Feline arterial thromboembolism (ATE) is an acute or peracute, and often devastating, condition that results from embolization of a thrombus within a peripheral artery. The prevalence of this condition is approximately 0.3% to 0.6%. Cats seem to be more susceptible than animals of other species to development of ATE, probably because of the higher prevalence of myocardial disease with left atrial enlargement in cats; the propensity for intracardiac thrombus formation seems to be higher for cats. Because of the severe clinical signs resulting from acute arterial embolization, a high proportion of cats are euthanized at initial presentation; however, a significant proportion of treated cats will be able to regain motor function to affected limbs and maintain a good quality of life. Long-term prognosis, however, may be limited by the type and severity of any underlying conditions.

**ETIOLOGY**

In most affected cats, initial thrombus formation occurs as a consequence of left atrial enlargement caused by significant cardiac disease, most commonly hypertrophic cardiomyopathy; however, any form of cardiomyopathy or congenital defect (e.g., mitral stenosis) affecting the left heart may result in ATE. A rare underlying cause of ATE is infective endocarditis, which results in embolization of septic thrombi in the systemic circulation. Occasionally, noncardiac conditions may result in ATE; the most common is pulmonary neoplasia with subsequent tumor embolism. For a very small proportion of cats with ATE, no underlying cause may be identified.

**PATHOPHYSIOLOGY**

Classically, the factors that predispose to excessive thrombus formation are described by Virchow’s triad: endothelial dysfunction, blood stasis, and a hypercoagulable state (FIGURE 1). All of these factors can contribute to ATE in cats. Dilation of the left atrium and/or left auricular appendage can lead to endothelial damage and blood stasis, both of which may be further exacerbated by left atrial systolic
hypercoagulable states may be harder to identify in cats. Platelet hyperaggregation has been implicated as a significant factor contributing to a hypercoagulable state in cats with ATE. Typical measures of coagulation, such as prothrombin time (PT) and activated partial thromboplastin time (aPTT), are better suited to identifying hypocoagulability and, thus, are of limited use for identifying hypercoagulable states because the range of values for these measurements can significantly overlap between normal and hypercoagulable patients. A more recent hemostasis test, calibrated automated thrombography, has been shown to be more sensitive than PT, aPTT, and rotational elastography and may have a potential role for monitoring hemostasis in cats. The gold standard for assessment of platelet function in cats is platelet aggregometry; however, performance of the test is very dependent on operator skill and experience.

For most cats with ATE, the initial thrombus forms in the left heart before either the entire thrombus, or a piece thereof, dislodges and enters the systemic circulation, eventually lodging in an artery of smaller diameter than itself. After a thrombus has formed and dislodged, the subsequent embolus not only directly and mechanically obstructs the affected artery but also triggers a cascade of vasoactive events that cause vasoconstriction of the collateral circulation. Occlusion of the systemic circulation to the affected area results in acute ischemia of the tissues supplied by the obstructed vessels, and clinical signs of ATE then develop. In most cats with ATE, the result is poor systemic perfusion and shock (maldistributive, cardiogenic, or both).

Of note, the clinical syndrome seen with ATE does not develop in cats that undergo surgical single or double ligation of the aorta alone; however, when 5-hydroxytryptamine (serotonin) is injected into the space between double ligatures, a syndrome similar to ATE results. In addition, administration of cyproheptadine (a serotonin antagonist) or high-dose aspirin (a thromboxane A2 inhibitor) before thrombus formation preserves collateral circulation and prevents paralysis, thus supporting the role of vasoactive mediators in the pathogenesis of the clinical syndrome of ATE.

**SIGNALMENT**

In studies of feline ATE, male cats are overrepresented. However, predominance of male cats in studies is considered to reflect the higher prevalence of hypertrophic cardiomyopathy among male than female cats. Typical age at presentation is approximately 8 to 12 years. Most cats are not pedigreed (i.e., usually domestic shorthair or longhair). Overrepresented pedigree breeds include Abyssinian, Birman, and Ragdoll as well as Maine Coon, Himalayan, Siamese, and Persian cats.

**CLINICAL SIGNS AND PRESENTATION**

The onset of clinical signs as a result of ATE is typically acute or peracute with little to no warning signs. The condition causes a significant degree of distress not just for the cat but also for the owners witnessing the event. Cats with ATE will experience severe pain in the affected limb(s), often vocalizing as a result and exhibiting clear signs of distress and discomfort. The exact nature of the clinical signs may vary, depending on the actual location of the thromboembolism within the peripheral circulation, which depends on both the size of the embolus and the vessel diameter at the various levels of the arterial tree. For most affected cats, the affected arteries are appendicular; however, nonappendicular arteries (e.g., mesenteric, renal, and cerebral) may also be embolized.

The most common presentation is that of distal aortic thromboembolism (saddle thrombus) at the level of the
aortic trifurcation, which is characterized by paralysis or paresis of one or both pelvic limbs. When both hind limbs are affected, one may be more severely affected than the other. Embolization can also occur within either of the brachial arteries and similarly results in lower motor neuron signs affecting the ipsilateral forelimb. A study of cats diagnosed with ATE in general practice veterinary clinics found that 1 limb was affected in 20.8% of cats, 2 limbs in 77.6% of cats, and 3 or more limbs in 1.2% of cats.¹ Cats display clear neurologic deficits in the affected appendages, often dragging the limb(s) in question. The severity of signs is relative to the degree of vascular occlusion; signs are more subtle when occlusion is partial. Other potential signs include vomiting, abdominal pain, or central nervous system abnormalities, depending on the location of the thrombus.² Affected cats are typically hypothermic, which may be a direct result of obstruction of vessels supplying the hindquarters but more often reflects systemic hypoperfusion and shock as a secondary consequence of the vasoreactive cascade resulting from tissue ischemia.

At presentation, some cats with ATE may have evidence of underlying cardiac disease (e.g., murmur, gallop rhythm, or arrhythmia). Nevertheless, absence of auscultation abnormalities in cats does not preclude underlying cardiac disease. Concurrent congestive heart failure (CHF) has been reported for 40% to 67% of cats with ATE;¹²¹³ therefore, some cats may also exhibit signs of CHF (e.g., dyspnea, tachypnea, orthopnea, lung crackles, and open-mouth breathing). Note that tachypnea and open-mouth breathing may also be manifestations of the acute pain of ATE. Most affected cats have no known history of heart disease; the sudden signs of ATE are the first indicators of severe cardiac disease.

Many cats with ATE are hyperglycemic, which is attributed to stress hyperglycemia resulting from epinephrine and cortisol release. Azotemia is also frequently encountered; the origin may be prerenal or renal. Typically, blood urea nitrogen (BUN) elevation is encountered more frequently and tends to be more severe than creatinine elevation.²¹ In these patients, prerenal azotemia occurs in response to poor systemic perfusion and shock. In patients with prerenal azotemia, the BUN:creatinine ratio may be elevated. Renal azotemia occurs as a direct consequence of renal ATE. Serum creatine kinase is usually severely elevated due to muscle ischemia. Hyperphosphatemia is also frequently encountered. Hyperkalemia, which can be severe and life-threatening, may result from reperfusion injury and is one of the most significant complications of ATE if perfusion to affected tissues is restored. Although hyperkalemia is most often seen after treatment for ATE, some cats may have this abnormality at presentation. Hyponatremia and hypocalcemia may also be observed. Results of routine coagulation assays such as PT and aPTT are often within reference range in cats with ATE; however, D-dimers may sometimes be elevated.

**DIAGNOSIS**

ATE in cats can typically be diagnosed on the basis of physical examination alone. Five cardinal signs are associated with appendicular ATE (the 5 Ps): pain, paralysis/paresis, pulselessness, pallor, and poikilothermy (FIGURE 2). Affected limbs are painful, and the muscles are often firm. Patients display lower motor neuron signs in affected limbs; severity ranges from mild paresis to complete paralysis. In one study, 34% of cats with ATE still had some motor function in affected limbs; likelihood of motor function was higher for those in which the forelimbs or 1 hind limb were affected.² Differential diagnoses for these neurologic signs include peripheral neuropathies (e.g., diabetic neuropathy), spinal cord disease (fibrocartilaginous embolism, neoplasia, trauma), and intracranial lesions; however, with the exception of fibrocartilaginous embolism and trauma, acute onset of signs is rare for these conditions and neoplastic causes are often associated with spinal pain rather than limb pain. Pulses distal to the level of the embolism are absent or very weak. Pulse quality may be challenging to determine, particularly in the forelimbs, even in the absence of ATE. Other factors that may affect interpretation of pulse quality include obesity and patient demeanor, particularly in the face of acute pain. Doppler evaluation of the affected limb is a quick and easy method that may help identify pulses in these circumstances. The nail beds and paw pads should be examined and compared with those of the nonaffected limbs; in cats with ATE, they are pale or even cyanotic, depending on the degree of ischemia. Poikilothermy is used to describe the affected limbs being cooler than nonaffected limbs as a result of diminished blood flow distal to the embolism. A recent study that used infrared thermography in cats with acute bilateral hind limb paralysis found that a cutoff value of 2.4°C (4.3°F) difference in temperature between ipsilateral affected and nonaffected limbs had excellent specificity.
Temperature assessment using this technique requires an infrared camera, availability of which may be limited, but as these devices become smaller and less expensive, this quick and noninvasive method of assessment may be useful, particularly in emergency practice settings. Differential measurements of blood glucose and serum lactate between affected and unaffected limbs may also provide supportive evidence of ATE. In peripheral venous blood samples from affected limbs, blood glucose is lower and lactate is higher than that in venous samples from nonaffected limbs or central veins. One study showed that an absolute blood glucose concentration difference between central and peripheral venous samples of 30 mg/dL or more was 100% sensitive and 90% specific for predicting ATE in cats. Similar cutoff values for the serum lactate concentration difference between affected and unaffected limbs predictive of ATE in cats have not been determined. Rarely, imaging of the implicated artery (e.g., ultrasonography of the vasculature, angiography, magnetic resonance imaging, or computed tomography with angiography) may be necessary to confirm the diagnosis or investigate the presence of underlying causes.

**CLINICAL MANAGEMENT**

**Emergency Treatment**

**Immediately Provide Analgesia and Oxygen**

Most cats with ATE experience severe pain and distress. Any cat in respiratory distress will need supplemental oxygen. Prompt and effective analgesia with opioids should be administered as soon as possible. Suitable analgesics for these patients are full µ-opioid receptor agonists (e.g., methadone, fentanyl, oxymorphone, and hydromorphone). Various prognostic factors should be considered when deciding to proceed with treatment versus euthanasia, all of which should be clearly understood.

**FIGURE 2.** Hallmarks of appendicular arterial thromboembolism (the 5 Ps).
discussed with clients. Factors that have been associated with nonsurvival are rectal temperature lower than 37°C (98.6°F), reduced heart rate, absence of motor function, and having more than 1 limb affected. Another negative prognostic indicator is confirmed concurrent CHF.

Assess Cardiac Function
After the patient has been stabilized with oxygen therapy and analgesia has been administered, assessment for CHF should be the next priority. Focused point-of-care ultrasonography and/or thoracic radiography should be performed to check for pleural effusion and/or pulmonary edema, respectively; however, patient stability should never be sacrificed to perform these diagnostics. In patients for which the index of suspicion for CHF is high (e.g., lung crackles) or those with confirmed evidence of CHF, diuretic therapy with furosemide (1 to 2 mg/kg IV or IM) should be administered and repeated hourly or even more frequently until an effect is reached. The frequency of dosing intervals may then be adjusted to effect. For cats with significant pleural effusion, thoracocentesis is required for stabilization.

Address Shock and Poor Perfusion
Most cats with ATE will exhibit signs of shock and poor systemic perfusion, which should be addressed promptly. These signs may be the result of maldistributive shock secondary to tissue ischemia distal to the embolus and the resultant release of vasoactive substances, cardiogenic shock secondary to significant cardiac disease, or a combination of both. Approaches for correcting systemic perfusion will therefore vary according to the underlying cause, which may be difficult to discern at the time of initial presentation. For dehydrated cats without CHF, fluid therapy may be considered, although great care must be taken when administering intravenous fluids to any patient with cardiac disease. Positive inotropes such as pimobendan (off-label) at 0.15 mg/kg IV or 0.3 mg/kg PO may therefore be a more useful consideration for patients with cardiac compromise and CHF, especially if echocardiography indicates systolic myocardial dysfunction. Many cats with ATE will have low rectal temperatures and may even have generalized hypothermia as a consequence of their shock and poor systemic perfusion. Although it may seem reasonable to attempt to actively warm these hypothermic patients, warming should be avoided until the patient’s systemic perfusion has been corrected because active warming results in peripheral vasodilation, which diverts more blood away from the core organs and therefore worsens core perfusion and the effects of shock.

Short-Term Management

Begin Antithrombotic Therapy as Soon as Possible
After the patient has been stabilized, antithrombotic therapy should be initiated. Antithrombotic therapies are given to prevent propagation of existing thrombi and prevent development of new thrombi. These drugs do not cause lysis of existing thrombi. As soon as possible, therapy with low–molecular weight heparin (e.g., dalteparin 75 to 150 U/kg SC q6h) or unfractionated heparin (250 to 300 U/kg q6h) should be initiated, although data regarding clinical outcomes following use of these medications for acute ATE management are lacking. Heparin therapy is typically discontinued 2 to 3 days after the patient has been stabilized and is receiving oral antithrombotics.

As soon as oral drug administration is tolerated by the patient, clopidogrel should be started. This drug is currently regarded as the mainstay of antithrombotic therapy in cats with ATE. Clopidogrel reversibly inhibits platelet aggregation via adenosine diphosphate antagonism. An initial loading dose of 75 mg PO per cat is recommended, followed by a maintenance dose of 18.75 mg PO per cat q24h. Clopidogrel is quite bitter, causing most cats to hypersalivate and become averse to administration. For this reason, the author recommends administering clopidogrel in gelatin capsules. Clopidogrel is typically well tolerated. At high doses (e.g., 75 mg/cat q24h), signs of excessive bleeding, such as bruising, may be observed.

Although aspirin was once a mainstay of therapy for feline ATE, it has since been superseded by clopidogrel, which has been demonstrated to be more efficacious at the secondary prevention of ATE (i.e., prevention of recurrence of ATE in cats that have previously experienced an ATE event). Aspirin irreversibly inhibits thromboxane A2 on platelets, thus inhibiting platelet aggregation. Because aspirin’s mechanism of action differs from that of clopidogrel, dual therapy with both drugs may be advantageous, although this combination has not been studied. Typical aspirin doses are 20.25 to 81 mg PO per cat q72h. A potential side effect of ATE is gastrointestinal
ulceration, which can be minimized by administering aspirin only after the patient has resumed eating.

**Thrombolytics Are Not Recommended**

Various thrombolytic medications have been used for cats with ATE, including tissue plasminogen activator,21,22 streptokinase,11 and urokinase.23 However, none of these medications has shown any beneficial effects on survival when compared with standard-of-care therapy with anticoagulants. Administration of thrombolytic medications has also resulted in significant complications, most notably life-threatening hyperkalemia, probably a result of reperfusion injury. For these reasons, thrombolytic medications are not recommended for cats with ATE.

**Rheolytic Thrombectomy Is Not Recommended**

Use of rheolytic thrombectomy in a small cohort of cats has been described.24 This catheter-based technique uses pressurized saline jets to physically lyse the thrombus and create a vacuum immediately adjacent to the catheter. Use of this technique has been associated with a hospital discharge rate of 50%; however, no data on long-term survival are available.24 This technique is also not currently recommended as a treatment for feline ATE.

**Monitor Pain**

Analgesia should be continued, and the patient should be regularly assessed for pain. For most cats, pain greatly eases after the first 24 to 48 hours. At this time, buprenorphine (a partial µ-opioid receptor agonist) may be sufficient for analgesia; it can also be used for cats with only mild ATE signs at presentation.

**Begin Physiotherapy**

To minimize the risk for muscle contracture, physiotherapy exercises, such as passive manipulation of affected limbs, should be started as soon as the patient is stabilized and pain is well controlled.

**Perform Additional Diagnostics**

After the patient is stable and comfortable, any additional diagnostics that are required to investigate underlying causes should be performed (e.g., echocardiography for patients with suspected or confirmed cardiac disease, hemogram, serum biochemistry, or ultrasonography of any implicated arteries).

**Monitor for Complications**

One of the most significant complications in cats receiving treatment for ATE is severe, life-threatening hyperkalemia and acidosis as a result of reperfusion injury to the tissues. For the first 48 to 72 hours after presentation, ATE patients should be closely monitored for these biochemical derangements, which should be promptly addressed with appropriate therapies (e.g., administration of dextrose, insulin and dextrose, calcium gluconate, or sodium bicarbonate, as indicated).

**Long-Term Management**

**Antithrombotic Therapy Should Continue**

Clopidogrel (at 18.75 mg/cat) should be continued because it has been shown to be clinically superior to aspirin for the secondary prevention of ATE.20 Dual therapy with clopidogrel and aspirin has been advocated by some clinicians, but studies of the efficacy of this approach are lacking.

For cats that are severely affected by ATE or those that have experienced recurrent episodes of ATE, low–molecular weight heparin or unfractionated heparin therapy can be continued long-term at home. However, given the required dosing frequency and the necessity for subcutaneous administration, these options are not routinely used for long-term management and should be considered only for compliant cats and very dedicated clients who should be clearly taught appropriate subcutaneous injection technique. Data regarding outcomes of cats receiving these medications long-term after ATE are limited.

**Cardiac Workup May Be Needed**

Because most cats with ATE have underlying cardiac disease, appropriate management of clinical consequences of heart disease is necessary. After cats with ATE have been stabilized, those with concurrent CHF will require ongoing diuretic therapy with furosemide (0.5 to 2 mg/kg PO q8 to q12h). Renal function should be assessed. For cats without evidence of renal disease, addition of an angiotensin-converting enzyme (ACE) inhibitor such as benazepril (0.5 to 1.0 mg/kg PO q24h) or enalapril (0.25 to 0.50 mg/kg PO q12 to q24h) may be considered and, if initiated,
followed by close monitoring of renal function. For cats with azotemia or known existing renal disease, ACE inhibitors should be used with caution. Pimobendan is sometimes used off-label at a dose of 0.3 mg/kg PO q12h for cats with evidence of systolic dysfunction. Various antiarrhythmic medications may also be used, depending on the type and severity of any concurrent arrhythmias. A detailed discussion of the management of heart disease and CHF in cats is outside the scope of this article; readers are directed to other resources for an in-depth review of this topic.19

**Exercises Should Continue**

After the patient has been discharged from the hospital, passive mobility exercises should be continued at home for at least several weeks until motor function improves and the risk for muscle contracture has subsided. Clients should be educated on how to perform the physiotherapy exercises.

**Newer Treatment Options**

Alternatives to low–molecular weight heparin or unfractionated heparin have been suggested for both short-term and long-term management of patients with ATE and include the oral factor Xa inhibitors rivaroxaban (0.5 to 1 mg/kg/day) and apixaban. To date, studies of these drugs in cats have evaluated only their pharmacokinetics, pharmacodynamics, and effects on coagulation assays in cohorts of healthy cats.25,26 Although clinical studies assessing the efficacy of rivaroxaban in feline ATE are ongoing, data on the efficacy of these drugs in clinically affected patients with ATE are currently lacking. It is recommended that these drugs should be administered in combination with, and not as a replacement for, standard clopidogrel therapy.19

Another newer anticoagulant drug is dabigatran, which is a direct thrombin inhibitor licensed for use in humans to prevent stroke and systemic embolism resulting from atrial fibrillation. This drug has not yet been investigated in cats.

**PROGNOSIS**

The long-term prognosis for cats with ATE is generally guarded to poor but may vary depending on the clinical severity and the precise underlying cause. A study of cats diagnosed with ATE in a general practice setting showed that, including those that were euthanized, only 12% survived at least 7 days after presentation.1 In that study, 61.2% of cats were euthanized at presentation and another 11.6% were euthanized (8.8%) or died (2.8%) within the first 24 hours after starting treatment; only 27.2% of cats overall survived more than 24 hours.1 Overall survival rates of approximately 30% to 40% and even as high as 73% have been reported previously; however, the higher survival rates were observed in referral practice settings, which may have introduced a referral bias.2,11-13 Conversely, the low overall survival rates observed in general practice may indicate a degree of inherent bias among general practitioners toward euthanasia for cats with ATE and perhaps reflect a perception of a hopeless prognosis. Nevertheless, survival rates in cats with only a single limb affected are as high as 70% to 80%.11,13 This number is even as high as 90% if some motor function remains at presentation.12 In many cases, the clinical signs of ATE significantly improve after the first 24 to 48 hours of therapy. In particular, the need for analgesia often reduces significantly after this initial period and underscores the need for effective analgesia from the time of presentation. Of cats that survive the first 48 to 72 hours, many will regain some or even all motor function within 1 to 2 months. For these reasons, it may be prudent to encourage clients to at least consider therapy for the initial 72 hours, which may result in increased overall survival rates.

Negative prognostic indicators include presence of a gallop heart rhythm,14 reduced heart rate,2,11 previous occurrence of ATE,14 having 2 or more limbs affected,1,2 and rectal temperature lower than 37°C (98.6°F).1,2 A rectal temperature lower than 37.2°C (98.9°F) at presentation has been shown to be associated with a survival rate of less than 50%.2 Several long-term complications may result as a consequence of ATE, and clients should be educated about the risks and warning signs of these complications. Muscle contracture can occur in affected limbs and may be mitigated by performing physiotherapy during
Clopidogrel therapy is considered the mainstay for prevention of ATE in at-risk cats, and its use in at-risk cats is currently recommended.19

hospitalization and having clients continue it at home. Some patients may experience skin necrosis and skin sloughing on affected limbs as a result of ATE-induced ischemia; these lesions sometimes become apparent only after 3 to 5 days. Lesions may be localized to the digits but may also affect larger portions of skin over the limb and may require surgical management. For some cats with ATE, perfusion may be so poor that entire limb necrosis necessitates amputation of the affected limb.3 In cats with persistent neurologic deficits, dragging of the limbs may lead to excoriations on the paws, and some cats may self-traumatize affected limbs. For these cats, gabapentin may be of some use for managing neuropathic pain, although its use in cats with ATE has not been investigated.

PREVENTION STRATEGIES

No studies evaluating the efficacy of any therapy for the primary prevention of ATE (i.e., preventing a first ATE event in an at-risk patient) have been published in the veterinary literature. Clopidogrel therapy is considered the mainstay for prevention of ATE in at-risk cats, and its use in at-risk cats is currently recommended.19 A randomized, positive-controlled trial comparing clopidogrel with aspirin for the secondary prevention of ATE demonstrated increased time to ATE recurrence or cardiac death among cats receiving clopidogrel after an initial ATE event.20 In that study, median time to ATE recurrence or cardiac death among cats receiving clopidogrel was 346 days versus 128 days for cats receiving aspirin.20 One of the biggest hurdles to preventing ATE is identification of at-risk cats because many cats with ATE have subclinical underlying cardiac disease, of which their owners may be unaware. When a murmur, gallop rhythm, or arrhythmia is detected in an asymptomatic cat, a workup for underlying cardiac disease is recommended. For cats identified as having cardiac disease, several echocardiographic parameters have been associated with increased risk for ATE, including moderate-to-severe left atrial enlargement, reduced atrial fractional shortening, reduced left atrial ejection fraction, increased left ventricular wall thickness, low left atrial appendage velocities, and spontaneous echo contrast (smoke). All of these parameters are considered indications for starting clopidogrel therapy in asymptomatic cats. Recent guidelines on the classification, diagnosis, and management of feline cardiomyopathies propose a staging system for describing the clinical effects of cardiomyopathy irrespective of the precise underlying cardiomyopathy. Cats classified as having stage B2 cardiomyopathy (asymptomatic cats with moderate-to-severe left atrial enlargement) are considered at a higher risk of developing CHF or ATE, and clopidogrel therapy is recommended for cats with this and all subsequent stages of cardiomyopathy.19 It is unknown if dual therapy with clopidogrel and either aspirin or a factor Xa inhibitor provides any additional benefit for the prevention of ATE. Although some factors associated with cardiogenic ATE may help provide a basis for prevention strategies, ATE may also result from noncardiogenic causes and, rarely, in the absence of any discernible cause; thus, preventive recommendations for these subsets of patients are limited. TVP

References


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Feline Arterial Thromboembolism

TOPIC OVERVIEW
Feline arterial thromboembolism (ATE) is an acute and often devastating condition; reported prevalence is 0.3% to 0.6%. This article provides an overview of ATE, including pathogenesis, diagnosis, treatment, prognosis, and prevention strategies.

LEARNING OBJECTIVES
After reading this article, the reader will be able to describe the pathogenesis of arterial thromboembolism (ATE) in cats. The reader will be able to recognize the clinical signs of ATE and describe the appropriate treatment and preventive strategies for this condition. The reader will also have an understanding of the prognostic factors associated with feline ATE.

1. Recommended therapy for feline arterial thromboembolism (ATE) may include all of the following EXCEPT:
   a. Tissue plasminogen activator
   b. Clopidogrel
   c. Low-molecular weight heparin
   d. Aspirin

2. Compared with aspirin, clopidogrel has been shown to have increased efficacy for the secondary prevention of feline ATE.
   a. True
   b. False

3. All of the following are cardinal signs of appendicular ATE EXCEPT:
   a. Pallor
   b. Paresis
   c. Pyrexia
   d. Pain

4. It is recommended to start clopidogrel prophylactically at which stage of feline cardiomyopathy?
   a. Stage A
   b. Stage B1
   c. Stage B2
   d. Stage C

5. Which of the following medications results in thrombolysis?
   a. Aspirin
   b. Unfractionated heparin
   c. Tissue plasminogen activator
   d. Low-molecular weight heparin

6. All of the following opioid drugs are appropriate for initial emergency treatment of cats with severe pain caused by ATE EXCEPT:
   a. Buprenorphine
   b. Fentanyl
   c. Hydromorphone
   d. Methadone

7. In cats with ATE, rectal temperature 37.2°C (98.9°F) at presentation is associated with approximately what percentage of survival?
   a. 100%
   b. <75%
   c. <50%
   d. <25%

8. Which of the following is the most common cause of feline ATE?
   a. Pulmonary neoplasia
   b. Idiopathic
   c. Infective endocarditis
   d. Hypertrophic cardiomyopathy

9. For primary prevention of feline ATE, clopidogrel has been shown to increase median time to initial ATE or cardiac death in at-risk patients.
   a. True
   b. False

10. One of the most severe and life-threatening complications that may occur in patients treated for arterial thromboembolism is
   a. Hypernatremia
   b. Hyperphosphatemia
   c. Hypermagnesemia
   d. Hyperkalemia