Vascular Encephalopathies in Dogs: Diagnosis, Treatment, and Prognosis*

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\textbf{ABSTRACT:} The use of contrast studies and advanced magnetic resonance imaging techniques allows characterization of type and time course of ischemia or hemorrhage. Many systemic diseases may predispose dogs to brain infarction; therefore, diagnosis of a suspect vascular disorder should prompt thorough screening for underlying disease(s). There is no known preventative or treatment for brain infarction in dogs. Overall, prognosis of ischemic brain injury is good, with outcome depending on underlying cause and severity of the vascular event.

Ischemic encephalopathies are characterized by acute, focal, and nonprogressive neurologic signs. Definitive diagnosis is difficult and requires critical evaluation of the minimum database followed by imaging with computed tomography (CT) or magnetic resonance imaging (MRI). There is no specific treatment for brain infarction in dogs. Supportive care with anticipation and treatment of complications is essential for a positive patient outcome. Recovery can be expected in most cases over a period of days or weeks. Long-term survival and risk of reinfarction depend on the underlying cause of the ischemic insult.

\textbf{DIAGNOSIS}  
Initial evaluation of patients with suspected ischemic encephalopathy should begin with the differential diagnosis, including trauma as well as metabolic (i.e., renal, hepatic, hyper- or hypo-coagulopathy, hypoglycemia), neoplastic, inflammatory, and toxic encephalopathies. Characterization of ischemic or hemorrhagic stroke should be attempted but is often impossible in animals before imaging studies. In humans, temporal relationships to the stroke event can help distinguish among different types of stroke.\textsuperscript{1} Embolic strokes tend to have a peracute onset with no premonitory signs, whereas thrombotic

\textsuperscript{*A companion article on incidence, risk factors, pathophysiology, and clinical signs appears on p. 196.}

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stroke can be preceded by transient ischemic attacks. Recognition of risk factors may help minimize additional events if the cause of the vascular event is ascertained. Initial diagnostic assessment should include systolic blood pressure (BP) measurement, a complete blood count, a serum biochemistry profile, and urinalysis. Coagulation tests such as D-dimer, prothrombin time, and partial thromboplastin time should also be included in the initial laboratory evaluation of patients suspected of having vascular disease of the brain. D-Dimer, a sensitive indicator of the activation of thrombin and plasmin, may be elevated following thromboembolism. This test is more specific than fibrin degradation products for thrombus formation and breakdown, but results may be elevated in patients with other diseases or conditions without thromboembolism, such as immune-mediated hemolytic anemia, neoplasia, liver or renal disease, congestive heart failure, and following surgical procedures.

D-Dimer has not been evaluated for use in diagnosing ischemic or hemorrhagic brain infarction in dogs but may prove useful in providing additional information before imaging in at-risk patients. Second-wave diagnostics should include thoracic radiography, abdominal ultrasonography, and initial (and possibly serial) CT or MRI scans. Additional diagnostics that may be considered include cerebrospinal fluid (CSF) analysis, echocardiography, measurement of antithrombin III levels, urine protein:creatinine ratio, and proteins induced by vitamin K antagonism (PIVKA) tests. The gold standard in diagnosing cerebrovascular insult related to focal ischemia in humans is cerebral angiography, which is rarely conducted in veterinary medicine.

Interpreting MRI and CT findings of cerebrovascular lesions is complex and depends highly on the type (i.e., focal or global ischemic, hemorrhagic) of infarction and time from vascular insult to imaging. MRI is now more widely available and very sensitive in detecting and characterizing ischemic and hemorrhagic infarction. CT is also used in the diagnosis of ischemic encephalopathies but is inferior to MRI in detecting ischemic infarction because of the following:

- Beam-hardening artifacts, which limit the ability of CT to effectively image the posterior fossa and brain stem
- Inferior contrast display
- Inability to provide detailed multiplanar views

MRI can typically detect ischemic infarction within minutes via diffusion weighting and perfusion studies.

Clinical studies in humans have demonstrated a sensitivity of only 20% within the first 6 hours of ischemic infarction and 80% within the first 24 hours using CT. However, CT is very sensitive in detecting acute hemorrhagic infarction because of the relationship between erythrocyte constituents and attenuation of x-rays with acute hemorrhage, resulting in hyperdense lesions. Ischemic and hemorrhagic stroke can be characterized as peracute, acute, subacute, and chronic. Peracute infarction can be detected with imaging 3 to 6 hours following an ischemic or hemorrhagic event. Acute infarction can be detected with imaging 6 to 24 hours following vascular insult. Subacute infarction occurs 24 hours to 6 weeks following vascular insult and can be subdivided into early (i.e., 24 hours to 1 week) and late (i.e., 1 to 6 weeks). Chronic infarction precedes imaging for longer than 6 weeks.

Brain infarction can also be categorized by distribution of loss of blood flow. Two general distributions are described and include lacunar (small) and territorial (large) infarctions. Lacunar infarctions are considered interruptions of blood flow to the small, deep penetrating arteries of the brain. In humans, these generally involve the arteries supplying the basal nuclei, thalamus, internal capsule, and brain stem. Territorial infarctions disturb blood flow to more expansive areas of the brain and, therefore, are the result of occlusion of large blood vessels such as the rostral, middle, and caudal cerebral
arteries. In veterinary patients, telencephalic and cerebellar infarctions are typically territorial and involve the middle and rostral cerebral arteries and rostral, middle, and caudal cerebellar arteries, respectively (Figure 1). In contrast, thalamic or midbrain infarctions are lacunar and involve the small striate arteries and perforating arteries of the rostral brain stem.

The MRI findings in ischemic encephalopathy (Table 1) result from water accumulation due to cytotoxic and vasogenic edema. In peracute and early subacute infarctions, the resultant cytotoxic or intracellular edema may be evident by 1 hour after infarction, but detection with T1- and T2-weighted images may be difficult. Magnetic resonance angiography (MRA) is a specialized technique using MRI that allows direct visualization of blood flow; MRA may prove useful in veterinary medicine in the future. Currently, MRA is more useful in humans because the orientation of the circle of Willis relative to surrounding structures of the head is better defined in humans than in dogs. Because of high variability in head shape and structure among various dog breeds, the relationship of the circle of Willis to surrounding structures of the head is inconsistent, limiting standardization. Therefore, when conducting MRA in dogs, a high degree of technical expertise and repeat scanning are essential to place the saturation bands necessary to image the vessels of interest. Diffusion-weighted MRI is a specialized technique that allows visualization of infarctions with increased sensitivity. Diffusion weighting is based on decreased diffusion of water from the intracellular space (i.e., cytotoxic edema formation) in brain tissue. Decreased diffusion of water molecules is detected as hyperintense lesions during diffusion weighting. Gradient echo sequencing is an advanced application of MRI that can also be used to diagnose stroke in dogs. The details of gradient echo sequencing are beyond the scope of this article and have been reviewed elsewhere.

Vasogenic (extracellular) edema formation is evident with MRI typically within 4 to 6 hours, but findings may be subtle within the first 24 hours after infarction. Vasogenic edema is detected as hyperintense lesions on T2-weighted scans and hypointense lesions on T1-weighted scans. A specialized T2 scanning modality (i.e., fluid-attenuated inversion recovery [FLAIR] imaging) is used to visualize vasogenic edema. With FLAIR imaging, CSF can be differentiated from edema. Edema appears hyperintense and CSF hypointense (Figure 2). Use of these sequences may prove helpful in detecting infarctions early (within 24 hours). T2-weighted FLAIR images are used by the authors for this purpose.

### Table 1. MRI Findings Associated with Focal Ischemic Infarction

<table>
<thead>
<tr>
<th>Stage</th>
<th>Time to Imaging</th>
<th>T2-Weighted MRI Findings</th>
<th>T1-Weighted MRI Findings</th>
<th>FLAIR Findings</th>
<th>Contrast Enhancement?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peracute</td>
<td>3–6 hr</td>
<td>Hyperintense</td>
<td>Hypointense</td>
<td>Hyperintense</td>
<td>No</td>
</tr>
<tr>
<td>Acute</td>
<td>6–24 hr</td>
<td>Hyperintense</td>
<td>Hypointense</td>
<td>Hyperintense</td>
<td>No</td>
</tr>
<tr>
<td>Subacute</td>
<td></td>
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<tr>
<td>Chronic</td>
<td>&gt;6 wk</td>
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<td>Variable</td>
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FLAIR = fluid-attenuated (T2-weighted) inversion recovery.
One to 8 weeks following infarction, enhancement of brain parenchyma with contrast agents using MRI (gadolinium diethylenetriamine penta-acetic acid [DTPA]) may occur because of disruption of the blood–brain barrier. A thin rim of contrast enhancement typically occurs at the periphery of the infarcted tissue. MRI findings in chronic cerebral infarction include increased signal intensity on T2-weighted images, decreased signal intensity on T1-weighted images, and enlargement of sulci and/or ventricles, which represent focal atrophy of infarcted regions. Because of restoration of the disrupted blood–brain barrier, contrast enhancement is rare more than 8 weeks after infarction.

Differentiation of thrombotic versus embolic infarction is based on distribution and number of ischemic territories and presence or absence of hemorrhage. Both embolic and thrombotic infarction are typically wedge shaped with sharp demarcation that extends to the cortical surface. In humans, thrombotic infarctions are often single and involve one vascular territory, whereas embolic infarctions are often multiple. In contrast, most suspected embolic infarctions in veterinary patients are single. In humans, hemorrhagic infarction is a common sequela to embolic infarction due to lysis or distal movement of the embolus, which can occur 6 hours to 2 weeks following initial obstruction of blood flow. This reestablishment of blood flow is not controlled by cerebral autoregulation. Hemorrhage following embolic infarction is considered uncommon in dogs. Thrombotic infarctions are typically permanent, but partial lysis with secondary hemorrhage can occasionally occur.

The MRI appearance of global ischemia is characterized by increased intensity on T2-weighted images in the watershed zones of major intracerebral arteries indicative of diffuse cerebral edema (Figure 3). These areas of the brain comprise tissue with the lowest cerebral perfusion pressure. Lesions most commonly involve white matter surrounding the ventricles and vulnerable regions such as the cerebral cortex, basal nuclei/caudate nucleus, thalamus, cerebellum, and hippocampus. Transcranial Doppler ultrasonography with characterization of abnormal flow patterns in the basilar artery has been shown to accurately diagnose brain damage and predict neurologic outcome following global cerebral ischemia in dogs.

CT is widely used in veterinary medicine to assess neurologic dysfunction. Temporal changes in CT findings following embolic or thrombotic infarction are similar to those of MRI but differ based on time from infarct to earliest detection (i.e., 3 to 6 hours). Findings may be inconsistent during the first 24 to 48 hours and are influenced by size and degree of ischemic insult as well as image resolution. Typical early CT findings include decreased beam attenuation, mottling, and poor margination. Decreased attenuation is the result of...
previously described cytotoxic and vasogenic edema formation.\textsuperscript{4,5,9} Mass effect is a result of focal edema formation, and maximal findings can be seen within 3 to 5 days of infarction.\textsuperscript{4} Enhancement with iodinated contrast media may be evident by 48 hours to 1 week after infarction and peaks at 1 to 3 weeks.\textsuperscript{5,8,9} As in CT, contrast enhancement during MRI is considered diagnostic for breakdown of the blood–brain barrier but is rarely seen before 1 week or after 8 weeks of infarction.\textsuperscript{9} Because embolic and thrombotic infarctions are typically hypointense on CT scans, contrast administration can result in isodense lesions, resulting in false-negative results.\textsuperscript{4} Comparing plain and contrast-enhanced CT study results is therefore important to accurately assess cerebral infarction.\textsuperscript{4}

When assessing hemorrhagic infarction or hemorrhage associated with focal ischemia, CT and MRI are useful (Table 2). MRI findings are temporally related based on imaging characteristics of the breakdown products of hemoglobin. Peracute hemorrhage with oxyhemoglobin appears isointense with occasional, slightly increased intensity on T2-weighted images versus CT, which is very sensitive in detecting peracute hemorrhage.\textsuperscript{4} If a patient’s hematocrit is normal, CT results in increased beam attenuation immediately following hemorrhage.\textsuperscript{4} Beam attenuation is linearly related to hematocrit.\textsuperscript{4} Therefore, in peracute hemorrhagic infarction, CT is preferred over MRI for diagnosis. As hemoglobin molecules age, beam attenuation decreases and the hemorrhagic area is not detectable with CT.\textsuperscript{4} MRI findings in acute hemorrhagic infarction include an initial low-intensity signal on T2-weighted images and an isointense signal on T1-weighted images resulting from the presence of deoxyhemoglobin.\textsuperscript{4,8} Because of vasogenic edema formation, there is typically high signal intensity surrounding areas of hemorrhage on T2-weighted images.\textsuperscript{7} In the early subacute stage, high signal intensity on T1-weighted images and low signal intensity on T2-weighted images are due to further oxidation of hemoglobin molecules with formation of intracellular methemoglobin.\textsuperscript{4,8} In late subacute hemorrhage, T1- and T2-weighted images are hyperintense because the extracellular methemoglobin concentration increases.\textsuperscript{8} Hyperintensity tends to move centrally toward the infarction because collateral circulation provides oxygen for hemoglobin oxidation near the periphery of the infarction.\textsuperscript{4} Chronic infarction appears hypointense on T1- and T2-weighted images because of the presence of hemosiderin and ferritin, which are deposited around the infarction by specialized brain tissue macrophages or Gitter cells.\textsuperscript{4,8}

CSF analysis can be a valuable aid in some cases of ischemic encephalopathy. Electroencephalography can also be used, but findings from it are often variable and nonspecific.\textsuperscript{19} Findings from CSF analysis may be influenced by underlying pathology and causes of infarction.
or hemorrhage. Culture and analysis of CSF may be beneficial when septic embolism is suspected. In cases of neoplasia, such as lymphoma, neoplastic cells may be visualized via cytologic assessment. In acute hemorrhagic, embolic, or thrombotic infarction, CSF findings may be normal or nonspecific and may include mononuclear or neutrophilic pleocytosis, elevated protein levels, and xanthochromia. Because of the risk of herniation through the foramen magnum, when CSF collection is being considered, caution should be exercised if increased intracranial pressure (ICP) is suspected.

**TREATMENT**

Treatment of vascular encephalopathy in animals is aimed at providing adequate supportive care, recognizing and treating neurologic and nonneurologic complications, maintaining adequate tissue oxygenation, and managing seizures and elevated ICP. Nonneurologic complications such as joint stiffness, contractures, decubital ulcers, pneumonia, urinary tract infection, malnutrition, and pulmonary embolism are common in humans and are potential complications in animals following ischemic encephalopathy. Because most animals with vascular encephalopathy are severely debilitated for prolonged periods, nursing management should be detailed to include proper head and neck positioning, physical therapy with range-of-motion exercises, adequate bedding with frequent turning, and prevention of decubital ulcers and nosocomial infection with strict adherence to patient cleanliness and catheter care. Enteral or (rarely) parenteral nutrition may be required because some animals may be unwilling or unable to eat for several days to weeks. Enteral nutritional support can be provided via nasogastric, pharyngostomy, esophagostomy, or gastrostomy tubes. Parenteral nutrition should be provided with caution in patients recovering from vascular accidents because they are often demented and can dislodge catheters and parenteral nutrition administration sets, possibly leading to sepsis and/or phlebitis.

Following a known ischemic or hemorrhagic vascular event, multiple neurologic and physical parameters must be monitored for recognition of neurologic complications, including level of consciousness, involuntary muscle movement, posture, heart rate, respiratory rate and rhythm, and cranial nerves. Worsening of any neurologic parameter or presence of seizures may indicate brain edema with increased ICP, the presence of (or worsening) intracranial hemorrhage, progression to mass effect, movement of existing emboli, or the presence of additional emboli or thromboses. Maintenance of normal tissue oxygenation, pulmonary ventilation, core body temperature, and intravascular volume is important to prevent these complications. Increased ICP can be caused by hypercapnia, hypoxia, overhydration, hypovolemia, fever, and impaired cerebral venous

<table>
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<tr>
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outflow from venous compression due to jugular venous catheter bandages or flexion of the head and neck.\textsuperscript{1} Obtunded patients suspected of having increased ICP secondary to hypercapnia should be treated with hyperventilation with a target partial pressure of carbon dioxide of less than 35 mm Hg. Monitoring partial pressure of carbon dioxide is essential because overzealous ventilation can lead to decreased venous return and cardiac output, possibly resulting in cerebral hypoxia and increased CSF lactate concentration.\textsuperscript{24} Mannitol therapy (0.5 to 1 g/kg IV over 10 to 20 minutes up to q8h) may be initiated to treat suspected elevated ICP.\textsuperscript{26} Oxygen supplementation via mask, nasal catheter, oxygen hood, or oxygen cage should be considered in patients with hypoxia assessed via pulse oximetry or arterial blood gas analysis. Causes of hypoxia in stroke patients include hypoventilation, aspiration pneumonia, or lung atelectasis.\textsuperscript{24} Additional measures of preventing and treating increased ICP may include implementing balanced fluid therapy with crystalloids and colloids, recognizing and treating seizures, monitoring central venous pressure, maintaining normal body temperature, extending the head and neck to increase perfusion and aid ventilation, and elevating the head 30° above horizontal.\textsuperscript{1,24} Treatment of hypovolemia should be approached with caution in patients with suspected cerebrovascular disease because high-dose crystalloid infusion may result in cerebral edema formation.\textsuperscript{4} Treatment with colloids such as dextran-70 or hetastarch is recommended.\textsuperscript{4} Seizure control with nonsedating anticonvulsant medication (e.g., felbamate, gabapentin, zonisamide, levetiracetam)\textsuperscript{6,27} should be considered. Use of sedating anticonvulsants such as diazepam, potassium bromide, and phenobarbital can result in inaccurate assessment of neurologic status during the recovery period. Prevention of hyperglycemia and maintenance of normal blood glucose may result in improved neurologic outcome.\textsuperscript{26} The proposed mechanism of injury during hyperglycemia is related to increased tissue lactic acid concentration. Experimental studies in diabetic dogs revealed that hyperglycemia 24 hours before a cerebral ischemic event resulted in significantly more severe neuropathology and decreased survival compared with dogs that were normoglycemic before ischemia.\textsuperscript{29} Hypertension is common in humans with cerebrovascular disease\textsuperscript{24} and has occasionally been documented in veterinary stroke patients (in which the causes of hypertension have been various).\textsuperscript{29} Stroke may be initiated in hypertensive patients as a result of vascular spasm. In humans and animals with ischemic encephalopathy, normal chemical autoregulation of cerebral blood flow is lost in injured brain regions, resulting in inability of the cerebral vasculature to adapt to changes in systemic BP, hypoxia, and hypercapnia.\textsuperscript{4,24} Because cerebral perfusion depends on ICP and mean arterial BP,\textsuperscript{26} it is important to maintain optimal systemic BP to prevent hemorrhage or brain hypoperfusion. Hypertension in stroke patients may be related to the underlying cause of the stroke event, including primary (essential) hypertension or underlying diseases such as renal failure, hyperadrenocorticism, or pheochromocytoma. Additional diagnostic differentials for hypertension may include stress following a stroke event, pain, or a result of the normal compensatory physiologic response to brain hypoxia or elevated ICP.\textsuperscript{24} In hypertensive patients, treatment should be initiated only if systolic BP persistently remains above approximately 170 to 180 mm Hg after addressing the aforementioned causes.\textsuperscript{24} Acute treatment of hypertension should be considered carefully because cerebral autoregulation is lost and perfusion in injured regions highly depends on systemic BP.\textsuperscript{4,15} Treatment with oral antihypertensives is preferred,\textsuperscript{24} but parenteral medications such as nitroprusside, intravenous \(\beta\)-blockers, calcium-channel blockers, or diuretics may be necessary in patients that cannot tolerate oral medications.\textsuperscript{3} Treatment of hypertension with thiazide diuretics alone or in combination with additional hypertensive medications has been shown to decrease the risk of ischemic stroke in humans.\textsuperscript{30} In our experience, most patients have been able to tolerate oral medications such
as enalapril (0.25 to 0.5 mg/kg bid), benazepril (0.25 to 0.5 mg/kg/day), and amlodipine (0.1 mg/kg/day) for BP control.

A widely investigated area of stroke therapy in humans is neuroprotection via \(N\)-methyl-\(D\)-aspartate (NMDA)–receptor antagonism, free radical scavengers, iron-chelating agents, and reduction of calcium-mediated injury.\(^{31-38}\) In addition, thrombolytic and antiplatelet therapies are currently being widely investigated. Antioxidant and antiexcitotoxic therapies are experimental at this time. In animal models, treatment of incomplete cerebral ischemia with neuroprotective antioxidants (e.g., deferoxamine and the 21-aminosteroid tirilazad) has shown mixed results, but some models have resulted in incomplete reduction of early metabolic failure and attenuation of cerebral edema.\(^{31,36,38,39}\)

Combination therapy with antioxidants and experimental antiexcitotoxic agents (i.e., selective NMDA-receptor antagonists) has resulted in improved cerebral blood flow and energy recovery.\(^{32,39}\) Neuroprotection with calcium-channel blockers has been beneficial in humans if administered within 6 hours of the initial ischemic event.\(^1\) The proposed benefit is due to blunting of calcium-mediated neuronal injury.\(^{40}\) Treatment with the thrombolytic agent tissue plasminogen activator is not widely used in humans and carries a significantly increased risk of intracranial hemorrhage following treatment.\(^{41,42}\) In our opinion, antithrombotic therapy with heparin is not recommended because of lack of proven benefit in humans and risk of hemorrhage following embolism or thrombosis.\(^{1,24}\)

In summary, there is no proven specific treatment of ischemic encephalopathy in animals. Potential treatment options in the acute phase include hyperosmotic and antioxidant therapy with mannitol as needed to control ICP and, possibly, the iron-chelating agent deferoxamine.\(^{31}\) Hypotension is a potential side effect of rapid deferoxamine administration.\(^{37}\) In addition to antioxidant and hyperosmotic therapy, administration of a calcium-channel blocker such as amlodipine is a potential treatment option during the acute phase of treatment with adequate BP monitoring. Antiplatelet therapy with low-dose aspirin (0.5 mg/kg PO q24h) may be initiated safely to most patients and has been shown to prevent recurrence due to platelet activation following acute stroke in humans.\(^{1,43}\) No controlled studies in veterinary patients have assessed these potential treatment options. Therefore, the dose, frequency, and timing of therapy are unknown at this time.

Glucocorticoid therapy is a controversial aspect of stroke treatment. There is no evidence in the human or veterinary literature that ascribes any proven benefit to the use of glucocorticoids in stroke patients.\(^6\) Glucocorticoid therapy is not recommended in human stroke patients.\(^{24}\) In our opinion, the use of glucocorticoids in veterinary stroke patients is not indicated because of lack of proven benefit, increased coagulation, risk of gastrointestinal complications, and increased risk of infection.\(^{24}\)

**PROGNOSIS**

There are no controlled studies in the veterinary literature regarding prognosis following cerebrovascular ischemia or hemorrhage. Early recognition of ischemic or hemorrhagic infarction as a possible cause of acute neurologic dysfunction is important to avoid unnecessary euthanasia of patients in which a poor prognosis may be incorrectly assumed.\(^6\) In humans, prognosis is reportedly related to size, location, cause, and progression of the stroke event.\(^4,6\) Most human deaths due to cerebrovascular accident are a result of nonneurologic complications such as aspiration pneumonia, deep vein thrombosis, or pulmonary thromboembolism.\(^4,24\)

In a recent retrospective analysis of 33 dogs with MRI or necropsy evidence of brain infarction, there was no association among the type of infarction (e.g., large or small), region affected (telencephalic, thalamic/midbrain, cerebellum), and presence or absence of concurrent medical conditions. Similarly, there was no association between the type or region of infarction and outcome. Dogs with medical conditions that predisposed them to the initial infarction had significantly shorter survival times and were significantly more likely to experience reinfarction compared with dogs with spontaneous or idiopathic stroke.\(^29\)

In our experience, patients with documented cerebral ischemic insult have generally recovered neurologically over several days to weeks. In general, recovery from cerebrovascular accident is more pronounced in domestic animals than in humans.\(^8\) In humans, fatal brain edema following stroke has been associated with a history of hypertension, heart failure, elevated leukocyte count, and greater than 50% hypodensity of middle cerebral artery territory on an early CT scan.\(^{44}\) In humans, global cerebral ischemia following cardiopulmonary resuscitation generally carries a grave prognosis with a 60% mortality rate due to severe brain damage, with only 3% to 10% of patients able to resume normal
activities. The prognosis for global ischemia in dogs is difficult to predict because there are no controlled studies and few case reports. A report of two cases (one dog and one cat) of suspected global ischemia showed neurologic improvement but with persistent blindness in both patients at 14 and 15 months, respectively.

REFERENCES

1. Which statement regarding the use of CT in diagnosing ischemic encephalopathies is incorrect?
   a. CT is inferior to MRI in detecting ischemic infarction.
   b. CT has demonstrated a sensitivity of 80% in detecting ischemic infarction within 24 hours after occurrence.
   c. Acute hemorrhagic infarction tends to result in hypodense lesions that are difficult to detect.
   d. Temporal changes in CT findings following ischemic infarction are similar to those of MRI but differ based on time from infarct to earliest detection.

2. Which is true when comparing lacunar and territorial infarctions?
   a. Lacunar infarctions are large, and territorial infarctions are small.
   b. In veterinary patients, infarctions of the cerebellum and telencephalon are typically lacunar.
   c. Thalamic infarctions are typically territorial.
   d. Lacunar infarctions involve small, deep, penetrating arteries of the brain.

3. Which statement regarding vasogenic and cytotoxic edema formation is true?
   a. Vasogenic edema is hypointense on T2-weighted MRI scans and hyperintense on T1-weighted MRI scans.
   b. FLAIR imaging is useful to differentiate CSF from vasogenic edema.
   c. Cytotoxic edema is extracellular water accumulation, and vasogenic edema is intracellular water accumulation.
   d. MRI can always detect cytotoxic edema within 1 hour of infarction.

4. Which area of the brain has been shown to be susceptible to global ischemia?
   a. hypothalamus
   b. brain stem
   c. cerebral cortex
   d. midbrain nuclei

5. Which best describes the characteristics of embolic infarctions in dogs?
   a. round, indistinct margins, multiple
   b. wedge-shaped, sharp demarcation, single
   c. wedge-shaped, sharp demarcation, multiple
   d. wedge-shaped, sharp demarcation, single, often associated with hemorrhage

6. Treatment goals in dogs with ischemic encephalopathies do not involve
   a. recognition and treatment of neurologic complications such as seizures and elevated ICP.
   b. maintenance of adequate tissue oxygenation.
   c. providing adequate nutritional support.
   d. immediate treatment with thrombolytic agents such as tissue plasminogen activator or streptokinase.

7. Which statement regarding elevated ICP is false?
   a. Overzealous ventilation can lead to decreased venous return and cerebral hypoxia.
   b. Elevation of the head 40% above horizontal can aid in lowering ICP and optimize blood flow to the brain.
   c. Mannitol can be administered up to three times within 24 hours.
   d. Elevated ICP can cause systemic hypertension.

8. Which anticonvulsant would be preferred for seizure management in a stroke patient with altered mentation?
   a. phenobarbital
   b. zonisamide
   c. potassium bromide
   d. diazepam

9. Which factor has the most profound effect on the prognosis of ischemic brain infarction in dogs?
   a. type (lacunar versus territorial)
   b. location
   c. underlying disease that caused the infarction
   d. degree of neurologic dysfunction at presentation

10. Which has been reported following recovery from global brain ischemia?
    a. persistent circling
    b. persistent blindness
    c. refractory seizures
    d. persistent torticollis