Atrial Fibrillation in Small Animals

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Atrial fibrillation (AF) is an arrhythmia that rarely requires emergency treatment but often complicates cardiac disease and requires emergency intervention. AF in small animals is usually associated with underlying structural cardiac disease, such as mitral regurgitation or dilated cardiomyopathy (DCM), and consequent congestive heart failure (CHF) but can occur in large- and giant-breed dogs without structural disease (this is known as primary, or lone, AF). AF in cats is always associated with severe cardiac disease. The arrhythmia has specific diagnostic criteria that, once learned, make identification of AF relatively easy. Therapy depends on the context in which AF occurs.

DIAGNOSTIC CRITERIA

Historical Information

Gender Predisposition
None.

Age Predisposition
Adult onset. If associated with underlying cardiac disease, appearance of AF depends on the usual age of onset of the primary cardiac disorder.

Breed Predisposition
Giant- and large-breed dogs (because of large atrial size).

Owner Observations
With primary AF, owners usually do not notice a problem. With secondary AF, there is often concomitant CHF—relapse, worsening, or initial episode.

Other Historical Considerations/Predispositions
In large and giant breeds, AF is occasionally associated with gastrointestinal disease. It can also be induced by irritation of the atrial myocardium with intravenous catheters (e.g., central lines). In cases of secondary AF, there is often a history of cardiac disease, with or without CHF.

Physical Examination Findings

Auscultation
• Arrhythmia: Irregularly irregular arrhythmia, often described as “tennis shoes in a tumble dryer.”
• Other signs of heart disease: Murmur (may be difficult to auscultate because of the arrhythmia), gallop sound (again, often difficult to identify).

$ indicates relative costs of any diagnostic and treatment regimens listed.
Pulse
• Pulse deficits are present, or there is a variable pulse quality from beat to beat.
• Tachycardia is always present with secondary AF; it is variable with primary AF.

Signs of CHF
• Tachypnea.
• Orthopnea.
• Dyspnea with left heart failure.
• Ascites with right heart failure.

Cough
Gagging cough. (See “Canine Congestive Heart Failure” in the March 2000 Standards of Care).

Laboratory Findings
AF is not associated with any specific laboratory abnormalities. Any abnormalities are secondary to CHF or other underlying diseases.

Other Diagnostic Findings
Echocardiography
• Echocardiography (ECG) is the primary diagnostic tool.
• AF is defined by five criteria, two of which (tachycardia and F waves) are inconsistent:
  — Supraventricular origin: The QRS complex is narrow (looks normal) unless there is a concomitant bundle-branch block.
  — Irregularly irregular: There is no predictable relationship between one QRS complex and another. No pattern can be identified.
  — No P waves: Uncoordinated atrial depolarizations (up to 800/min) result in a lack of P waves. Fluctuations in baseline or changes in T waves can be misinterpreted as P waves.
  — Tachycardia: Always present in cases of secondary AF (with severe cardiac disease). Often >210 bpm in dogs, >250 bpm in cats. Rate is usually normal with primary AF.
  — Presence of F waves: Fibrillation waves may or may not be seen in the baseline.
• Other ECG abnormalities may be present, including evidence of ventricular enlargement or conduction abnormalities. Other arrhythmias, such as ventricular premature contractions, may be present.

Thoracic Radiography
• With left-sided CHF: Pulmonary edema; cardiomegaly, especially of the left atrium in most cases; pulmonary venous (± pulmonary artery) engorgement.
• With right-sided CHF: Pleural effusion, ascites, right heart enlargement (much more difficult to identify than left heart enlargement), globoid cardiomegaly with pericardial effusion, enlargement of the caudal vena cava.
• May be unremarkable with primary AF.

Echocardiography
• Echocardiography will usually identify the type of underlying severe cardiac disease in cases of secondary AF.
• M-mode echocardiography of the mitral valve will show a lack of A waves and irregularly irregular ventricular contractions.

Intracardiac Electrography
Electrograms recorded within the atria show rapid, uncoordinated atrial depolarizations.

Ambulatory ECG (Holter Monitoring)
Holter monitors may be required in cases in which clinicians cannot differentiate primary (lone) or secondary AF with a clinical examination. Resting heart rates on Holter recordings will be normal in primary AF and elevated in secondary AF.

Summary of Diagnostic Criteria
• ECG evidence of AF, based on criteria defined above.
• Auscultable arrhythmia.
• Variable presence of cardiac disease and CHF.
• Signalment: Large- and giant-breed dogs are most commonly affected and often have primary (lone) AF.

Differential Diagnosis
• Ventricular tachycardia or ventricular premature contractions: Can also be irregularly irregular and can be mistaken for AF on physical examination. Can be ruled out with ECG.
• Supraventricular (atrial) tachycardia: Usually very regular arrhythmia but can occur under similar circumstances as AF. Usually differentiated from AF on the basis of regularity. Also ruled out with ECG.
• Respiratory sinus arrhythmia: A regular arrhythmia. Can be ruled out with ECG.

TREATMENT RECOMMENDATIONS
Initial Treatment
Secondary AF (with Heart Disease and CHF)
In all cases, heart rate control is the primary objective. This is directed at the atrioventricular (AV) node, since reduction of AF potentials is not possible. Decreasing AV nodal conduction is required. Rate control does not require IV intervention in the vast majority of cases but is usually achieved with oral medications. Rate control is achieved by:
• Reduction in Sympathetic Tone
  — Controlling CHF decreases sympathetic tone; therefore, treating the CHF will help reduce the ventricular response rate.
  — Low-dose β-adrenergic blocker therapy: Inhibition of the adrenergic receptors will decrease AV nodal conduction and ventricular response rate. High-dose β-blocker therapy is contraindicated if severe myocardial failure exists (e.g., DCM, large-breed mitral regurgitation, severe aortic insufficiency) because they are negative inotropes.
— Common β-blocker regimens for dogs with AF include atenolol (6.25–25 mg/dog PO bid), propranolol (0.1–0.5 mg/kg PO tid), and metoprolol (5–50 mg/dog PO tid). In cats, atenolol (6.25 mg/cat PO bid) can be attempted.

• **Increase in Vagal (Parasympathetic) Tone**
  — Opposes the high sympathetic tone.
  — Digoxin is the only parasympathomimetic commonly used to increase vagal tone. Doses are initiated at 0.22 mg/m² bid in dogs and titrated to achieve serum (digoxin) levels of 1–2 ng/ml (6–8 hours post-pill). Cats: 0.01 mg/kg PO q48h (one-quarter of a 0.125-mg pill/cat every other day).

• **Inhibition of AV Nodal Cell Depolarization**
  — Achieved with Ca²⁺-channel blockers, specifically diltiazem (0.5–1.5 mg/kg PO tid); long-acting formulations, such as Dilacor XR (Watson Pharmaceuticals) can be used (approximately 10 mg/kg q12–24h). The negative inotropic effects of diltiazem at these doses are small and balanced by vasodilator properties.
  — Verapamil is generally avoided because of greater negative inotropic effects. Amlodipine has no effect on AV nodal cells.

• **Control of CHF**
  — Achieved with diuretics, angiotensin-converting enzyme (ACE) inhibitors, vasodilators, and digoxin.
  — In cases with AF, control of the heart rate (described above) is often essential for successful control of CHF. (See also “Canine Congestive Heart Failure” in the March 2000 Standards of Care).

**RESOURCE LIST**
- **Atenolol** (Tenormin, AstraZeneca): 0.5–2.0 mg/dog PO bid; 6.25 mg/cat PO bid.
- **Propranolol** (Inderal, AstraZeneca): 0.1–0.5 mg/kg PO tid.
- **Metoprolol** (Lopressor, Novartis; Toprol, AstraZeneca): 5–50 mg/dog PO sid-tid.
- **Digoxin** (Lanoxin, GlaxoSmithKline): Dogs—0.22 mg/m² PO bid and titrated to achieve serum (digoxin) of 1–2 ng/ml (6–8 hr post-pill). Cats—0.01 mg/kg PO q48h (one-quarter of a 0.125-mg pill per cat every other day).
- **Diltiazem** (Cardizem, Aventis Pharmaceuticals): 0.5–1.5 mg/kg PO tid.
- **Diltiazem** (long-acting) (Dilacor XR, Watson Pharmaceuticals): 10 mg/kg PO q12–24h.
- **Procainamide** (Procan, Parkedale Pharmaceuticals): 8–20 mg/kg slow IV, 10–20 mg/kg PO tid.

**CHECKPOINT**
Some clinicians believe that certain giant-breed dogs (Irish wolfhounds) develop primary AF as part of subsequent DCM. Other authors have failed to see progression of AF to DCM in these cases.

**Primary AF (without Underlying Heart Disease or CHF)**
- No rate control is usually necessary.
- Cardioversion may be attempted.
- In rare cases in which AF is associated with extracardiac disease (e.g., gastrointestinal disease), treatment of the extracardiac disease can result in spontaneous reversion of the AF to sinus rhythm. This is common in cattle and can occur occasionally in small animals.

**Alternative/Optional Treatments/Therapy**

**Electrocardioversion**
Electrocardioversion is generally unsuccessful in most cases of primary AF due to the duration of the AF prior to detection. It may be successful in cases of primary AF of short duration (<1 month). However, it is often difficult to determine the duration of primary AF in nonathletic animals. Electrocardioversion should not be attempted in cases of secondary AF because the likelihood of success is minimal and the risk can be significant.

**Chemical Cardioversion**
Again, generally unsuccessful except in cases of iatrogenic AF (catheter-induced), in which procainamide (8–20 mg/kg slow IV) can be used to convert the patient back into sinus rhythm. Quinidine can be used for cardioversion of chronic primary AF but is associated with potentially severe side effects and low success rate and is not recommended by this author.

**AV Nodal Ablation and Ventricular Pacing**
Permanently prevents any AF potentials from reaching the ventricles. Rate is controlled by a ventricular pacemaker. This is rarely attempted in veterinary patients.

**Atrial Electrocautery (Maze Procedure)**
The atrial myocardium is scarred by catheter-based electrocautery to interrupt the reentrant arrhythmias responsible for AF (a maze is created, allowing only a single path for impulses to reach the AV node), resulting in resumption of sinus rhythm. Has not been attempted in veterinary medicine.

**Patient Monitoring**

**Primary AF (without Underlying Heart Disease)**
- Monitoring of the resting heart rate is advised (yearly or twice yearly). An ECG might be required, although auscultation usually suffices.
- May be an early sign of DCM in some giant breeds. It is unknown whether these patients develop myocardial failure.
subsequent to chronic AF (even with normal heart rates). Echocardiographic monitoring may be advisable (every 12–24 months) to ensure persistence of normal myocardial function.

**Secondary AF (with Cardiac Disease and CHF)**
- Monitoring heart rate (ventricular response rate) with changes in therapy is essential. This usually requires an ECG because the irregular tachycardia makes it difficult to auscultate the heart rate accurately. The optimal heart rate in dogs with secondary AF is 140–160 bpm, although this may be difficult to achieve in some cases and usually takes several weeks to optimize.
- Monitoring of the CHF is necessary. Respiratory rate should be counted, and thoracic radiographs may be necessary to confirm resolution of pulmonary edema. (See also “Canine Congestive Heart Failure” in the March 2000 *Standards of Care*).
- Serum (digoxin) should be measured approximately 5–7 days after initiating therapy to ensure an appropriate serum concentration (1–2 ng/ml).

**Home Management**

**Primary AF**
Owners can maintain a heart-rate diary (recording a heart rate once a week) to confirm the status of the condition.

**Secondary AF**
Owners will generally be unable to measure heart rates in these patients (See also “Canine Congestive Heart Failure” in the March 2000 *Standards of Care*).

**Milestones/Recovery Timeframes**

**Primary AF**
No change is anticipated over time. If the heart rate increases with time (months or years), further evaluation via echocardiography might be required to determine if myocardial failure is developing.

**Secondary AF**
Improvement in CHF is anticipated within 2–3 days (improved respiration, decreased respiratory rate). A decrease in ventricular response rate is anticipated within the first week. Further adjustments in drug therapy should be anticipated at this time (within 1 week) to optimize heart rate and CHF control. Ultimate rate control may take several weeks to achieve or may never attain the anticipated response.

**Treatment Contraindications**
β-blockers should never be used with Ca<sup>2+</sup>-channel blockers because they can cause profound AV nodal blockage, resulting in third-degree AV block and death. These drugs can each be used as monotherapy or in combination with digoxin for optimizing rate control.

**PROGNOSIS**

**Favorable Criteria**
- **Primary AF:** Generally considered a benign condition in nonathletic animals.
- **Secondary AF:** Rapid resolution of clinical signs (CHF) and reduction in heart rate to target rate (140–160 bpm) within 7 days.

**Unfavorable Criteria**
- AF in cats is considered a poor prognostic indicator because it is associated with severe cardiac disease.
- Concomitant myocardial failure is a poor prognostic indicator.

**RECOMMENDED READING**


