



# Tetanus

## Definition

Tetanus is an acute, often fatal spastic paralytic disease caused by *Clostridium tetani*, mediated by its extremely potent neurotoxin tetanospasmin.

➡ Exam hook: Disease is toxin-mediated; bacteria remain localized at wound site.

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## Transmission & Epidemiology

### Source of Infection

- Spores widely present in:
  - Soil 🌱
  - Dust
  - Animal feces

### Portal of Entry

- Wound contamination:
  - Puncture wounds (e.g., rusty nail )
  - Lacerations, burns, crush injuries
- Skin-popping (subcutaneous drug injections in addicts)
- Neonatal tetanus :
  - Infection of umbilical stump
  - Circumcision wounds
  - Common in developing countries due to unhygienic delivery practices

### Predisposing Factors (Very Important )

- Presence of necrotic tissue → creates anaerobic environment
- Poor blood supply → reduced oxygen tension

 These conditions favor spore germination.

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## Pathogenesis

## Virulence Factor: Tetanospasmin

- Produced by: Vegetative *C. tetani* at wound site
- Nature: A-B type polypeptide exotoxin (neurotoxin)
- Potency: One of the most powerful toxins known

## Transport to CNS

- Toxin enters peripheral motor neurons
- Travels via retrograde axonal transport
- Reaches spinal cord & brainstem
- Binds to ganglioside receptors at inhibitory synapses

## Mechanism of Action (Core Concept )

- Tetanospasmin is a zinc-dependent endopeptidase
- Cleaves proteins required for neurotransmitter vesicle release
- Blocks release of inhibitory neurotransmitters:
  - Glycine
  - GABA

➡ Loss of inhibition → continuous motor neuron firing  
→ spastic paralysis

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### 🧩 Flowchart: Mechanism of Tetanospasmin

Wound contamination with spores → Anaerobic conditions develop → Spore germination → Vegetative cells produce tetanospasmin → Toxin enters peripheral nerves → Retrograde axonal transport to CNS → Binding to inhibitory interneurons → Blocked release of glycine & GABA → Unopposed motor neuron activity → Sustained muscle contraction → Spastic paralysis (tetany) 💥

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### ⚖️ Comparison: Tetanus vs Botulism

Feature	Tetanus toxin	Botulinum toxin
Neurotransmitter affected	GABA, Glycine (inhibitory)	Acetylcholine
Effect	Removes inhibition	Blocks excitation

Type of paralysis	Spastic	Flaccid
Site of action	CNS	Neuromuscular junction
Transport	Retrograde	Local/peripheral

➡ Exam favorite: Both are zinc endopeptidases, but effects are opposite.


### Antigenic Property

- Only one antigenic type
- Hence single toxoid sufficient for immunization 

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## Clinical Features

### Cardinal Manifestations

- Spastic paralysis (continuous muscle rigidity)
- Trismus (lockjaw)  - earliest & most common sign
- Risus sardonicus - fixed grimace due to facial muscle spasm

- Opisthotonos – severe arching of back due to extensor spasm
- Hyperreflexia – exaggerated reflexes
- Respiratory failure – spasm/paralysis of respiratory muscles (major cause of death)

 Mnemonic


 “LOCKed, SMILEd, and ARCHed”

Lockjaw – Risus sardonicus – Opisthotonos


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## Laboratory Diagnosis

- Primarily clinical diagnosis 
- No reliable microbiologic or serologic test
- *C. tetani* rarely isolated from wound

Microscopic Appearance (Spotter-worthy )

- Gram-positive, motile, anaerobic bacillus
- Terminal spore → “drumstick” / “tennis racket” appearance 



## Treatment of Tetanus



### Goals of Therapy

1. Neutralize unbound toxin
  2. Prevent further toxin production
  3. Control muscle spasms
  4. Provide supportive care
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### Management Steps


#### 1 Neutralization of Toxin

- Tetanus Immune Globulin (TIG) (human-derived)
- Neutralizes circulating toxin only
- Does NOT reverse toxin already bound to neurons

#### 2 Control of Infection

- Metronidazole (preferred) or Penicillin G
- Eliminates vegetative bacteria at wound site

### 3 Supportive Therapy

- Airway protection & ventilatory support
- Muscle relaxation:
  - Benzodiazepines (e.g., diazepam )

### 4 Wound Care

- Thorough cleaning
  - Surgical debridement to remove necrotic tissue
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### Flowchart: Management of Tetanus


Suspected tetanus → Secure airway & respiratory support → Administer TIG → Start metronidazole / penicillin G → Control spasms with benzodiazepines → Debride wound thoroughly → Begin active immunization with toxoid after stabilization

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### Prevention



## 1 Active Immunization

- Tetanus toxoid (formaldehyde-treated inactivated toxin)
- Given as part of DTaP vaccine 

Schedule:

- Primary childhood series
- Booster every 10 years

## 2 Post-Exposure Prophylaxis

- Clean wound

Clean wound + immunized (<10 yrs): → No treatment needed

Clean wound + non-immunized / >10 yrs: → Toxoid booster

Dirty wound: → Toxoid booster + TIG → Antibiotics (metronidazole/penicillin) → Half TIG infiltrated locally, remainder IM at separate site

## Key Notes ★

- Human TIG preferred over equine antitoxin
- Combined toxoid + TIG = Passive-Active Immunity



### One-Glance Summary Table

Organism	Disease	Transmission	Toxin Action	Prevention
<i>Clostridium tetani</i>	Tetanus	Spores enter wounds	Blocks GABA & glycine → spastic paralysis	Toxoid vaccine (DTaP)