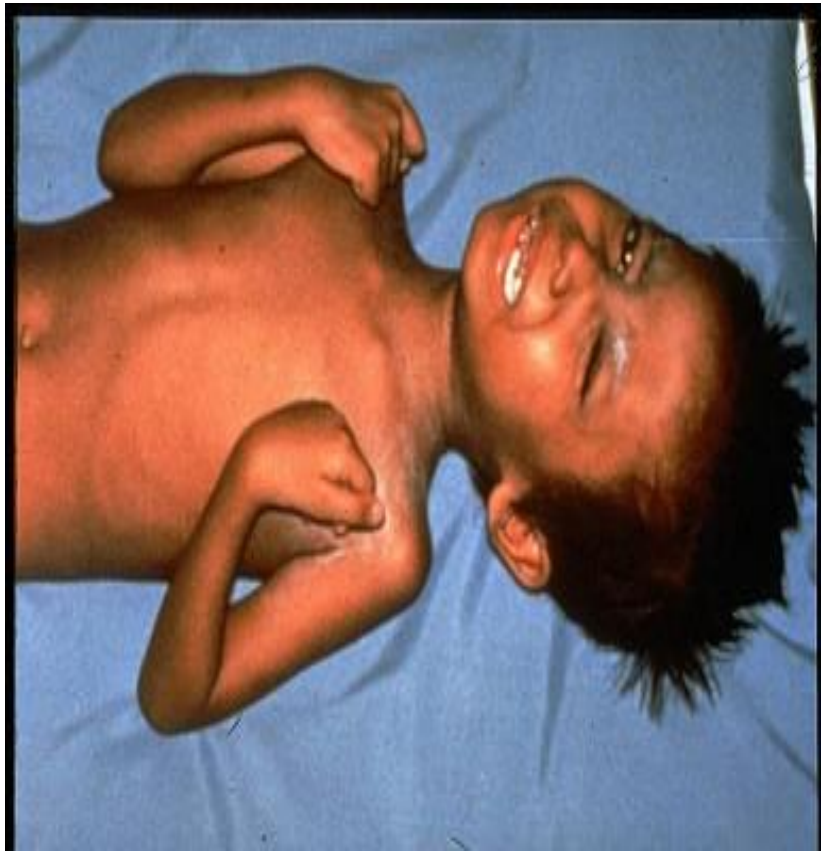




Newborn showing risus sardonicus and generalized spasticity







CEPHALIC TETANUS : A Rare Form of Localized Tetanus
Courtesy : Google image on tetanus)

Types of tetanus: local, cephalic, generalized, neonatal

- *Incubation period: 3-21 days, average 8 days.*

Uncommon types:

- **Local tetanus:** persistent muscle contractions in the same anatomic area as the injury, which will however subside after many weeks; very rarely fatal; milder than generalized tetanus, although it could precede it.

- **Cephalic tetanus:** occurs with ear infections or following injuries of the head; facial muscles contractions.

Most common types:

Generalized tetanus

- descending pattern: lockjaw → stiffness of neck → difficulty swallowing → rigidity of abdominal and back muscles.
- Spasms continue for 3-4 weeks, and recovery can last for months
- Death occurs when spasms interfere with respiration.

Neonatal tetanus:

- Form of generalized tetanus that occurs in newborn infants born without protective passive immunity because the mother is not immune.
- Usually occurs through infection of the unhealed umbilical stump, particularly when the stump is cut with an unsterile instrument.

Methods of diagnosis

- Based on the patient's account and physical findings that are characteristic of the disease.
- Diagnostic studies generally are of little value, as cultures of the wound site are negative for *C. tetani* two-thirds of the time.
  - When the culture is positive, it confirms the diagnosis of tetanus
- Tests that may be performed include the following:
  - Culture of the wound site (may be negative even if tetanus is present)
  - Tetanus antibody test
  - Other tests may be used to rule out meningitis, rabies, strychnine poisoning, or other diseases with similar symptoms.

Clinical treatment

- If treatment is not sought early, the disease is often fatal.
- The **bacteria** are killed with antibiotics, such as penicillin or tetracycline; further toxin production is thus prevented.
- The **toxin** is neutralized with shots of tetanus immune globulin, TIG.
- Other drugs may be given to provide sedation, relax the muscles and relieve pain.
- Due to the extreme potency of the toxin, immunity does not result after the disease.

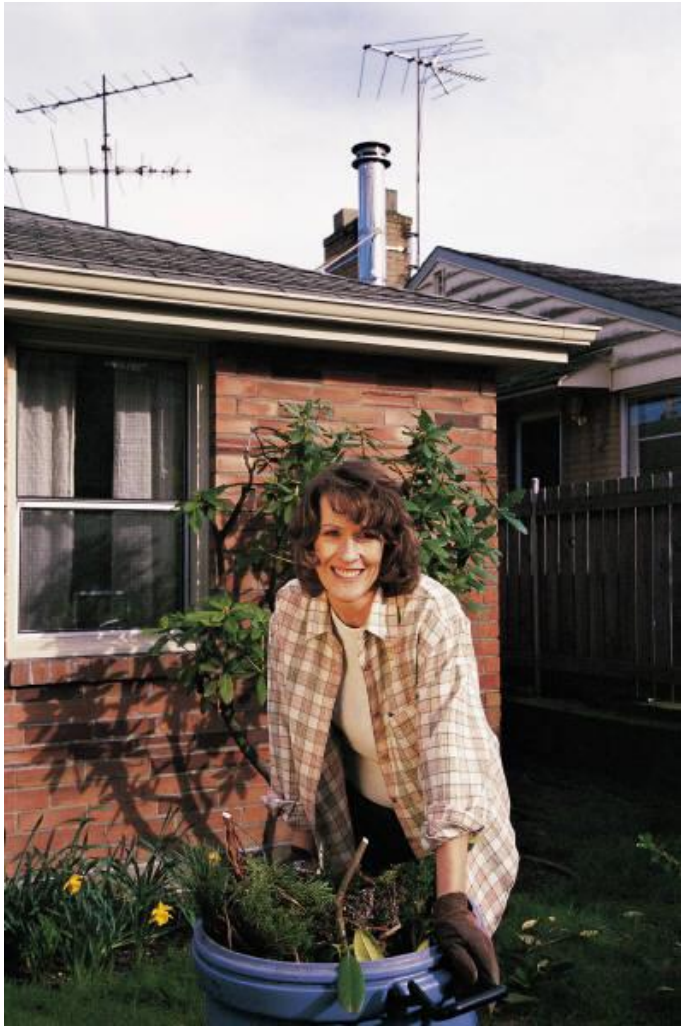
Method of prevention - immunization

- A person recovering from tetanus should begin active immunization with tetanus toxoid (TT) during convalescence.
- The tetanus toxoid is a formalin-inactivated toxin, with an efficiency of approx. 100%.
- The DPT vaccine includes tetanus, diphteria and pertussis toxoids; it is routinely given during childhood. After 7 years of age, only TT needs to be administered.
- Because the antitoxin levels decrease over time, booster immunization shots are needed every 10 years….??

What else can be done?

- Remove and destroy the source of the toxin through surgical exploration and cleaning of the wound (debridement).
- Bedrest with a nonstimulating environment (dim light, reduced noise, and stable temperature) may be recommended.
- Sedation may be necessary to keep the affected person calm.
- Respiratory support with oxygen, endotracheal tube, and mechanical ventilation may be necessary.

Have you had your Tetanus Shot?



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