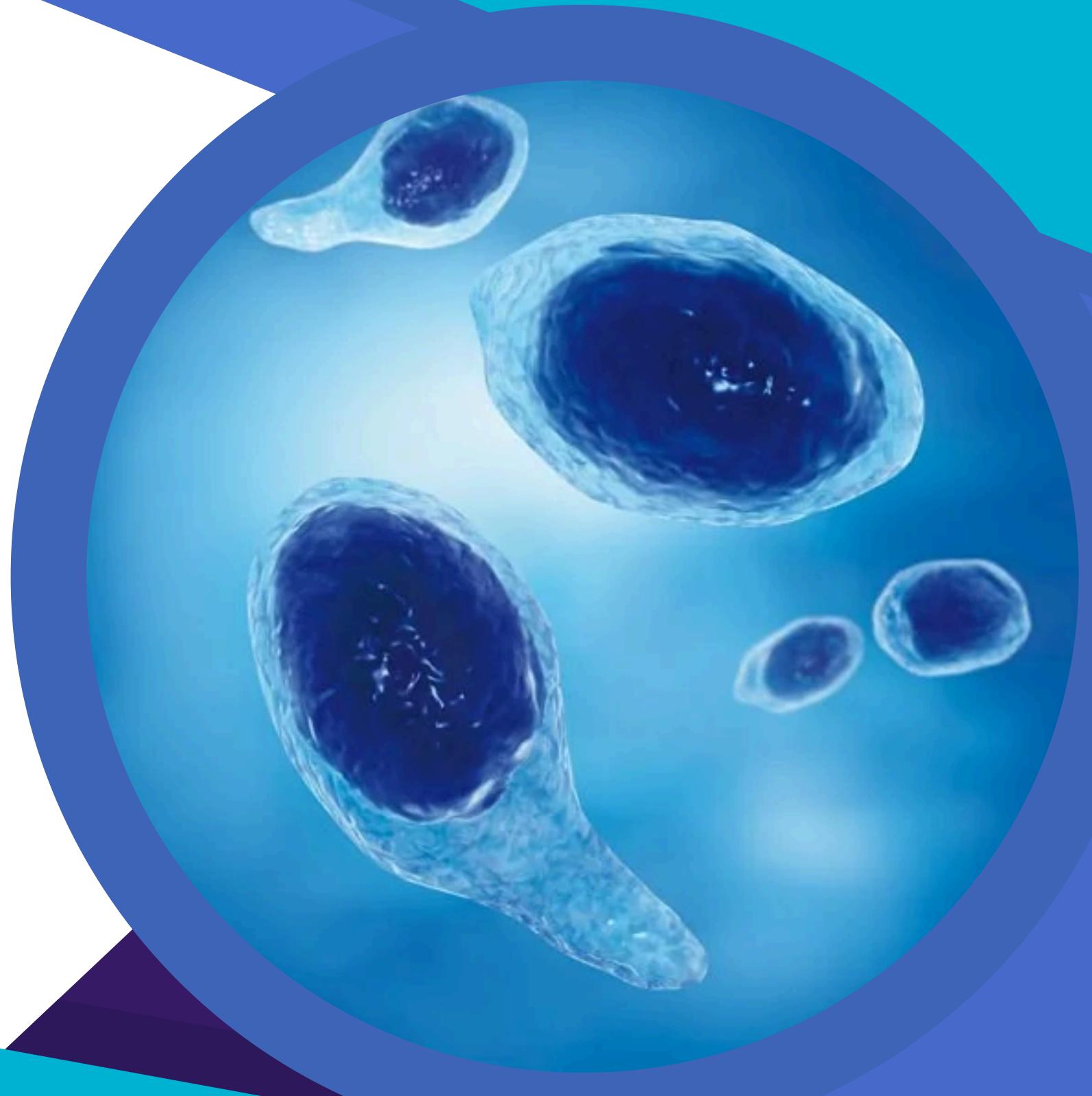




TETANUS *lockjaw*

a disease characterized by muscular spasm, caused by a neurotoxin produced by ***Clostridium tetani***.



LIST OF CONTENTS

BASIC INTRODUCTION

1

TREATMENT

CLINICAL SIGNS

2

MEDICATIONS

DIAGNOSIS

3

FOLLOW-UP

INTRODUCTION

Tetanus is a bacterial infection that is caused by a specific neurotoxin produced by *Clostridium tetani* in necrotic tissue. Horses seem to be the most sensitive of all species. In general, the occurrence of *C. tetani* in the soil, especially in cultivated soil, and the incidence of tetanus in horses is higher in the warmer parts of the various continents.



PATHOPHYSIOLOGY

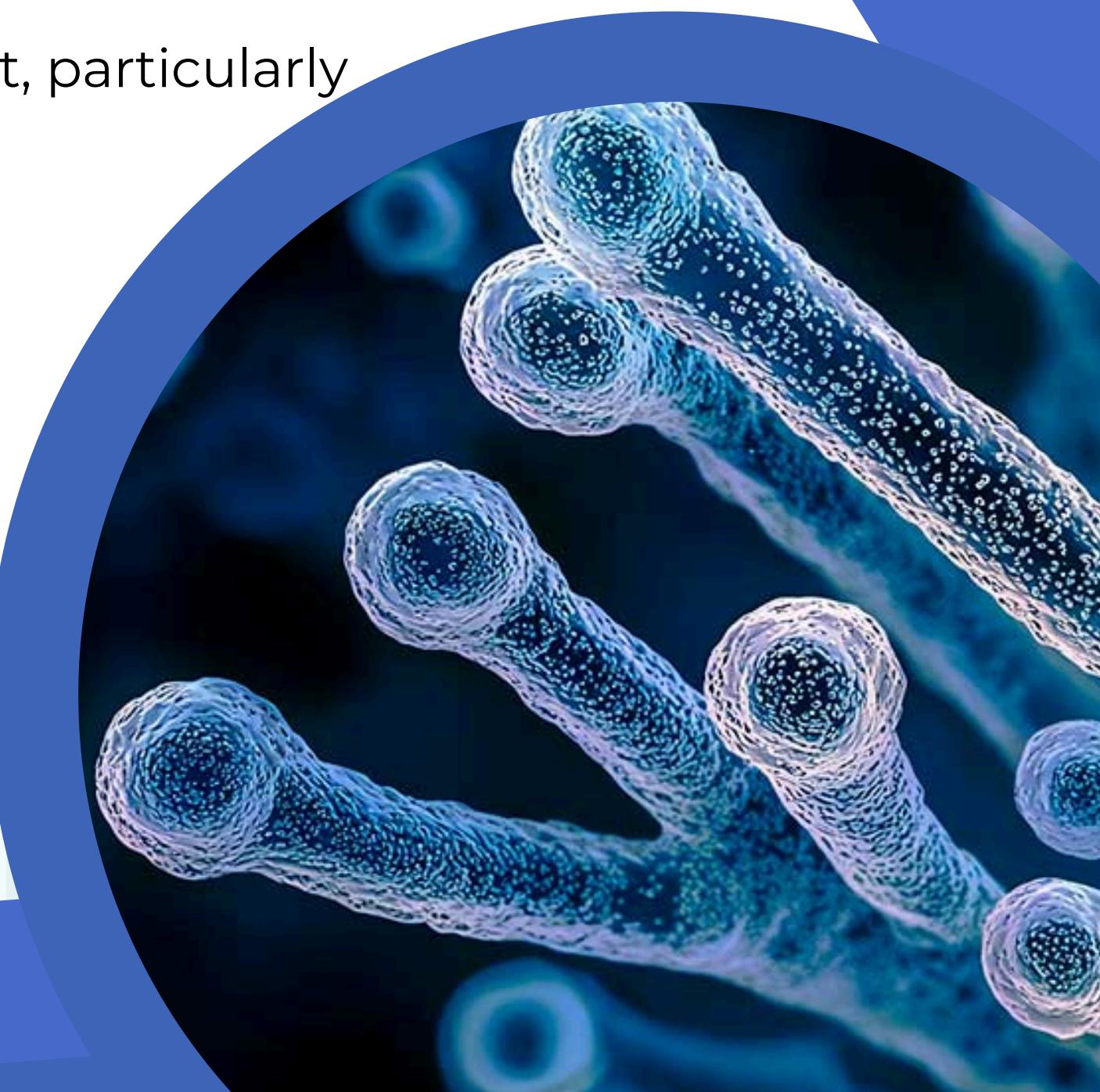
✓ CLOSTRIDIUM TETANI

- anaerobe, Gram-positive, spore-forming bacillus.
- terminal, spherical spores are widespread in the environment, particularly in soil and mammalian feces as well as intestinal tracts

✓ MODE OF ENTRY

gains access to the animal via a contaminated wound

- The **oxygen tension** within the wound must be **low** to allow **germination**.
- Concurrent infection with other bacteria, foreign bodies or necrosis in wound help produce an anaerobic tissue environment.
- C. tetani organisms proliferate locally.



PATHOPHYSIOLOGY

✓ TOXIN PRODUCTION

- Death/lysis of organisms → release **tetanospasmin** (main neurotoxin)

✓ ACTION OF TETANOSPASMIN

- Tetanospasmin travels to the CNS via the hemolymphatic system and peripheral motor nerves.
- The toxin acts on presynaptic inhibitory interneurons in the ventral horn of the spinal cord.
- It cleaves **synaptobrevin**, a protein necessary for release of the neurotransmitters glycine and γ-aminobutyric acid.
- This results in a loss of **motor neuron inhibition**, **hypertonia** and **muscular spasm**.



PATHOPHYSIOLOGY



2 OTHER EXOTOXINS OF CLOSTRIDIUM TETANI

- **Tetanolysin** - increase local tissue necrosis and promotes proliferation within the wound.
- **Non-spasmogenic toxin** - have a sympathomimetic effect.



INCUBATION PERIOD

- highly variable, but it is usually **1–3 weeks**.
- The spores can survive in tissue and germinate after wound healing if conditions then become favorable.
- Castration wounds and injection sites have also been associated with the development of tetanus



SYSTEMS AFFECTED



Neuromuscular

The toxin causes spasmotic, tonic contractions of the voluntary muscles and paralysis



Respiratory

Spasms affecting the larynx, diaphragm, and intercostal muscles lead to respiratory failure



Skeletal

The spasms may be severe enough to cause bone fractures

INCIDENCE/PREVALENCE

Horses are exquisitely sensitive to the toxin, and the disease has a worldwide distribution.

A higher incidence may be associated with poor husbandry.

There may be a higher incidence in warmer areas.

SIGNALMENT/GENETICS

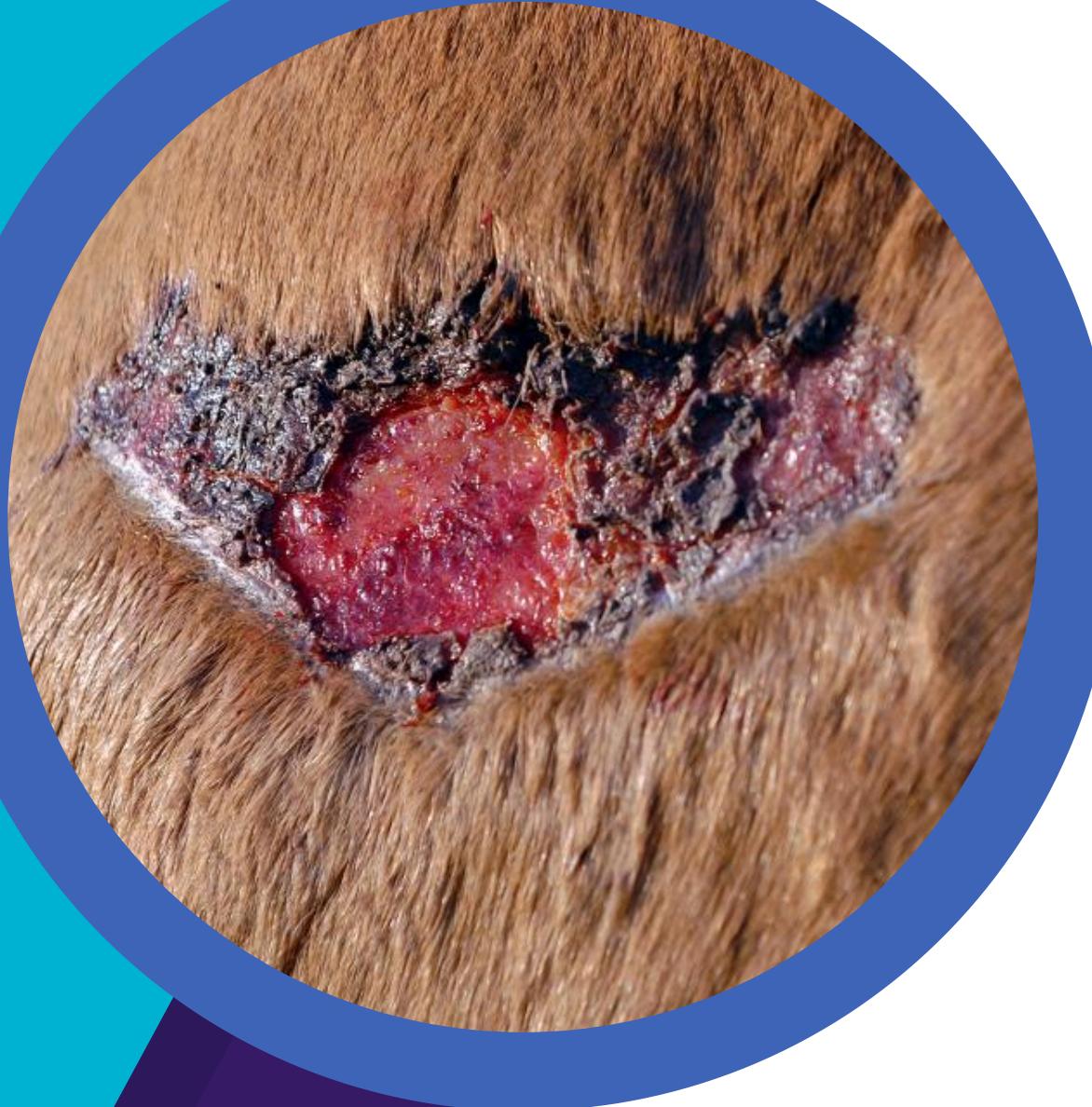
No sex, age, or breed predilections. Genes are also not involved.

RISK FACTORS

Unvaccinated horses that have sustained a contaminated soft tissue wound or penetrating wound to the foot are most at risk. Tetanus may occur even in vaccinated horses.

CLINICAL SIGNS

- History of a wound **1–4 weeks** earlier.
- The first signs may be local stiffness, lameness, colic.
- The progression of signs depends on the extent of infection, vaccination status, and the age and size of the horse.
- Generally, signs progress within 24 hours, with the horse beginning to exhibit a **stiff/spastic gait**.

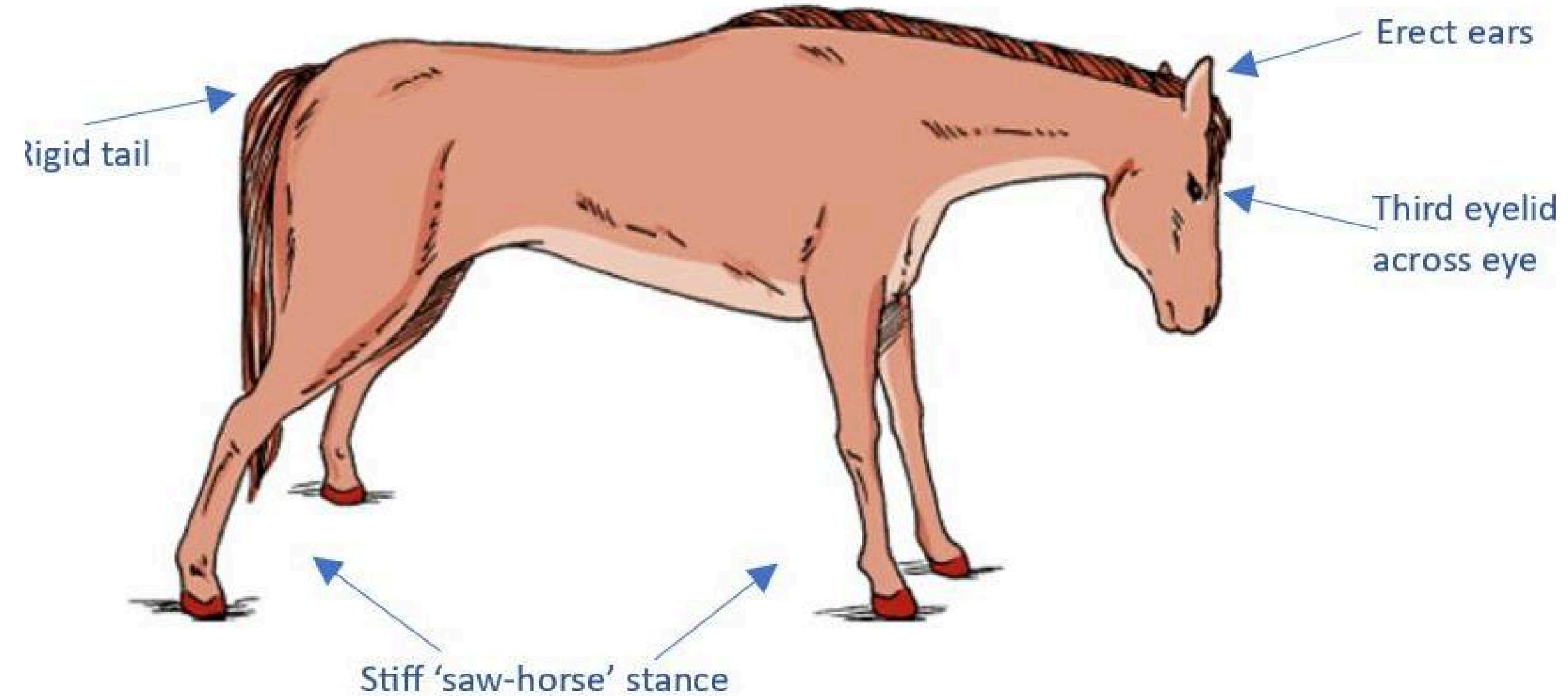


CLINICAL SIGNS

- **Trembling, raised tail-head, flared nostrils, and erect ears** are seen.
- Preferential effects on postural muscles result in the characteristic “**sawhorse stance.**”
- **Retraction of the eyes and protrusion of the third eyelid** occur following a stimulus (noise or menace).
- Spasm of the masseter muscles causes inability to open the mouth (“**lockjaw**”)



CLINICAL SIGNS



CLINICAL SIGNS



PROLAPSE OF
THIRD EYELID



SAWHORSE STANCE



LOCKJAW

CLINICAL SIGNS



- Dysphagia results in saliva accumulation and aspiration of feed material.
- Increased rectal temperature and profuse sweating occur due to prolonged muscle spasms.
- All signs are exacerbated by stimulation and excitement.
- As the disease progresses:
 - Recumbency with difficulty or inability to rise.
 - Severe extensor rigidity may occur.
 - Horses may have difficulty urinating and defecating.
- Respiratory failure occurs in fulminant (severe) cases.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- Laminitis
- Hypocalcemia
- Rhabdomyolysis
- Rabies
- Myotonia

CBC/BIOCHEMISTRY/URINALYSIS

- Nonspecific
- Hemoconcentration and a stress leukogram may be present.
- May see hyperfibrinogenemia and leukocytosis with secondary aspiration pneumonia



DIAGNOSIS

OTHER LABORATORY TESTS

- Anaerobic culture of *C. tetani* may be attempted from a wound

IMAGING

- No specific diagnostic indications
- Thoracic radiography or ultrasonography if aspiration pneumonia is suspected.
- Ultrasonography of wound sites may help confirm anaerobic infection.

PATHOLOGIC FINDINGS

- Nonspecific.
- May demonstrate a *C. tetani*-infected wound.
- Secondary traumatic injury or aspiration pneumonia may be present.



TREATMENT

✓ APPROPRIATE HEALTHCARE

- Initial treatment is aimed at neutralizing unbound toxin and preventing further release by eliminating the infection.
- Appropriate nursing care, particularly if the horse is recumbent, is vital to maximize the chances of a successful outcome.
- Fluid therapy may be required to maintain hydration.
- Nasogastric fluids have the added benefit of hydrating colonic contents; however,
- IV or rectal administration of fluids may be necessary in horses where a nasogastric tube cannot be maintained.

TREATMENT

✓ NURSING CARE

- Confine to a quiet, dark stall with deep bedding.
- Minimize auditory stimulation with ear plugs.
- Use padded walls and/or a padded helmet to minimize injury.
- Frequently turn recumbent horses (every 2–4 hours).
- Recumbent horses unable to rise may benefit from slinging.
- Manual rectal evacuation and/or urinary catheterization may be necessary.



TREATMENT

ACTIVITY

- Restrict activity as much as possible through confinement and sedation.

DIET

- High-quality feed and free-choice water should be made easily accessible.
- If the horse is dysphagic, a nasogastric tube can be placed for administration of feed, water, and electrolytes.
- The tube can be left in place to avoid the stress of repeated passage.
- In some cases, passage of a nasogastric tube is not possible, and feeding via esophagostomy, gastrostomy, or parenteral nutrition may be required.

TREATMENT

✓ CLIENT EDUCATION

- Appropriate tetanus prophylaxis should be discussed.

✓ SURGICAL CONSIDERATIONS

- Debride the wound and maximize exposure to air
- Esophagostomy or gastrostomy may facilitate feeding in severely dysphagic cases.
- Tracheostomy may be necessary if laryngeal spasm and respiratory obstruction have occurred.

MEDICATIONS



DRUGS OF CHOICE

- **Tetanus antitoxin** — 100–200 U/kg IV or IM (single dose) to bind circulating toxin.
- **Acepromazine** — 0.05–0.08 mg/kg IV or IM every 3–6 hours as required.
- **Phenobarbital** — 6–12 mg/kg slow IV, followed by 6–12 mg/kg PO every 12 hours, alone or in combination with acepromazine.
- **Penicillin G (potassium or sodium)** — 22,000–44,000 IU/kg IV every 6 hours for 7–10 days.

MEDICATIONS



DRUGS OF CHOICE

- Consider intrathecal administration of 50 mL TAT (20–30 mL in foals) after removal of an equal amount of cerebrospinal fluid from the atlanto-occipital space (requires general anesthesia) or via lumbosacral puncture in the standing horse. (Most beneficial early in the disease process.)
- Local infiltration of the wound with procaine penicillin and/or tetanus antitoxin (3,000–9,000 IU) may help eliminate infection and neutralize toxin present at the site.
- Vaccination with tetanus toxoid — clinical disease does not result in sufficient immune response.
- Use a separate injection site for the antitoxin.



MEDICATIONS



PRECAUTIONS

- TAT has been associated with the development of Theiler's disease (serum hepatitis).
- General anesthesia and intrathecal TAT administration can result in significant complications (meningitis, seizures).



POSSIBLE INTERACTIONS

- TAT will bind tetanus toxoid — these agents should be administered at different sites.
- Phenothiazine drugs (e.g., acepromazine) may potentiate barbiturates, causing more profound CNS depression if used together.

MEDICATIONS



✓ ALTERNATIVE DRUGS

- **Magnesium ($MgSO_4$)** administered via IV constant rate infusion induces muscle relaxation, and reduces the requirement for other muscle relaxants and sedatives in human tetanus patients.
- Monitoring of serum Mg levels and ECG for signs of toxicity (widening of the QRS complex) is recommended during therapy.
- **Haloperidol** — 0.01 mg/kg IM every 7 days for long-acting sedation.
- **Diazepam** — 0.01–0.4 mg/kg IV every 2–4 hours.
- **Macrolides (in foals only), tetracyclines, and metronidazole** are alternatives to penicillin that may also be effective in eliminating vegetative *C. tetani* at the infection site.

FOLLOW-UP



PATIENT MONITORING

- Regular physical examination.
- Serial monitoring of packed cell volume (PCV), total protein concentration, and/or urine specific gravity to monitor hydration.



PREVENTION/AVOIDANCE

- Initial vaccination with 2 doses of tetanus toxoid given 3–4 weeks apart.
- Annual toxoid booster thereafter is the current recommendation (although new evidence suggests horses may have protective antibody titers for at least 3 years after the initial vaccine course).
- Tetanus toxoid should be administered in the case of a wound if there has not been vaccination within the past 6 months.



FOLLOW-UP



PREVENTION/AVOIDANCE

- Pregnant mares should be given a toxoid booster 4–6 weeks prior to expected parturition.
- Experimental studies indicate that immunity to tetanus challenge is present 8 days after administration of toxoid in horses.



POSSIBLE COMPLICATIONS

- Myopathy
- Aspiration pneumonia
- Trauma (fractures, decubital ulcers)
- Idiopathic acute hepatic disease (Theiler's disease) is a rare complication of TAT administration



FOLLOW UP



EXPECTED COURSE AND PROGNOSIS

- Horses that are recumbent and unable to rise have a grave prognosis, particularly if progression has been rapid.
- The presence of dyspnea and dysphagia may also negatively influence survival.
- Horses that retain the ability to stand and ambulate have a fair prognosis.



FOLLOW UP

EXPECTED COURSE AND PROGNOSIS

- Clinical signs may persist for weeks; however, survivors generally stabilize after **7 days** and begin to show improvement after **2 weeks**.
- **Recovery** may take as long as **6 weeks** but is usually complete.
- The attitude of the individual horse and the ability to provide ideal nursing care are important factors affecting outcome.
- The **overall mortality rate** in horses is reported to be **50–80%**.





REFERENCES



Lavoie, J.-P. (2019). Blackwell's Five-Minute Veterinary Consult: Equine (3rd ed.). Wiley Blackwell.
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