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Canine Tick Paralysis

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TICK PARALYSIS affects nearly all warm-blooded land animals, including humans.[1] When it is diagnosed in the early stages, tick paralysis is easily treatable using pharmacotherapy (Table 1). However, in the absence of treatment, mortality rates of up to 10% in humans and 7% in domestic animals have been reported.[1]

Unlike tick-borne diseases that are caused by infection with a pathogen, such as Lyme disease and Rocky Mountain spotted fever, tick paralysis is caused by transmission of a neurotoxin secreted by the salivary glands of gravid (egg-laden) female ticks during feeding.[2,3] The neurotoxin responsible for tick paralysis was first isolated in 1966[4]; it causes a form of motor paralysis similar to that associated with Guillain-Barré syndrome in humans.[3]

Causative Agent

Unlike fleas, ticks cannot jump. Instead, they climb on foliage and "hitch a ride" on nearby humans or animals. As many as 64 species of ticks have the potential to induce paralysis in animals.[5] In North America, the six species typically implicated in cases of tick paralysis are *Dermacentor andersoni* (the Rocky Mountain wood tick), *D. variabilis* (the American dog tick), *Amblyomma americanum* (the lone star tick), *A. maculatum* (the Gulf Coast tick), *Ixodes scapularis* (the black-legged tick), and *I. pacificus* (the western black-legged tick).[3] It is thought that gravid females are responsible for neurotoxin transmission because they feed for a longer period and inject greater amounts of neurotoxin into the host than do males and nongravid females.[6]

Pathogenesis

Tick paralysis primarily affects a dog's motor pathways, although its autonomic and sensory pathways also may be compromised.[5] The neurotoxin depolarizes the acetylcholine

Table 1. Tick Control Products for Dogs[a]

Product (Manufacturer)	Active Ingredient(s)	Minimum Age	Route of Administration	Frequency[b]
Frontline Plus for Dogs (Merial)	Fipronil, (*S*)-methoprene	8 weeks	Topical	Monthly
Frontline Spray (Merial)	Fipronil	8 weeks	Spray	Monthly
Frontline Top Spot for Dogs (Merial)	Fipronil	8 weeks	Topical	Monthly
K9 Advantix (Bayer Animal Health)	Imidacloprid, permethrin	7 weeks	Topical	Monthly
ProMeris for Dogs (Fort Dodge Animal Health)	Metaflumizone, amitraz	8 weeks	Topical	Monthly
Proticall (Intervet/Schering-Plough Animal Health)	Permethrin	4 weeks	Topical	Monthly
Revolution[c] (Pfizer Animal Health)	Selamectin	6 weeks	Topical	Monthly
Vectra 3D (Summit VetPharm)	Dinotefuran, permethrin, pyriproxyfen	7 weeks	Topical	Monthly
Virbac Long-Acting KnockOut Spray (Virbac Animal Health)	Permethrin, pyriproxyfen	12 weeks	Spray	Monthly
Virbac Pyrethrin Dip (Virbac Animal Health)	Pyrethrins, piperonyl butoxide, N-octyl bicycloheptene dicarboximide, di-N-propyl isocinchomeronate	12 weeks	Dip	No more than once every 7 days

[a] These products also are approved for control of other parasites.
[b] See the product label for information on the exact dosing frequency.
[c] Approved for control of *Dermacentor variabilis* infestation.

release mechanism in the presynaptic nerve terminals, causing a reduction in secretion.[7] Because of the reduction in bioavailable acetylcholine, the animal develops flaccid paralysis (loss of muscle control).

Respiratory failure may occur because of pulmonary edema, respiratory muscle paralysis, or aspiration of esophageal or gastric contents. Affected dogs usually experience paralysis of the esophageal muscles and may develop megaesophagus (esophageal dilation). Saliva and ingested food or fluid then collect in the esophagus and are regurgitated into the pharynx and mouth. Because of the loss of the gag reflex, the animal cannot clear this material from its airway, potentially causing aspiration pneumonia.[5] Death usually results from respiratory failure caused by paralysis of the diaphragm, intercostal muscles, and muscles in the upper and lower airways.[4]

Clinical Signs

On average, clinical signs of tick paralysis begin to appear 6 to 9 days after tick attachment, but a range of 3 to 10 days has been reported.[7] Patients may present with mild signs of hindlimb incoordination and paresis. However, as the disease progresses, they may begin to experience dyspnea, vomiting, facial weakness, loss of reflexes, and an inability to stand.[7] Respiration initially may be tachypneic and forced, but as the neurotoxin moves through the body, respiration can become bradypneic or labored, especially on expiration. In the later stages of the disease, the patient may become cyanotic, a characteristic sign of severe hypoxemia.[8] As the paralysis ascends, the animal may be unable to sit, move its limbs, or lift its head.[5]

When the toxin progresses cranially, muscles in the pharynx, larynx, esophagus, and trachea begin to weaken, resulting in gagging, vomit-

Glossary

Atelectasis Total or partial lung collapse

Crepitation Crackling or grating sound or sensation

Cyanosis Bluish discoloration of the skin and mucous membranes resulting from deficient oxygenation of the blood

Decubitus Pressure-induced skin ulceration that can occur following a period of immobility; also called *bed sore*

Hypercapnia Increased concentration of carbon dioxide in the blood; also called *hypercarbia*

Hypoventilation Abnormally slow and shallow respiration resulting in an increased level of carbon dioxide in the blood

Hypoxemia Insufficient oxygenation of arterial blood

Hypoxia Deficiency in the amount of oxygen reaching the body tissues

Neurotoxin Chemical substance that attacks the nervous system

Ticks That Cause Paralysis

Six tick species are typically implicated in cases of tick paralysis in North America.[3]

Mat Pound, USDA Agricultural Research Service, Bugwood.org

Dermacentor andersoni
(the Rocky Mountain wood tick)

Source: CDC/James Gathany

Dermacentor variabilis
(the American dog tick)

Source: CDC/James Gathany

Amblyomma americanum
(the lone star tick)

Mat Pound, USDA Agricultural Research Service, Bugwood.org

Amblyomma maculatum
(the Gulf Coast tick)

Source: CDC/James Gathany

Ixodes scapularis
(the black-legged tick)

Source: CDC/James Gathany

Ixodes pacificus
(the western black-legged tick)

ing, drooling, and coughing.[7] Megaesophagus and decreased gag reflex may contribute to respiratory distress as a result of the pooling of saliva and the inability to swallow.[7] The patient also may experience facial paralysis.[7]

The patient's temperature is usually within the normal range in the early stages of the disease.[5] However, animals with low body mass may develop hypothermia, which can increase as a result of stress.[5] Conversely, affected animals can develop hyperthermia because of a reduced ability to pant.[9]

Unlike tick-borne diseases involving transmission of pathogens, which can proliferate and continue to cause clinical signs in the infected animal after the tick has been removed, tick paralysis occurs only while the tick is attached and transmitting the neurotoxin into the animal's bloodstream. Therefore, the patient's clinical signs usually resolve within 1 to 3 days after the tick has been removed.[5] If the animal does not receive treatment, progressive pulmonary dysfunction can occur, leading to hypoxia, hypercapnia, respiratory acidosis, and death.[5]

Prevalence

Cases of tick paralysis have been reported worldwide but are expected to occur more frequently in tick-endemic areas. Tick paralysis is most prevalent in the northwestern and southeastern regions of North America, as well as the eastern coast of Australia and southern Africa.[1] In North America, the greatest number of human cases of tick paralysis occur during April, May, and June when *Dermacentor* spp are most actively seeking hosts.[10] In such regions as the southern United States, tick activity can occur through-

out the year, increasing the potential for human and animal cases of tick paralysis and other tick-borne diseases. Although tick paralysis is relatively rare, it is among the top eight most common tick-borne diseases reported in humans in the United States.[11]

Diagnosis

When a patient presents with clinical signs of tick paralysis, the veterinary technician should obtain a thorough history. On physical examination, a large, blood-engorged female tick may be found, usually around the patient's head or neck, in the ear canals, around the genital area, or between the toes.[6,12]

No specific tests are available for diagnosing canine tick paralysis. Electroencephalography, complete blood cell count, serum chemistry profile, and urinalysis usually yield normal results.[5,7] However, lateral thoracic radiography may show the presence and degree of disease-associated complications, such as pulmonary edema and megaesophagus.[5] Arterial blood gas analysis may reveal hypoxemia, hypercapnia, and low pH[7]; in severely affected patients, arterial carbon dioxide pressure values may exceed 60 mm Hg (normal: 35–45), indicating the presence of respiratory acidosis.[8] In patients with pulmonary edema, crepitation, which indicates the presence of fluid in the airway, may be heard on auscultation.[8]

Diagnostic differentials include botulism, snakebite, coonhound paralysis, distal denervating disease, acute peripheral neuropathy, and generalized multifocal myelopathy.[5] In regions where ticks are endemic, tick paralysis should be considered when a patient presents with leg weakness, megaesophagus, unexplained vomiting, or acute left-sided congestive heart failure.[5]

Treatment

Tick removal is the most important element of treatment. Because the animal will continue to exhibit clinical signs until the tick is removed, thorough examination of the patient by the veterinarian is essential. Clipping long or matted hair is recommended, and using acaricidal dips helps ensure that all ticks are killed. However, the stress associated with clipping and bathing may increase hypothermia, especially in fractious or nervous patients.[5] Therefore, the veterinarian may prescribe a sedative and give the patient time to calm down before he or she performs these procedures.[5]

Severely affected animals may require hospitalization. Patients with respiratory acidosis should receive oxygen supplementation as supportive care, and patients with bradypneic, arrhythmic, or weak breathing patterns should be intubated and ventilated to correct hypoventilation.[8] Mechanical or manual ventilation also may be required.[5] The veterinarian may administer

propofol as a constant-rate infusion in patients requiring endotracheal intubation and ventilator support.[9]

During the first 48 hours of hospitalization, intravenous fluids typically are not required and may actually contribute to or worsen pulmonary edema.[5] However, the patient should be evaluated periodically for signs of dehydration.[9] If intravenous fluids are warranted, they should be administered at maintenance or submaintenance levels.[5] If the patient's packed cell volume is greater than 50%, colloids (e.g., hetastarch, pentastarch) administered slowly at submaintenance levels are recommended over crystalloids (e.g., Normosol-R, normal saline solution), as the latter can exacerbate pulmonary edema.[5] The veterinarian will avoid medication that interferes with neuromuscular transmission, such as tetracyclines, aminoglycosides, or procaine penicillin.[10] Furosemide can be given intravenously to reduce pulmonary edema.[5]

Food and water should be withheld until paralysis resolves.[13] The patient should be placed in sternal recumbency to facilitate respiration and reduce hypostatic congestion.[9] If the patient must lie in lateral recumbency, it should be rotated at least every 4 hours to prevent hypostatic pneumonia.[9] In addition, patients that are left immobile and in positional stasis for long periods are at risk for developing tissue necrosis and decubiti over bony protuberances.[8] Regional edema also may be associated with positional stasis because drainage of the lymphatic system is impeded. Accumulation of secretions and atelectasis in the lower regions of the lung may occur, along with contractures and stiffening of muscles and ligaments.[8]

Depending on the individual patient's needs, a variety of supportive therapies may be indicated, including the following:

- Application of eye protectants, such as ocular lubricants or artificial tears, to prevent eye dryness[5]
- Catheterization or manual bladder expression if the patient is unable to empty its bladder[9]
- Suctioning of the pharynx, larynx, and proximal esophagus to minimize respiratory distress associated with esophageal dysfunction and the pooling of saliva[5] (in animals with severe dyspnea, general anesthesia may be needed to allow oxygen administration, esophageal suction, and pulmonary drainage)[5]

Prevention

The technician should educate clients about the importance of preventing tick paralysis and other tick-borne diseases, especially in endemic areas. Owners should know the proper way to remove ticks from their pet (see box to the left). Regularly examining the pet's haircoat for ticks is essential. Clients should be instructed on the proper use of veterinarian-prescribed acaricides (Table 1) to help prevent their pet from becoming infested.

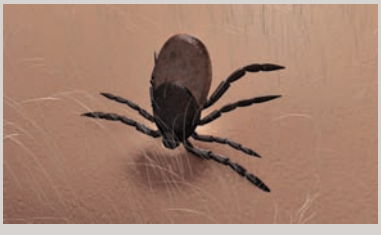
Clients also should be told about environmental controls, including keeping grass cut short and trimming bushes. To help control ticks, acaricides can be sprayed in the yard and along fences. It is important that these products be used according to the instructions on the label. Acaricides for use indoors also are available, but application by a licensed exterminator is recommended.[3] These products should be allowed to dry before animals or humans return to the area.[3]

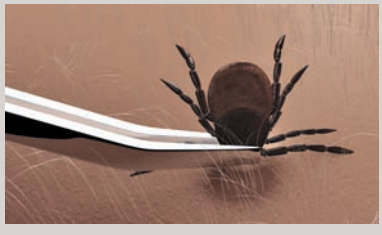
Role of the Technician

When a dog presents to the veterinary clinic with clinical signs of tick paralysis, the technician is responsible for obtaining an accurate and complete patient history, noting clinical signs and progression of paralysis, collecting

Tips for Tick Removal[3,a]

1. Using tweezers or forceps, grasp the tick as close to the pet's skin as possible.





2. Remove the tick using slow, steady pressure.
3. Do not crush or twist the tick or yank it from the pet's skin because the tick's mouthparts may become detached and remain in the skin, potentially causing injury or infection.
4. Do not use a lit match or other products, such as petroleum jelly or nail polish, to remove the tick.

[a]US Army Center for Health Promotion and Preventive Medicine. *Tick Removal.* Accessed July 2008 at chppm-www.apgea.army.mil/ento/TickEduc/Tickremoval.pdf.

specimens for analysis, and providing supportive care. The technician can play a major role in reducing the incidence of this disease by educating clients about environmental controls and preventive measures that will help the pet — and the owner — remain tick-free. VT

References

1. Powell C. *WSU Veterinary College Warns Llama and Alpaca Owners of Tick Paralysis.* Accessed July 2008 at www.wsunews.wsu.edu/pages/publications.asp?Action=Detail&PublicationID=11136.
2. US Army Center for Health Promotion and Preventive Medicine, Entomological Sciences Program. *Tick Paralysis.* Accessed July 2008 at chppm-www.apgea.army.mil/ento/facts/TickParalysis-JusttheFactsJan05.pdf.
3. Companion Animal Parasite Council. *Ectoparasites: Ticks Guidelines.* Accessed July 2008 at www.capcvet.org/?p=Guidelines_Ticks&h=0&s=0.
4. Doube BM. Cattle and the paralysis tick *Ixodes holocyclus. Aust Vet J* 1975;51(11):511-515.
5. Tick paralysis. In: Kahn CM, ed. *The Merck Veterinary Manual.* 9th ed. Whitehouse Station, N.J.: Merck & Company; 2005:1073-1076.
6. Samuels MA. *Manual of Neurologic Therapeutics.* Philadelphia: Lippincott Williams & Wilkins; 2004:272-273.
7. Cuddon P. Tick bite paralysis. In: Tilley LP, Smith FWK, eds. *The 5-Minute Veterinary Consult: Canine and Feline.* Baltimore: Williams & Wilkins; 1997:1104-1105.
8. Mellema M, Haskins S. Supportive care of the poisoned patient. In: Peterson ME, Talcott PA, eds. *Small Animal Toxicology.* 2nd ed. St. Louis: Elsevier Saunders; 2006:73-75, 96-97, 121-122.
9. Fischer N. *Tick Poisoning in Animals — In Depth.* Accessed July 2008 at www.tickalert.org.au/paraldet.htm.
10. Dworkin MS, Shoemaker PC, Anderson DE. Tick paralysis: 33 human cases in Washington State, 1946–1996. *Clin Infect Dis* 1999;29(6):1435-1439.
11. Spach DH, Liles WC, Campbell GL, et al. Tick-borne diseases in the United States. *N Engl J Med* 1993;329(13):936–947.
12. Wright RE, Barker RW. *Common Ticks of Oklahoma and Tick-Borne Diseases.* Accessed July 2008 at www.okrangelandswest.okstate.edu/pdfFiles/OSUextPubs/F-7001.pdf.
13. Braund KG. *Clinical Syndromes in Veterinary Neurology.* St. Louis: Mosby; 1994:273-274.

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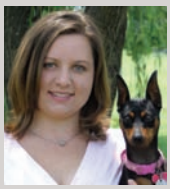

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Lisa is a veterinary technician at St. Francis Animal Hospital in Augusta, Ga. In addition to her full-time job, Lisa is working toward a bachelor's degree in biology at Augusta State University and hopes to enter veterinary school.

Arterial Blood Pressure Monitoring

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MEASURING ARTERIAL BLOOD PRESSURE, which has been an integral part of human medicine for many years, can be critical to the outcome of equine patients. During physical examination, the cardiovascular status of a patient typically is evaluated by checking its oral mucous membranes, heart rate, and pulse. In some patients, it also would be informative to measure the blood pressure, especially in horses undergoing general anesthesia and in critically ill patients, including critically ill neonatal foals, patients in septic shock, or patients with colic, laminitis, or epistaxis.[1] (See the box below.) Other conditions that may require arterial blood pressure monitoring include acute hemorrhage, colitis, cardiac failure, and anaphylactic reactions.

Understanding the Blood Pressure Components

Arterial blood pressure, which is the product of cardiac output, blood volume, and blood vessel resistance, is the force with which blood flows through the arteries to the periphery of the body. These thick-walled muscular vessels transport blood under high pressure to deliver such vital components as oxygen and nutrients to peripheral tissues and organs.

Three parameters are used to assess arterial blood pressure: systolic, diastolic, and mean arterial pressures. The systolic pressure is the maximum pressure generated by the left ventricle during contraction, and diastolic pressure is the minimum pressure achieved during relaxation. Mean arterial pressure (MAP) is the major determinant of perfusion pressure to most organs of the body and should be closely monitored during anesthesia.[1] Horses need to maintain blood pressure within a range that is adequate for blood delivery, called perfusion, to peripheral tissues, ensuring adequate delivery of oxygen and nutrients and removal of metabolic waste products. Under normal conditions, horses maintain constant arterial blood pressure to perfuse their whole body. However, in certain disease states and when undergoing general anesthesia, levels often are altered.[2]

The MAP required to perfuse organs and tissues is approximately 60 mm Hg. In adult horses, normal systolic pressure is 110 to 160 mm Hg and normal diastolic pressure is 90 to 110 mm Hg, creating a MAP of 110 to 120 mm Hg. Under anesthesia, the MAP in a horse should remain above 60 mm Hg.

Normal values for neonatal foals (<1 month old) are much lower than those for adult horses and vary by patient, depending on age and cardiovascular status. It is not uncommon for a neonatal foal to have a systolic pressure of 80 to 100 mm Hg, diastolic pressure of 40 to 60 mm Hg, and MAP of 50 to 60 mm Hg.

Why Arterial Blood Pressure Monitoring Is Important

- Allows better assessment of the patient's condition
- Provides the ability to monitor the patient's response to therapy
- Provides a database to assist in patient care
- Determines the effect of anesthesia on the patient's cardiovascular system
- Allows educated assumptions relating to cardiac performance, myocardial oxygen consumption, vascular tone, and organ and tissue perfusion

Blood Pressure Measurement: Indirect Method

Step 1. Select the correctly sized blood pressure cuff, such as an adult cuff for an adult patient.

Step 2. Wrap the cuff around the foal's or adult horse's tail, making sure that it is centered over the coccygeal artery.

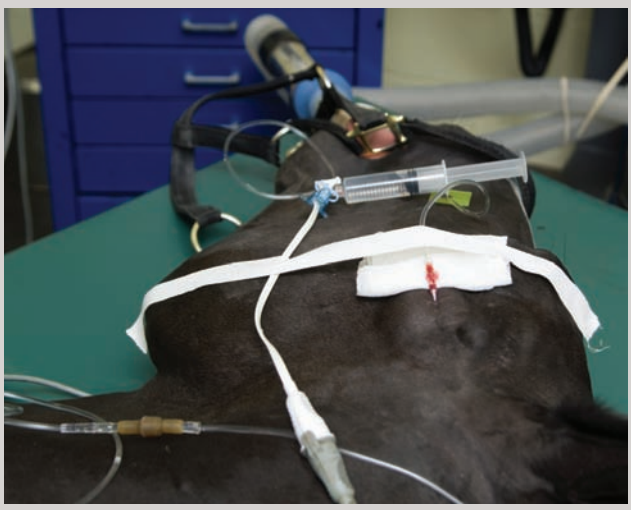
Courtesy of Mid-Atlantic Equine Medical Center; A. G. Photography

Step 3. Monitor the blood pressure readings.

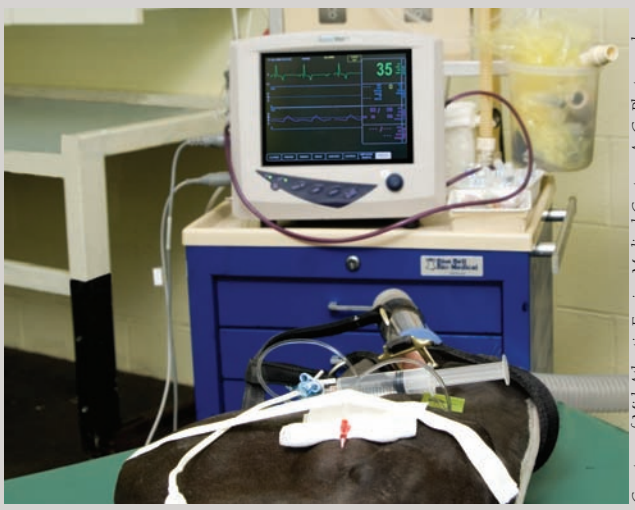
Blood Pressure Measurement: Direct Method

Step 1. Clip and sterile prep the area over the artery. Inset: Catheters and extension set for arterial catheter placement.

Step 2. After putting on sterile gloves and removing the catheter from the package, insert the catheter into the transverse facial artery until you see bright-red blood in the hub of the catheter.



Step 3. Attach the extension set to the catheter and tape it in place.



Step 4. Monitor the blood pressure readings.

Courtesy of Mid-Atlantic Equine Medical Center; A. G. Photography

Physiologic factors that can affect the MAP include ventricular failure, myocardial depression (as occurs with anesthesia), hypovolemia, positive-pressure ventilation, and the presence of endotoxins.

Changes in blood pressure include hypertension (high blood pressure) and hypotension (low blood pressure). Hypertension is not common in horses, but hypotension can develop when a horse is undergoing general anesthesia and in certain disease states, such as colitis, septic shock, or hemorrhagic shock. Hypotension also can result from decreased fluid volume in the blood vessels, decreased blood vessel tone, or "leaky" blood vessels. If the patient develops hypotension (MAP <60 mm Hg), there will be less blood volume delivered to peripheral tissues and decreasing oxygen and nutrient delivery. The average arterial pressure level at which blood ceases to flow is about 20 mm Hg.[2]

Blood Pressure Measurement

There are two methods of assessing blood pressure in horses: direct and indirect. The indirect method of monitoring arterial blood

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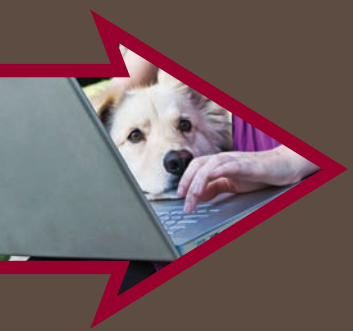

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Some Precautions to Address

When monitoring blood pressure in equine patients, the following information should be considered:

- The cuff must be the correct size and placed properly over the artery to obtain accurate blood pressure readings using the indirect method.
- An arterial pulse can be used to count beats of the pulse and compare with the heart rate to help detect pulse deficits.
- You cannot directly measure blood pressure by palpating the pulse in a peripheral artery.
- If you have difficulty catheterizing an artery, it may indicate low blood pressure (hypotension — most commonly the result of hypovolemia).
- If both the systolic and diastolic pressures are low, the animal is most likely hypotensive.
- Alterations in blood pressure are a good index of depth of anesthesia — blood pressure falls as the depth of anesthesia deepens.

pressure is noninvasive and more commonly used in neonatal foals or adult horses that are not anesthetized. This method is not as accurate as the direct method and provides a trend rather than an absolute determination of the blood pressure level.

The indirect method (shown on page 479) can be done in one of two ways.[2,3] The first involves an oscillometric monitor and placement of an air-filled cuff around a peripheral limb or at the base of the tail. Accuracy depends on using the correctly sized cuff as well as correctly placing the cuff over the artery. This type of indirect method can be inaccurate when the heart rate is slower than 25 beats/min and the blood pressure is low — less than 60 mm Hg. It also is less accurate in standing adult horses.

The second way to measure indirect blood pressure involves the use of an ultrasonic Doppler apparatus (not shown here), which consists of a Doppler crystal and an aneroid gauge within a cuff that is placed over a peripheral artery.[2,4–6] The cuff is inflated until a pulse is heard, which measures the systolic blood pressure on the gauge. This can be accurate to a blood pressure reading of 40 mm Hg.[7]

Direct measurement is more accurate and is considered the gold standard. It is advantageous when monitoring patients that are undergoing general anesthesia or critically ill patients but requires catheterization of an artery.

The direct method (shown on page 480) is invasive and requires catheterization of a peripheral artery but is more accurate and provides a continuous reading.[2,4] However, there is risk for potential complications, including infection and hematomas. With this method, a 17- to 21-gauge catheter is placed into a metatarsal, metacarpal, facial, or transverse facial artery and is connected to a monitor or an aneroid manometer through extension sets and a three-way stopcock. When using a manometer, a pressure bag or syringe of heparinized saline is used to flush the lines, open the arterial line to the manometer, and

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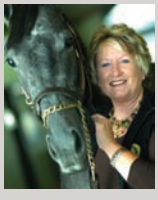

Deborah is most known for her involvement in forming the American Association of Equine Veterinary Technicians, and she continues to serve as executive director. For the past 7 years, she has done consulting work for equine practices and the industry. In addition, Deborah is a member of the Association of Veterinary Practice Managers and Consultants, Texas Association of Registered Veterinary Technicians, Texas Veterinary Medical Association (Hon), and United States Equestrian Foundation.

create pressurization so that the mean pressure is read between the bounce of the needle.

Regardless of which method you use, there are precautions that should be addressed (see box on page 482).

Role of the Technician

Monitoring arterial blood pressure in the equine patient is an important tool in your role in the overall assessment of a patient's condition. Alterations in arterial blood pressure can provide a warning of an abrupt change in the patient's condition.

It is a useful tool to monitor critically ill equine patients, especially critically ill neonatal foals and horses undergoing general anesthesia. Because they cannot verbally tell us what is going on, measuring blood pressure aids our understanding of their condition and provides an invaluable tool in monitoring their response to treatment. VT

References

1. Gay CC, McCarthy J, McCarthy M, et al. The value of arterial blood pressure measurement in assessing the prognosis in equine colic. *Equine Vet J* 1977;9:202-204.
2. Smarick S. Direct and indirect blood pressure: indications and methods. *Proc Int Vet Emerg Crit Care Symp*:2007.
3. Rose RJ, Rossdale PD. Techniques and clinical applications of arterial blood collection in the horse. *Equine Vet J* 1981;13:70-73.
4. Giguere S, Knowles HA Jr, Valverde A, et al. Accuracy of indirect measurement of blood pressure in neonatal foals. *J Vet Intern Med* 2005;19(4):571-576.
5. Gay CC, McCarthy J, McCarthy M, et al. A method for indirect measurement of arterial blood pressure in the horse. *Aust Vet J* 1977;53:163-166.
6. Latshaw H, Fessler JF, Whistler SJ, Geddes LA. Indirect blood pressure measurement of mean blood pressure in the normotensive and hypotensive horse. *Equine Vet J* 1979;11:191-194.
7. Nout YS, Corley KT, Donaldson LL, Furr MO. Indirect oscillometric and direct blood pressure measurements in anesthetized and conscious neonatal foals. *J Vet Emerg Crit Care* 2002;12(2):75-80.

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CE Article #1 Canine Tick Paralysis

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1. _____ is a clinical sign characteristic of patients presenting with tick paralysis.
   a. Inappetence
   b. Diarrhea
   c. Hindlimb incoordination
   d. Pneumothorax

2. High arterial carbon dioxide pressure levels are associated with
   a. pulmonary edema.
   b. respiratory alkalosis.
   c. metabolic acidosis.
   d. respiratory acidosis.

3. Mortality rates of up to _____ have been reported in domestic animals when the neurotoxin-transmitting tick is not removed.
   a. 5%
   b. 7%
   c. 10%
   d. 15%

4. Which of the following practices is recommended during tick removal?
   a. Applying petroleum jelly to the tick
   b. Grasping the tick with tweezers or forceps
   c. Yanking the tick off the skin
   d. Using a match to make the tick back up

5. In the later stages of tick paralysis, the patient may become cyanotic, a characteristic sign of severe
   a. hyperthermia.
   b. respiratory acidosis.
   c. hypoxemia.
   d. edema.

6. Tick paralysis is caused by transmission of
   a. a salivary neurotoxin.
   b. an allergen.
   c. a bacterium.
   d. a cyanotoxin.

7. The most important aspect of tick paralysis treatment is
   a. administering IV Normosol-R.
   b. removing the tick.
   c. applying acaricides.
   d. bathing the patient.

8. After tick attachment, clinical signs of tick paralysis usually begin within
   a. 1 to 3 hours.
   b. 6 to 9 days.
   c. 1 to 2 weeks.
   d. 6 to 9 months.

9. In animals with tick paralysis, _____ secretion is reduced.
   a. norepinephrine
   b. dopamine
   c. acetylcholine
   d. serotonin

10. Drugs that are contraindicated for use in patients with tick paralysis include
    a. tetracyclines.
    b. aminoglycosides.
    c. procaine penicillin.
    d. all of the above