



Tick Paralysis

Basics

OVERVIEW

- Paresis—weakness of voluntary movement
- Paralysis—lack of voluntary movement
- Tetraparesis—weakness of voluntary movements in all legs
- Tetraplegia—absence of all voluntary movement in the legs
- “Lower motor neuron paralysis” is the loss of voluntary movement caused by disease of the nerves that connect the spinal cord and muscles
- “Tick paralysis” is a lower motor neuron weakness of all legs (tetraparesis) to paralysis of all legs (tetraplegia), characterized by relaxed muscles or muscles without tone (known as “flaccid paralysis”); caused by nerve toxins found in the saliva of females of certain tick species
- Also known as “tick-bite paralysis”

GENETICS

- No genetic basis

SIGNALMENT/DESCRIPTION OF PET

Species

- United States—dogs; cats appear to be resistant
- Australia—dogs and cats

SIGNS/OBSERVED CHANGES IN THE PET

- Pet walked in a wooded or open area approximately 1 week before onset of signs
- Onset—gradual; starts with unsteadiness and weakness in the rear legs

Disease Caused by North American Ticks

- Once nervous system signs appear, rapidly ascending (that is, moving from rear legs to front legs and then head), generalized lower motor neuron weakness of all legs (tetraparesis”) to paralysis of all legs (tetraplegia)
- Pet becomes extremely weak to recumbent in 1–3 days, with decreased reflexes (known as “hyporeflexia”) to lack of reflexes (known as “areflexia”) and decreased muscle tone (known as “hypotonia”) to lack of muscle tone (known as “atonia”)
- Pain sensation is preserved
- Cranial nerve dysfunction—not a prominent feature; may note facial weakness and reduced jaw tone; sometimes a change in voice (known as “dysphonia”) and difficulty swallowing (known as “dysphagia”) may be seen early in the course of disease; the “cranial nerves” are nerves that originate in the brain and go to various structures of the head (such as the eye, face, and tongue)
- Paralysis of breathing muscles (known as “respiratory paralysis”)—uncommon in cases in the United States;

may occur in severely affected pets

- Urination and defecation usually are normal

Disease Caused by an *Ixodes* Tick

- Nervous system signs—much more severe and rapidly progressive; ascending motor weakness (that is, moving from rear legs to front legs and then head) can progress to paralysis of all legs (tetraplegia) within a few hours
- Excessive salivation, enlarged esophagus (the tube running from the throat to the stomach; condition known as “megaesophagus”), and vomiting (forceful ejection of stomach contents up through the esophagus and mouth) or regurgitation (passive, backward movement or return of food or other contents from the esophagus into the throat or mouth) are characteristic
- Dilated and poorly responsive pupils
- High blood pressure (known as “hypertension”); rapid, irregular heartbeats (known as “tachyarrhythmias”)
- Fluid buildup in the lungs (known as “pulmonary edema”)
- Paralysis of breathing muscles—much more common than seen with North American related tick paralysis; dogs and cats progress to difficulty breathing (known as “dyspnea”); bluish discoloration of the skin and moist tissues (known as “mucous membranes”) of the body caused by inadequate oxygen levels in the red blood cells (known as “cyanosis”); and respiratory paralysis within 1–2 days, if not treated

CAUSES

United States

- *Dermacentor variabilis*—common wood tick
- *Dermacentor andersoni*—Rocky Mountain wood tick
- *Amblyomma americanum*—lone star tick
- *Amblyomma maculatum*—Gulf Coast tick

Australia

- *Ixodes holocyclus*—Australia paralysis tick; secretes a far more potent nerve toxin than that of the North American species

RISK FACTORS

- Environments that harbor ticks
- United States—*Dermacentor variabilis*: wide distribution over the eastern two-thirds of the country and in California and Oregon; *Dermacentor andersoni*: from the Cascades to the Rocky Mountains; *Amblyomma americanum*: from Texas and Missouri to the Atlantic Coast; *Amblyomma maculatum*: the Atlantic and Gulf of Mexico seaboard
- Australia—*Ixodes holocyclus*: limited to the coastal areas of the east; especially associated with areas of bush and scrub

Treatment

HEALTH CARE

- Inpatient—any nervous system dysfunction suggesting tick paralysis; hospitalize until either a tick is found and removed or appropriate treatment to kill a hidden tick is performed
- Inpatient supportive care—essential until pet begins to show signs of recovery
- Oxygen cage—for pets with decreased ability to breathe (known as “hypoventilation”) and low levels of oxygen in the body (known as “hypoxia”)
- Artificial ventilation—for pets with breathing failure or respiratory paralysis
- Intravenous fluid therapy—generally not required, unless recovery is prolonged

ACTIVITY

- Keep pet in a quiet environment
- *Ixodes* tick paralysis—keep pet in a cool, air-conditioned area; toxin is temperature sensitive; avoid activity to prevent increase in body temperature

DIET

- Withhold food and water, if pet has difficulty swallowing (dysphagia) or vomiting/regurgitation

Medications

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive

- United States—if the tick cannot be found, treat the pet with a systemic insecticide (such as fipronil [Frontline®]) applied to the skin (known as “topical treatment”) or, alternatively, use an insecticidal dip, following directions for use on the product label; often the only treatment needed
- Australia—must neutralize circulating nerve toxin, depending on severity of clinical signs; if severe, phenoxybenzamine, an α -adrenergic antagonist appears to be beneficial; acepromazine can be used as an alternative medication

Follow-Up Care

PATIENT MONITORING

- Non-*Ixodes* tick—reassess nervous system status after tick removal at least daily—should see rapid improvement in muscle strength
- *Ixodes* tick—monitor nervous system status and breathing and circulatory functions continuously and intensively even after tick removal, because of the residual effect of the nerve toxin

PREVENTIONS AND AVOIDANCE

- Vigilantly check for ticks after possible exposure (at least every 2–3 days); signs do not occur for 4–6 days after tick attachment
- Routine topical application of fipronil (Frontline) or weekly insecticidal baths prevent tick paralysis (by keeping ticks off the pet or by killing the ticks before the nerve toxin has reached a level in the pet's body to cause signs)
- Short-term immunity develops after exposure to *Ixodes* nerve toxin

POSSIBLE COMPLICATIONS

- No long-term complications, if the pet survives the sudden (acute) effects of the nerve toxin
- Death

EXPECTED COURSE AND PROGNOSIS

- Non-*Ixodes* tick—prognosis good to excellent, if ticks are removed; recovery occurs in 1–3 days (3 days maximum)
- *Ixodes* tick—prognosis often guarded; recovery prolonged; death in 1–2 days, without treatment

Key Points

- Non-*Ixodes* tick—good nursing care is essential, although the pet's recovery is rapid after removal of ticks (often within 24–48 hours)
- *Ixodes* tick—signs often continue to worsen despite tick removal (weakness often intensifies 24–48 hours after tick removal); thus more aggressive treatment to neutralize the nerve toxin must be undertaken

Notes

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