Canine Atrial Fibrillation

Abstract: Atrial fibrillation (AF) is the most commonly diagnosed supraventricular tachyarrhythmia in dogs. It typically develops when atrial enlargement occurs secondary to underlying cardiovascular disease. Electrocardiographically, AF is characterized by disorganized atrial electrical activity resulting in an absence of P waves and a rapid, irregular ventricular rate. The hemodynamic consequences of AF include decreased cardiac output and the development of clinical signs of heart failure. Therapeutic management focuses on controlling ventricular rate or restoring and maintaining sinus rhythm using antiarrhythmic medication and, in some cases, biphasic transthoracic electrical cardioversion. The prognosis varies and is especially guarded in the presence of significant underlying cardiac disease, such as dilated cardiomyopathy.

Mechanisms and Pathophysiology

Atrial fibrillation (AF) is a common, sustained supraventricular tachycardia in dogs. It is characterized by disorganized, rapid atrial electrical activity resulting in loss of atrial contribution to ventricular filling as well as an irregular and typically rapid ventricular response rate. The physiologic consequences of AF include a reduction in cardiac output and development or worsening of clinical signs of heart failure. AF is most commonly diagnosed in association with atrial enlargement secondary to underlying cardiovascular disease, although not all dogs with atrial enlargement develop AF. The primary therapeutic goal is to improve cardiac output either by controlling ventricular rate or by converting AF to sinus rhythm. Therapeutic management continues to present clinical challenges.

Mechanisms for the formation and maintenance of AF are multifactorial. Abnormal electrical activity—either ectopic triggers (such as those occurring at the pulmonary veins) or reentrant waves—causes AF. Normally, a wave of depolarizing atrial myocardial cells makes atrial tissue refractory to immediate stimulation by a second electrical impulse. In reentry, abnormal paths of electrical activity allow multiple wavelets of electrical activity to travel across the atria without encountering tissue in a refractory state and to continue to depolarize myocardial tissue in a circuitous manner. The presence of fibrosis, inflammation, and wall stretch within the atrial myocardium influences the paths of electrical activity. Atrial enlargement sustains reentry by creating sufficient area to allow waves of electrical activity to move about without terminating each other. Giant-breed dogs may have enough atrial mass, even in the absence of cardiovascular disease, to have an increased risk of developing AF.

Atrial arrhythmias can be triggered by alterations in autonomic tone. Elevated parasympathetic tone increases dispersion of refractoriness, affecting wavelet activity and contributing to both discontinuation and perpetuation of AF. In dogs with advanced cardiac disease and heart failure, excessive adrenergic tone contributes to elevated heart rates, and successful
heart failure therapy alone may reduce sympathetic tone and decrease heart rate. The presence of sustained AF results in electrical and structural remodeling of the atria that perpetuates AF—that is, “AF begets AF.” Sustained elevations in heart rate (>200 bpm in dogs) can result in tachycardia-induced atrial and ventricular systolic dysfunction, even in the absence of underlying cardiovascular disease. Structural remodeling also occurs during heart failure, which often coexists with AF, via angiotensin II– and aldosterone-mediated fibrosis. Angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, and aldosterone antagonists inhibit the renin–angiotensin–aldosterone system (RAAS), reducing fibrosis and the rate of AF recurrence after conversion to sinus rhythm in human patients and, experimentally, in dogs.

Incidence and Etiology

It is generally not possible to ascertain how long a dog has had AF before diagnosis, but the history is often assumed to be chronic. The intermittent, or paroxysmal, presence of AF is not routinely recognized in dogs. Most dogs with AF have atrial enlargement secondary to underlying cardiovascular disease. The most common congenital and acquired cardiovascular diseases contributing to the development of canine AF are listed in BOX 1. The documented incidence of AF is higher in large- and giant-breed dogs than in small-breed dogs, and dilated cardiomyopathy is the most commonly recognized concurrent cardiovascular disease. One possible reason for the lower rate of AF in small-breed dogs with chronic degenerative valve disease is that even in the presence of atrial enlargement, atrial mass may not be adequate to sustain AF. The breeds most commonly reported to have AF and underlying cardiomyopathy are the Irish wolfhound, Great Dane, Newfoundland, and Doberman pinscher. Lone AF occurs in the absence of obvious cardiovascular disease and is also more common in giant breeds such as the Irish wolfhound. Male dogs are more often affected.

History and Clinical Presentation

Although most dogs with AF exhibit characteristic clinical signs, those with lone AF may have no demonstrable signs at the time of diagnosis. Clinical presentation is affected by the severity of underlying cardiovascular disease and the presence of heart failure, and signs may be exacerbated by exercise. Dogs often have a history of lethargy, weakness, and exercise intolerance. Other reported abnormalities at presentation include syncope, cough, dyspnea, ascites, and anorexia.

Physical Examination

The most consistent abnormalities associated with AF are a rapid heart rate and an irregular heart rhythm. The irregularity of the rhythm may not be readily appreciated when the heart rate is extremely fast. Estimation of heart rate based on auscultation or palpation of pulses is often inaccurate. The intensity of the first heart sound is usually variable with AF. A murmur, typically systolic, may be auscultated in dogs with underlying cardiac disease. Often, the character of the murmur is inconsistent from beat to beat and depends on heart rate. Pulse quality may be normal to decreased with an irregular rhythm, and pulse deficits may be appreciated, especially with higher heart rates. Clinical evidence of hemodynamic instability includes weakness, syncope, decreased pulse quality, pulse deficits, and pale mucus membranes. Signs of congestive heart failure include tachypnea, dyspnea, ascites, and jugular venous distention.

Electrocardiography

Electrocardiography (ECG) is required to definitively diagnose AF, which is distinguished by a lack of identifiable P waves and an irregular ventricular rate characterized by a variable R-to-R interval. In
most cases, the QRS complexes are narrow and predominately upright in lead II (FIGURE 1). In the absence of organized atrial activity, the baseline is characterized by undulations identified as fibrillatory “f” waves. Pathologic atrial electrical activity bombards the atrioventricular (AV) node at a rate that is frequently >300 bpm. Normal cycling of the AV node through active and refractory states prevents some of these electrical impulses from passing through to the ventricles, resulting in an irregular ventricular rate. Occasionally, QRS complexes are conducted with a bundle branch block pattern that has a wide, bizarre morphology resembling complexes of ventricular origin (FIGURE 2). Ventricular ectopic complexes may coexist with AF, particularly in dogs with dilated cardiomyopathy (FIGURE 3). ECG criteria for ventricular enlargement, including tall R waves or deep S waves, suggest the presence of left or right ventricular enlargement, respectively. With pericardial or pleural effusion, QRS complexes may be uncharacteristically small. Before the onset of AF, dogs with underlying cardiovascular disease and enlarged atria may have an increased incidence of atrial ectopic activity, heralding the impending development of AF.

Compared with in-hospital ECG, 24-hour Holter analysis provides a better overall estimate of heart rate in the clinical setting and home environment. In one report, the resting heart rate obtained with in-hospital ECG overestimated the Holter-derived heart rate by 15% to 25%. Obtaining an accurate estimation of the average heart rate is of particular benefit when administering or adjusting antiarrhythmic medication. The ideal ventricular rate during therapeutic management of AF has not been adequately established, although the target rate for large- and giant-breed dogs is considered to be lower than for small-breed dogs. Often, the goal is to achieve a heart rate that allows the dog to remain free of clinical signs.

Additional Diagnostic Tests
Additional diagnostic tests—including echocardiography, thoracic radiography, blood pressure measurement, and hematologic testing—provide complementary information regarding the presence and severity of any underlying cardiac disease or comorbid conditions and associated hemodynamic compromise. Echocardiography is essential for evaluating
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Therapeutic management of atrial fibrillation focuses on either controlling ventricular rate (rate control) or restoring and maintaining sinus rhythm (rhythm control).

Thoracic radiography provides an estimate of cardiac size and confirms the presence or absence of congestive heart failure, including pulmonary venous congestion, pulmonary edema, and pleural effusion. Indirect measurement of blood pressure is invaluable when assessing hemodynamic status and monitoring the effects of antiarrhythmic medications, especially those that can result in systemic hypotension. However, a rapid heart rate and irregular rhythm may impair accurate indirect blood pressure measurement. A complete blood cell count and serum biochemistry profile may provide useful information regarding concurrent diseases, especially when making therapeutic decisions regarding antiarrhythmic therapy or anesthesia for biphasic transthoracic electrical cardioversion. Thyroid function testing is recommended when evaluating dogs with systolic dysfunction or those receiving antiarrhythmic therapy that can alter thyroid function, such as amiodarone.  

**Treatment**

The principal goals of therapy are to resolve clinical signs of heart failure and improve cardiac output and quality of life. Historically, in human medicine, conversion to sinus rhythm was the preferred treatment objective primarily because rhythm control restores coordinated atrial and ventricular activity to improve cardiac structure and function, characterizing underlying cardiac disease, assessing atrial size, and detecting possible atrial thrombus formation. Initial echocardiographic indices of systolic function (fractional shortening and ejection fraction) commonly underestimate the true level of myocardial contractility and should be repeated once the ventricular rate or cardiac rhythm is controlled. As a result of the irregular rhythm characteristic of AF, color Doppler recordings are frequently inconsistent in documenting variations in the severity of valvular regurgitation and peak transaortic and transpulmonic outflow velocities (FIGURE 4). Doppler indices of diastolic function may be of little use because of the absence of A waves on transmitral inflow profiles.

FIGURE 2

Examples of AF and ventricular tachycardia electrocardiograms.

Lead II rhythm strip illustrating AF with wide complexes caused by aberrant conduction due to a bundle branch block. Note the absence of P waves. Ventricular ectopic complexes (*) are present. Paper speed = 25 mm/sec; 1 cm = 1 mV.

In this example of ventricular tachycardia, P waves are identified unrelated to ventricular QRS complexes in the lead II recording. The presence of P waves differentiates ventricular tachycardia from AF with aberrant conduction from a bundle branch block. Paper speed = 25 mm/sec; 1 cm = 1 mV.

QuickNotes

Examples of AF and ventricular tachycardia electrocardiograms.
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Electrocardiogram from a dog with dilated cardiomyopathy showing AF and a ventricular ectopic complex (*). Paper speed = 25 mm/sec; 1 cm = 1 mV.

Diac output and clinical signs while potentially reducing the incidence of stroke. However, multiple studies performed to evaluate the issue of rhythm or rate control in human patients have had mixed results. Many human patients do not remain in sinus rhythm control, and an improvement in mortality has not been consistently documented. Successful conversion to sinus rhythm is technically challenging and is more likely to occur with acute AF or in the absence of underlying cardiac disease (not the common presentation in dogs). Although many antiarrhythmic medications have been used for conversion to sinus rhythm in humans, very few medications have had similar effects in dogs. Amiodarone, diltiazem, quinidine, and verapamil have been reported to restore sinus rhythm in a few dogs. Restoration and maintenance of sinus rhythm is more challenging in cases of chronic AF due to the presence of atrial remodeling. Maintaining ventricular rate within a normal range optimizes cardiac output even in the presence of AF, making adequate heart rate control a reasonable therapeutic goal.

Once a diagnosis of AF is established, initial therapy is based on the type and severity of underlying cardiac disease, the presence of clinical signs, and the hemodynamic status. Occasionally, a dog may present with a normal ventricular rate despite having received no antiarrhythmic medication (e.g., a giant-breed dog with lone AF). It is unclear whether these dogs benefit from therapeutic intervention, but there is ample evidence that a subset will develop ventricular dysfunction. Holter analysis may reveal significant elevations in heart rate, clarifying the need for antiarrhythmic therapy. Acute therapeutic intervention is recommended in dogs with evidence of hemodynamic compromise, including profound weakness, collapse, and systemic hypotension. IV antiarrhythmic therapy is required with acute, severe hemodynamic instability, whereas oral therapy is sufficient for most other cases. Although the ultimate goal of therapy may be restoration of sinus rhythm, the immediate goal is generally a reduction in ventricular rate. Any respiratory distress should be stabilized before further diagnostic testing.

Appropriate heart failure therapy, including diuretics, oxygen therapy, and positive inotropic support, should be administered for concurrent decompensated heart failure. Dogs with concurrent cardiovascular disease and heart failure typically have faster heart rates at presentation than dogs without heart failure; appropriate heart failure therapy can effec-
Antiarrhythmic medications used to manage atrial fibrillation include diltiazem, digoxin, amiodarone, and β-adrenergic blockers.

Pharmacotherapy

Antiarrhythmic medications used to manage canine AF are listed in TABLE 1. IV procainamide or diltiazem may be considered for initial, acute therapy. In the presence of a wide QRS tachycardia that cannot be definitively identified as supraventricular or ventricular in origin, procainamide is an ideal first-line IV therapeutic agent because of its broad spectrum of activity with atrial and ventricular arrhythmias. AF can develop in the perioperative or intraoperative period after the administration of opioids or as a result of gastrointestinal (GI), respiratory, or neurologic diseases that elevate parasympathetic tone. Successful conversion of vagally induced acute AF to sinus rhythm using IV lidocaine boluses or a constant-rate infusion of amiodarone has been reported. In one study, reported side effects of IV amiodarone included pruritus, erythema, and angioedema and were attributed to substances in the carrier solvent of the solution rather than to the drug itself; these

<table>
<thead>
<tr>
<th>Drug</th>
<th>Recommended Dose</th>
<th>Type of Atrial Fibrillation</th>
<th>Potential Adverse Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class 1A</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procainamide*</td>
<td>5–15 mg/kg IV slowly to effect</td>
<td>Acute</td>
<td>GI disturbance, hypotension, proarrhythmia</td>
</tr>
<tr>
<td>Class 1B</td>
<td></td>
<td></td>
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<tr>
<td>Lidocaine</td>
<td>2 mg/kg bolus IV</td>
<td>Acute</td>
<td>GI disturbance, tremors, seizures</td>
</tr>
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<td>Class II</td>
<td></td>
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<td></td>
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<tr>
<td>Atenolol</td>
<td>0.25–1 mg/kg PO q12h</td>
<td>Chronic</td>
<td>Bradycardia, AV block, negative inotropy, hypotension, weakness</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>0.25–1 mg/kg PO q12h</td>
<td>Chronic</td>
<td>Bradycardia, AV block, negative inotropy, hypotension, weakness</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>0.25–1 mg/kg PO q12–24h</td>
<td>Chronic</td>
<td>Bradycardia, AV block, negative inotropy, hypotension, weakness</td>
</tr>
<tr>
<td>Class III</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>5 mg/mL at 3 mL/min CRI to effect*</td>
<td>Acute</td>
<td>Erythema, pruritus, angioedema</td>
</tr>
<tr>
<td></td>
<td>10–20 mg/kg PO q24h loading for 5–7 days; 5–10 mg/kg PO q24h maintenance</td>
<td>Chronic</td>
<td>GI disturbance, bradycardia, hepatotoxicity, neutropenia, thrombocytopenia, corneal deposits, proarrhythmia</td>
</tr>
<tr>
<td>Class IV</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diltiazem*</td>
<td>0.05–0.2 mg/kg IV to effect or cumulative dose of 0.25 mg/kg</td>
<td>Acute</td>
<td>Bradycardia, AV block, negative inotropy, GI disturbance, weakness</td>
</tr>
<tr>
<td></td>
<td>0.5–2 mg/kg PO q8h</td>
<td>Chronic</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2–4 mg/kg PO q12h (extended release)</td>
<td>Chronic</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digoxin*</td>
<td>0.003–0.01 mg/kg PO q12h</td>
<td>Chronic</td>
<td>GI disturbance, AV block, proarhythmia, increased risk of toxicity with hypokalemia and renal failure</td>
</tr>
</tbody>
</table>

*Commonly used.
AV = atrioventricular; CRI = constant-rate infusion; GI = gastrointestinal.
effects resolved when amiodarone administration was discontinued.

During management of chronic AF, antiarrhythmic medications are selected based on the presence and severity of underlying cardiac disease, comorbidities such as hepatic or renal disease, administration frequency relative to owner compliance, and the cost and risk of adverse effects. Oral antiarrhythmic medications that are useful for managing chronic AF include diltiazem, digoxin, amiodarone, and β-adrenergic blockers (e.g., atenolol). Although rate control is the main objective in the presence of chronic AF and concurrent cardiovascular disease, conversion to sinus rhythm occasionally occurs. Antiarrhythmic agents are selected to slow atrial activity and prolong AV nodal conduction, resulting in a reduction in ventricular rate. Digoxin has mild positive inotropic effects that may be beneficial for dogs with concurrent systolic dysfunction. Because of its parasympathomimetic effects, digoxin can result in vagally mediated GI disturbance and may not be as effective in patients with heart failure and increased sympathetic tone. An important adverse effect of digoxin is proarrhythmia. Because digoxin has a narrow therapeutic window, a low dose is recommended (especially in patients with azotemia), and blood levels should be monitored on a routine basis. Administration of quinidine, amiodarone, or verapamil concurrently with digoxin can elevate serum digoxin levels, increasing the likelihood of toxicity. Hypokalemia sensitizes the myocardium to digoxin and increases the risk of toxic effects. Serum potassium values should be monitored closely, particularly in patients receiving diuretic therapy. In contrast to digoxin, diltiazem—a nondihydropyridine calcium channel blocker—has negative inotropic effects resulting from a decrease in calcium-induced contraction. Standard diltiazem tablets are administered three times a day, whereas extended-release preparations are administered twice a day, potentially improving owner compliance. In dogs and humans, coadministration of digoxin and diltiazem provides better heart rate control than digoxin alone, and this combination is often used for initial therapy of AF.25,36

Amiodarone is also considered effective initial therapy for managing AF in humans and dogs.37 Amiodarone, which is categorized as a Vaughan-Williams class III antiarrhythmic, is unique for its additional class I and ancillary class II and IV effects. Because it has a long half-life, amiodarone is typically administered at a loading dose for several days to facilitate reaching therapeutic levels. Conversion to sinus rhythm occurred in 35% of dogs and adequate heart rate control was achieved in 76% of dogs in a retrospective report of 17 dogs given amiodarone.17 Adverse effects in dogs are typically noncardiac in nature and include corneal deposits, neutropenia, thrombocytopenia, agglutination (positive Coombs test result), GI disturbance, and (most commonly) elevations in hepatic enzyme activity.17,30–41 Preexisting abnormal elevations in hepatic enzyme activity may be exacerbated by amiodarone, but elevations may resolve in weeks to months following a decrease in dose or discontinuation of the drug.17 A complete blood cell count, a serum biochemistry panel, and thyroid function test results should be evaluated before amiodarone is administered and routinely throughout therapy.

If adequate heart rate control is not achieved with a combination of digoxin and diltiazem or amiodarone, switching to the alternative therapy can be tried. If heart rate control continues to remain insufficient, additional therapy with a β blocker may be of benefit. The simultaneous administration of β blockers and calcium channel blockers should be approached with caution to avoid excessive bradycardia, AV block, decreased contractility, and hypotension. β Blocker administration is contraindicated in dogs with unstable heart failure because of negative inotropic effects. However, β blockers may be valuable in patients with stable heart failure, especially in the presence of increased sympathetic tone. β Blockers act by impeding sympathetic nervous system and RAAS activation, resulting in antiarrhythmic and antifibrillatory effects. Commonly used β blockers include atenolol and carvedilol. In addition to its β-blocking effects, carvedilol has antioxidant and α-blocking-mediated vasodilatory effects that are of potential benefit when treating dogs with heart failure.

Oral quinidine and procainamide are class IA sodium channel blockers that are infrequently used to treat supraventricular arrhythmias in dogs.29,42 Although earlier reports of their use indicated that they may be effective at restoring sinus rhythm and controlling ventricular
rate, their applications are limited by important adverse effects, including GI disturbance, proarrhythmia, the need for frequent administration, and numerous drug interactions.

Potential disadvantages of administering antiarrhythmic medications for rhythm or rate control include the probable requirement for long-term drug administration and the potential for adverse effects and drug interactions. The benefits of administration should outweigh the risk of adverse effects. Proarrhythmia is correlated with prolongation of the QT interval, which predisposes patients (especially those with bradycardia, hypomagnesemia, and hypokalemia) to torsades de pointes and sudden death. Amiodarone, sotalol, and quinidine contribute to QT interval prolongation, although sudden death is considered less likely with amiodarone in human patients. 37

Cardioversion
For dogs that can tolerate anesthesia, biphase transthoracic electrical cardioversion may be valuable for immediate conversion to sinus rhythm. With this technique, a shock is delivered at a specific time in the cardiac cycle, synchronized with the QRS complex, beginning with a low amount of energy (35 to 50 J) and increasing incrementally by 50 J up to a total of 200 J as required for successful conversion. 43 More than one shock is often necessary for conversion. The antiarrhythmic medication amiodarone can be administered to facilitate electrical cardioversion and is typically required to maintain sinus rhythm after successful cardioversion. 45 Maintaining sinus rhythm after cardioversion can be a formidable challenge, especially in the presence of atrial enlargement. 44

Long-Term Management
Comprehensive management is necessary for dogs with concurrent cardiovascular disease and heart failure. Therapeutic support may consist of diuretic therapy, angiotensin-converting enzyme inhibitors, positive inotropes, and other medications as deemed appropriate. The patient’s activity level may improve with therapy, although excessive activity is typically discouraged. Dogs with underlying cardiovascular disease should be fed a moderately sodium-restricted diet, and high-sodium foods should be avoided. Ideally, follow-up evaluations are performed at least every 3 to 4 months to monitor drug therapy and cardiovascular disease progression.

Future Directions
Advances in nonpharmacologic therapy of AF in human patients have resulted from a desire to avoid problems associated with the efficacy and adverse effects of antiarrhythmic medication. Nonpharmacologic alternatives consist of surgery and catheter-based procedures to interrupt electrical activity or ablate ectopic foci, as well as pacemaker or defibrillator implantation. 45, 46 Many of these procedures have been evaluated in canine studies but are not yet clinically available in veterinary medicine. 47, 48 Novel antiarrhythmic medications with broad spectra of activity and minimal adverse effects continue to be introduced. 49 Evaluation of cardiac biomarkers in human patients with AF (e.g., N-terminal probrain natriuretic peptide [NTproBNP] concentrations) helps guide therapy, and values are positively correlated with prognosis. 50 In a study evaluating NTproBNP concentrations in 30 dogs with either dilated cardiomyopathy or chronic degenerative valve disease, 43 dogs with AF tended to have higher serum NTproBNP concentrations than dogs in sinus rhythm, which may eventually help guide therapy.

Prognosis
Prognosis depends on the presence and severity of the underlying disease process. Survival is thought to be shorter for male dogs and for large- and giant-breed dogs with underlying structural cardiac disease. 51, 52 Reported survival times are significantly shorter in dogs with AF and concurrent dilated cardiomyopathy, and mortality exceeds 50% in the first 2 weeks following diagnosis of AF in Doberman pinschers. 52 The development of AF is often associated with clinical deterioration and shorter survival.

Conclusion
Despite the relative frequency of diagnosis, AF continues to present therapeutic challenges. Dogs identified as having AF should be evaluated for underlying cardiovascular disease, hemodynamic instability, and the presence of comorbidities that would influence therapeutic decisions. Additional studies are required to better understand the mechanisms of AF initiation and maintenance, evaluate potential medical and surgical treatment options, and perfect therapeutic strategies.

QuickNotes
The goals of therapy are to resolve clinical signs of heart failure and improve quality of life.
References

23. Bonagura JD, Ware WA. Atrial fibrillation in the dog: clinical findings and prognosis. JAAHA 1986;22:111-120.
1. Which is not used to control AF in dogs?
   a. oral diltiazem
   b. oral amlodipine
   c. IV procainamide
   d. transthoracic biphasic electrical cardioversion

2. Which is not an identifying characteristic of AF on an electrocardiogram?
   a. irregular rhythm
   b. no P waves
   c. fibrillatory “f” waves
   d. tall P waves resulting from atrial enlargement

3. Which statement regarding antiarrhythmic therapy in AF is false?
   a. The combination of digoxin and diltiazem may control the heart rate better than digoxin alone.
   b. Digoxin has parasympathomimetic effects.
   c. Lidocaine can be used for managing chronic AF.
   d. Sotalol and amiodarone can cause prolongation of the QT interval, resulting in proarrhythmia.

4. Which antiarrhythmic medication: adverse effect pairing is incorrect?
   a. amiodarone: elevations in hepatic enzyme activity
   b. atenolol: thyroid dysfunction
   c. digoxin: GI disturbance and proarrhythmia
   d. diltiazem: AV block

5. Which drug can reach toxic levels in dogs with renal failure or hypokalemia?
   a. procainamide
   b. atenolol
   c. digoxin
   d. carvedilol

6. Treatment goals for dogs with AF include
   a. conversion to sinus rhythm.
   b. reduction in ventricular rate.
   c. improvement in cardiac output and clinical signs.
   d. all of the above

7. Which of the following is not a potential mechanism for developing or sustaining AF?
   a. genetically altered sarcomeric proteins
   b. atrial enlargement and increased wall stress
   c. multiple wavelets of electrical activity interacting within the atria
   d. structural and electrical remodeling of the atria

8. Which breed(s) is/are more commonly reported to have AF?
   a. Irish wolfhound
   b. Great Dane
   c. Doberman pinscher
   d. all of the above

9. Which antiarrhythmic medication or class is not correctly matched with its inotropic effect on the ventricles?
   a. β blockers: negative inotropy
   b. procainamide: no effect
   c. calcium channel blockers: positive inotropy
   d. digoxin: positive inotropy

10. Which common cardiovascular disease(s) is/are diagnosed concurrently with AF?
    a. dilated cardiomyopathy
    b. chronic degenerative valve disease
    c. patent ductus arteriosus
    d. all of the above